Sudden Oak Death: Interactions of the Exotic Oomycete *Phytophthora ramorum* with Naïve North American Hosts

Matteo Garbelotto and Katherine J. Hayden
Department of Environmental Science, Policy and Management, University of California, Berkeley, California, USA

Ten years after a threatening and previously unknown disease of oaks and tanoaks appeared in coastal California, a significant amount of progress has been made toward the understanding of its causal agent *Phytophthora ramorum* and of the novel pathosystems associated with this exotic organism. However, a complete understanding of the ecology and epidemiology of this species still eludes us. In part, our inability to fully understand this organism is due to its phylogenetic, phyleogeographic, phenotypic, and epidemiological complexities, all reviewed in this paper. Most lines of evidence suggest that the high degree of disease severity reported in California is not simply due to a generalized lack of resistance or tolerance in naïve hosts but also to an innate ability of the pathogen to survive in unfavorable climatic conditions and to reproduce rapidly when conditions become once again favorable.

Few plant pathogens have received as much attention as *Phytophthora ramorum* since it was first described only a decade ago. A threatening and previously unknown disease of oaks and tanoaks appeared almost simultaneously in several coastal locations of California around 1994 and was quickly named sudden oak death (SOD). The causal agent of this disease was identified only in 2000, when an undescribed *Phytophthora* species was consistently isolated from diseased oaks (*Quercus* spp.) and tanoaks (*Notholithocarpus densiflorus*) and shown to be the causal agent of the observed outbreaks. Discovery and research milestones of this decade included (i) the determination that *P. ramorum* was also the causal agent of a leaf and branch dieback of ornamental plants, later named ramorum blight, which affected rhododendrons and viburnums in German and Belgian commercial plant nurseries (123, 124), (ii) the discovery that ramorum blight was also present in U.S. and European nurseries outside Belgium and Germany (29, 37, 114), (iii) the determination that *P. ramorum* was mostly an aerially dispersed oomycete (25, 28, 86) and in fact the first forest *Phytophthora* ever to have been described with such a transmission mode in the temperate zone (several others were to be discovered in the years to follow), (iv) the discovery that infectious airborne sporangia were not produced in significant numbers on the bole lesions responsible for oak and tanoak mortality but rather were extremely abundant on foliar lesions of a newly discovered host, California bay laurel (*Umbellularia californica*) (25, 28), and on the leaves and twigs of tanoaks (26, 27), (v) the progressive discovery that over 100 species could be infected by the pathogen, including both U.S. native and ornamental species (29, 43, 114), (vi) the determination that the source of the California infestation could be traced back to infected ornamental plants (22, 81, 101) and that the pathogen itself was comprised of four genetically distinct lineages, each with a fixed mating allele (52, 66, 67, 117, 125), (vii) evidence that the pathogen could be retrieved from soil and water in both forests (25, 41) and nurseries (107), (viii) the understanding, based on the sequencing of the entire genome (115), that while sexual reproduction in *P. ramorum* currently appears to be absent and not fully functional (5, 10, 119), the species had reproduced sexually in the past, (ix) the discovery that the number of infested nurseries worldwide was greater than originally determined (e.g., see references 24, 75, and 90), with novel wild outbreaks in some European Union countries (9) and novel infestations of water courses in the United States (94), and (x) the 2010 report of a new disease of planted Japanese larch in Great Britain and Ireland, named sudden larch death, in which larches not only are rapidly killed by *P. ramorum* but also act as the main infectious host (11, 122).

It has become clear that *P. ramorum* has a level of epidemiological and evolutionary complexity unusual for a forest disease. Unlike many other forest phytophthoras (reviewed in reference 58), it is comprised not only of distinct populations but of multiple evolutionary phenotypically distinct lineages (8, 36, 66, 67, 117), which have evolved in isolation for hundreds of thousands of years (22, 49, 51, 81, 101). Lineages are not uniformly distributed in North America and Europe, and the pathogen is still adapting to new habitats and hosts, as evidenced by the emergence of sudden larch death (11, 122). Furthermore, *P. ramorum* causes at least three types of disease (lethal cankers, leaf and branch dieback, leaf blotches or spots) on different hosts. While multiple disease forms are not uncommon in the pathosystems of agricultural phytophthoras, other major forest phytophthoras, such as *P. cinnamomi*, *P. alni*, and *P. lateralis*, are primarily known to cause only root and butt rots (6, 7, 58, 98). The discovery of an aerial phytophthora in forest systems was a dramatic departure from the expected pattern and has expanded the understanding of phytophthora disease etiology and epidemiology in forests. Surveys for *P. ramorum* led to the discovery that at least two other newly described species have similar host ranges and canker, dieback, and leafspot symptoms but appear to be associated with considerably less mortality (127). *P. nemorosa* (57) and *P. pseudosyringae* (69) may have the genetic signature of recent introductions to North American forests (76) but have not been found to have a similar complexity of multiple clonal lineages and origins. Finally, the epidemiology of SOD is also complex, as only some hosts are...
both infectious and lethally affected, while others are lethally affected noninfectious dead ends, and yet others are infectious but have symptoms that are nonconsequential to their fitness. This particular life cycle is different from that of heteroecious plant pathogens, in which alternation between hosts is required for reproduction (reviewed in reference 97), and is more akin to the relationship between infectious and noninfectious hosts observed in zoonotic diseases (3).

**P. ramorum** highlights the indirect effects of human commercial and industrial activities on natural ecosystems: in this case, a pandemic affecting forest trees has been clearly driven by the long-distance trade of infected ornamental plants used not for reforestation but for landscaping in the urban-wild interface. Given this scenario, it should be no surprise that in less than a decade, **P. ramorum** has driven a significant policy shift in how plant pathogens are diagnosed by the regulatory agencies of many countries, moving from a diagnosis exclusively dependent on morphological identification of cultures to a more articulated process in which culturing, immunological, and nucleotide sequence-based detection assays must be utilized (2, 35).

**P. ramorum** has been the object of hundreds of papers, including one describing the sequencing of the entire genome (115) published only 3 years after the isolation of the pathogen in California (47). Several reviews are available on this pathogen, including those focusing on the general ecology (58, 106), the early history of research (45), disease management (105), and pathogen diversity and the associated diseases (51, 52). In this review, we focus on the biology, ecology, host-pathogen interactions, and invasion genetics of the pathogen in California and Oregon forests. While the review hinges on research performed specifically on the lineage of the pathogen present in California and Oregon forests (see below), occasionally and out of necessity, it will refer to research performed on lineages present elsewhere. While results from other lineages may be generally applicable to the entire species, the reader should be aware of the distinction.

**P. RAMORUM: AN UNUSUALLY COMPLEX FOREST PATHOGEN**

Although **P. ramorum** was first isolated in German and Belgian nurseries in the mid-1990s (123), it was only after the pathogen was found to be the causal agent of SOD in California (47) that research on this previously unknown species gained momentum. In fact, the formal description of the species (124) and the paper describing its role as the causal agent of sudden oak death (104) were published just a few months apart. Ten years after those two milestone papers, a search using the scientific binomial and the disease name “sudden oak death” returned 495 publications. This large number highlights the importance given to this organism by researchers around the world, undoubtedly also because of the significant regulations imposed on this organism by several countries. Its presence both in commercial nurseries and in forests has required the attention of researchers normally working on very different groups of organisms, thus multiplying efforts and outcomes.

**P. ramorum** is in the so-called clade 8 of the genus **Phytophthora** and is a close relative of **P. lateralis**, **P. hibernalis**, and **P. foliorum** (73). The origin of **P. ramorum** is still unknown, but the recent discovery in Taiwan of **P. lateralis** (7), a species also presumed to be exotic and present in California and Oregon, suggests that this group of phytophthoras may have an eastern Asian origin. **P. ramorum** itself includes 4 genetically and phenotypically distinct lineages (8, 36, 66, 67, 117), suggesting an independent evolution of these four groups in geographically isolated areas such as islands or deep-set valleys. It is a heterothallic species requiring two distinct mating types (A1 and A2) to complete its sexual cycle and form oospores. Interestingly, each lineage carries almost exclusively one mating type. The North American NA1 and NA2 lineages carry exclusively the A2 mating type, while the European EU1 and EU2 lineages carry almost exclusively the A1 mating type (54, 66, 100, 125, 126). Lineages appear to have evolved in isolation for tens if not hundreds of thousands of years (49), and, perhaps as a result, the species as a whole seems to have lost some of its original sexual functionality. Many crosses appear to be infertile and result in aborted oospores (10). Nonetheless, sexual reproduction was common at some point in the history of this species, as evidenced by heterozygosity throughout its genome (115). A study on A1-A2 progeny revealed a high incidence of non-Mendelian inheritance and of aneuploidy suggestive of significant differences in synteny (gene arrangements), and possibly in chromosomal number, between lineages (119). Nonetheless, two studies have determined that a percentage of oospores generated from laboratory crosses may be viable (5, 35), highlighting the importance of avoiding intermixing of lineages carrying different mating types (Fig. 1). All genotyping completed so far indicates that no sexual recombination has occurred recently in forests or nurseries (66, 81, 100, 101, 118). However, it should be pointed out that U.S. nurseries harbor multiple lineages with opposite mating types (54, 117, 126), and recombined genotypes may have emerged during nursery outbreaks, only to have been destroyed when measures were taken to control these episodes. In U.S. forests, only the A2 mating type is currently present; a release of A1 in forests could lead to large populations of each type and eventually result in successful mating and oospore production. At present, one stream in Humboldt County, CA, is infested with both the EU1 and NA1 lineages of the pathogen (20), but no plant infections by the EU1 lineage have been reported in nature in the

---

**FIG 1** A viable, thick-walled oospore of **Phytophthora ramorum** produced by crossing an NA1 with an EU1 genotype. Photo by D. Huberli, UC Berkeley, presently at the Department of Agriculture and Food, Western Australia.
United States. The potential for the production of oospores is of great concern, not only because sexual recombination may accelerate adaptation (42) but also because oospores are known to survive through much harsher environmental conditions than any other structure produced by Phytophthora spp. (reviewed in reference 68).

In the absence of sexual reproduction, P. ramorum makes use of an effective mode of asexual reproduction common to phytophthoras (59, 68), based on the prolific production of two types of propagules, sporangia and chlamydospores, serving as infectious and survival structures, respectively (25, 41, 91, 114). Sporangia are 25 to 97 μm in length, 14 to 34 μm in width (124), and deciduous, allowing them to become airborne; in fact, air transmission, albeit in association with water droplets, is the prevalent spread pathway of this pathogen in forests. Although sporangia can germinate directly in the presence of high relative humidity (128), observed infection rates are greatest where there is a film of water on the plant surface for a minimum of 6 to 9 h (65, 113), and field observations have indicated that most infection in California occurs in the presence of rainfall (25, 26). The need for a long-lasting film of water on plant surfaces for abundant infection to occur seems to suggest that most plant infection by P. ramorum is not determined by direct sporangium germination but rather by the zoospores that sporangia contain; instead, a controlled experiment determined that artificial inoculations were more successful when using zoospores than when using sporangia alone (128). Sporangia of P. ramorum normally contain approximately 30 motile and biflagellate zoospores (128), which may undergo several rounds of additional germination and encystment, albeit with vigor decreasing at each additional encystment (88). Hence, primary, and possibly to some degree secondary, germination phases of zoospores are probably driving the majority of infections. Zoospores of Phytophthora spp. may remain active for hours or as long as days (59, 68). The longevity of P. ramorum zoospores is supported by the observations of Moralejo and Descals (88) and Hübner et al. (65), who report increasing incidences of infection for up to 48 h after the addition of P. ramorum zoospores to a wet plant surface. P. palmivora cells have been observed to remain active for up to 84 h in vitro (4), suggesting that zoospore activity over this length of time is possible in nature.

Infection by zoospores not only depends on the presence of water on plant surfaces but also is strongly regulated by temperature. Under controlled laboratory conditions, infection of bay laurel leaves by zoospores was optimal at 19°C (65), and in a region-wide analysis of disease severity, the highest disease incidence was associated with an average maximum temperature of ca. 17°C, compared to ca. 16°C in uninfested plots (84). Other factors may enhance infection levels, in spite of a slightly less-than-optimal temperature, as supported by evidence that tanoak infection may enhance infection levels, in spite of a slightly less-than-optimal temperature range for sporangia germination and sporulation in petri dishes were completely arrested when cultures were exposed to 45°C for 2 h or 40°C for 24 h. Flash treatments at 55°C were never effective in affecting growth and sporulation of the pathogen in culture; however, 1 h at 55°C completely arrested its growth (109). P. ramorum could not be recovered from artificially infected oak stems 2 to 4 cm in diameter after 1 week at 55°C (109), but a separate study reported that a longer 2-week treatment at 55°C was required to eliminate its recovery from infected bay laurel leaves (60), possibly because of the chlamydospores that are prolifically produced in this host but mostly absent on oaks.

DISEASE ETIOLOGY AND EPIDEMIOLOGY

By combining results from field and laboratory experiments, the following scenario can be suggested to describe the epidemiology of SOD in California coastal forests. These forests are adapted to long, dry summers and include many evergreen species (12, 121); most of these woody species are hosts of P. ramorum, with black oak being a notable exception. Additionally, temperatures in areas colonized by P. ramorum follow a clear gradient, with temperatures in dry summers being lower (30-year average range, 23 to 29°C) on the coast and higher inland (30-year average range, 27 to 33°C) (99). That trend is somewhat reversed in the wet winter, but rarely do temperatures drop to zero in these forests.

The pathogen appears to oversummer in infected plant material (bole lesions, twig and foliar infections), in soil, and in water (25, 39, 40, 72). However, only infected leaves and twigs of infectious hosts seem to initiate the yearly disease cycle (39). A study by Chimento et al. on bay laurel leaves (14) suggests that the pathogen may oversummer both through mycelium and/or sporangia and through chlamydospores present in infected leaves. In a hot and dry site, in fact, respiration of the pathogen was measured by reverse transcription PCR (RT-PCR) and the results indicated that the pathogen was alive, but the percentage of successful culturing was significantly lower than in two other more mesic sites, implying that survival in hot conditions was mediated by chlamydospores whose germination was not triggered by the standard plating protocol. A significantly higher degree of isolation success from symptomatic bay leaves collected in the other sites may suggest that survival in less harsh conditions may be mediated instead by easily culturable mycelium or sporangia. Fichtner et al. (40, 41) have also documented the pathogen’s survival through the summer months in leaves in the soil, but the ability of the pathogen to start aerial infections from the soil, although possible in laboratory experiments, has yet to be demonstrated in nature.

Abundant sporangial production has been documented in conjunction with rainfall on bay laurel leaves and tanoak twigs and leaves (Fig. 2), while more limited sporulation has been observed on redwood needles (27). Sporangia are released during rain events, which allows for the spread of the pathogen in rain splash (25, 28, 89). The presence of thick coastal fog does not seem
to be an adequate substitute for rainfall, as evidenced by the observation of almost all new infections during the rainy spring months and not during the foggy summers (26, 27). Although sporangia have not been captured in rain traps further than 10 m from a source (25), indirect evidence based on the rate of enlargement of infestations (55) and on spatial autocorrelation analyses of alleles (81) suggests that their movement range is significantly greater than 10 m. It may be useful to extrapolate a rate of dilution of infectious propagules from the spatial autocorrelation curve produced by Mascheretti et al. (81), which suggests that 20% of all infectious sporangia produced may reach 100 m from their source. An interesting bimodal pattern of dispersal has been deduced from spatial autocorrelation analyses, suggesting that in the right climatic conditions (abundant rainfall, strong winds, large numbers of sporangia already present), a significant number of sporangia may travel as far as 3 to 5 km. This range of spread is also confirmed by observations of new foci of infection 3 to 5 km away from established infestations in Oregon, where an active eradication program is under way (48, 55). A bimodal spread pattern matches the spread dynamics of particles comparable in size to *P. ramorum* sporangia, whether organismal or inorganic, that in the presence of sufficient wind speed are picked up and not released for a distance directly proportional to the size and weight of the airborne particle (93). In this respect, the spread dynamics of *P. ramorum* are similar to those reported for *P. capsici*, whose sporangia are normally transmitted for relatively short distances in a turgid state, and seem to differ from those of other phytophthoras, including *P. infestans*, whose sporangia remain vital upon drying and can be spread dry for much longer distances (reviewed in reference 38).

Although sporangia are produced during the entire rainy season, infection appears to be more effective when temperatures are relatively warm (26, 65, 113); hence, rainfall and warm temperatures are both required to sustain abundant infection. Accordingly, data from field surveys show that in years when rainfall ends in March, the rate of new infection is minimal, while rainsfalls in April, especially in May, and in June appear to be correlated with higher rates of new infections (44). It is currently unclear how infection of plant hosts is attained by zoospores; although no wounds are required for infection to occur (28, 56), evidence suggesting that stomata on leaves and lenticels on bark may be the major avenues of infection is still circumstantial (15, 91). Experiments on bay laurels, tanoaks, and oaks show that successful infection is positively correlated with the number of zoospores released and hours of leaf wetness, but the dynamics of successful infection differ among these important hosts. Maximum rates of infection of bay laurel and tanoak leaves have been obtained by inoculum concentrations of $10^4$ to $10^5$ zoospores ml$^{-1}$ and incubation times of 6 to 9 h, but some infection can be obtained with much lower inoculum loads (i.e., density of infectious zoospores) and exposure times (56, 65); similar patterns have been observed in rhododendrons and other hosts (56, 113). In contrast to the relatively lenient requirements for laboratory infection of foliar hosts (56, 114), only concentrations above $10^4$ zoospores ml$^{-1}$ will result in successful artificial inoculations of mature stems of true oaks (46), suggesting that high levels of inoculum are required for oak infection in nature. This experimental evidence well matches field observations describing significant infection only for oaks near an infectious host (23, 71, 77, 82) and only in years when sporangial production is high due to the presence of rainfall in the late spring (72).

Individual tanoaks and bay laurels do not require high inoculum loads for infection to occur. They may thus occasionally be infected at distances of a few kilometers from an existing infestation and start a new localized outbreak, allowing the disease to move from stand to stand (80, 100). This spread makes control of new infestations particularly difficult, as often new satellite outbreaks tend to coalesce (34), making their extirpation practically impossible.

The highest levels of inoculum in California have been mea-
sured on and under infected bay laurel trees (25, 27, 43), and a large number of hosts may be infected in conjunction with the presence of bay laurel trees and favorable environmental conditions. Symptoms in these hosts can range from leaf spots (e.g., honeysuckle, big leaf maple, and Western starflower) to tip wilting (e.g., Douglas fir) and branch dieback (e.g., madrones, huckleberries, manzanitas) (43). The epidemiological role played by these hosts (with the notable exception of oak species; see below) appears to be minimal or is still unknown, and their incidence of disease or rates of mortality do not appear to be at levels high enough to threaten their survival or compromise their populations. This conclusion is based on the assumption that these hosts do not in fact support significant sporulation; however, the discovery in the United Kingdom and Ireland that infected larches support abundant \textit{P. ramorum} sporulation (122) is a clear warning that the dynamics of spread may change as the pathogen encounters novel hosts. On the other hand, tanoak twig and leaf infection also play an important epidemiological role, as they support abundant production of sporangia. Tanoak foliar infections follow a pattern very distinct from that on bay laurels: while infections of bay laurels are clearly associated with parts of the leaves where water accumulates (tips, lower blades), tanoak twigs, petioles, and midveins appear to be highly susceptible to infection and lesions are clearly associated with them, often spreading from the twig onto the leaves following the midvein or vice versa (Fig. 2) (29, 56). Oak leaves are rarely infected (29, 120).

**ECOLOGY AND IMPACTS OF SOD IN CALIFORNIA FORESTS**

At the time of this review, the three main groups of hosts affected by \textit{P. ramorum} in California are oaks in the red group (specifically coast live oak \textit{[Quercus agrifolia]}, Shreve’s oak \textit{[Q. parvula var. shrevei]}, and California black oak \textit{[Q. kelloggii]}), tanoaks, and California bay laurels. Canyon live oaks, belonging to the so-called intermediate oak group, can also be infected by \textit{P. ramorum} (110), but the impact on this species still needs to be fully assessed. Although bay laurel represents the most infectious vector for \textit{P. ramorum}, all evidence suggests that the impact of the disease on this species is minimal (31). In the field, infections are exclusively in the leaves, with a limited percentage of the canopy being infected and with the lower, shadier branches displaying the highest levels of infection (72). The early senescence and abscission of infected leaves are most frequent in the warmest, driest forest types and much less so in cooler, moister forests (26). On the contrary, in areas with significant infestations of \textit{P. ramorum}, the bay laurel component of the plant community appears to be on the rise, due to their rapid vegetative growth that takes advantage of the space made available by the death of oaks and tanoaks (16, but see reference 103). A strong positive correlation is present between the overall stand incidence of bay laurels and levels of SOD-related mortality of oaks and tanoaks, indicating the primary infectious role played by this species (18, 30, 53, 78, 82, 83). Additionally, sudden oak death incidence has been associated with higher fuel loads (74, 116) and fire-related mortality of medium-sized redwoods has been observed to be significantly higher in areas with higher SOD incidence (87), providing evidence of the cascading effects of the abundance of bay laurels in forest stands affected by SOD.

Bay laurel populations may differ in their susceptibility to infection by \textit{P. ramorum}; in particular, Oregon provenances appear to be significantly less susceptible than most California ones, while a few California populations appear to be extremely susceptible. Although this variability seems to be regulated both genetically and by the environment (1, 65), it is measurable and has a significant impact on disease levels. Where bay laurel populations are most resistant, tanoaks appear to be the primary and necessary infectious host; where populations are most susceptible, it appears that high bay laurel susceptibility may lead to high disease incidence even where climatic conditions are not ideal for the spread of SOD (65).

Lethal lesions in oaks and tanoaks appear to be initiated on the bole for both tree genera, with several notable differences: (i) tanoaks of all ages can be infected, while oaks less than 10 cm in diameter at breast height (DBH) are almost never infected; (ii) lesions can be anywhere on tanoaks, while they tend to be on the lower part of oak boles or where bay laurel branches intersect large oak branches and stems; (iii) although the so-called “bleeding” on the outer bark in correspondence to under-bark lesions (a symptom also known also as gummosis in orchard tree species infected by several \textit{Phytophthora} spp.) is extremely variable in intensity depending on the tree, this symptom is often lacking in infected tanoaks. Although the reasons behind these differences are not fully understood, it is reasonable to presume they are in general agreement with a much higher susceptibility of tanoaks. Inoculation experiments have shown that inoculum loads necessary to infect tanoaks are lower than those needed to infect oaks (56). In both species there is a demonstrated correlation between size of the tree and its probability of becoming infected, with infection being significantly more prevalent in larger trees (17, 82, 102).

The mechanisms leading to tree death are currently unknown, but two nonmutually exclusive explanations point to cambial girdling (104) and to occlusion of vessels in the xylem (19, 96) as major mechanisms of tree decline. While observations show that lesions effectively girdle the cambium of infected trees, tree species have been known to survive for years after cambial death (13), making an experimental support of this mechanism hard to obtain. On the other hand, experimental evidence has been gathered on significant changes in water potential and on the presence of tyloses in the xylem in association with colonization by \textit{P. ramorum} (19, 96, 108). Drying of the canopy may occur suddenly and in a short time period (hence the “sudden” in SOD), but often it happens several years after lesions have girdled the cambium. Because of this delayed response of the canopy, secondary organisms such as decay fungi and bark beetles (82, 83) appear to accelerate the death process. While it may be true that these secondary organisms accelerate the decomposition process and the onset of the final and visually discernible demise of the tree, it has yet to be proven that they actually increase the mortality rate. In general, most of the decay fungi and bark beetles involved are regarded as secondary agents, capable only of attacking severely compromised trees. One unexpected effect of these secondary attacks is that SOD-infected trees which are still green (but with significant cambial girdling and xylem occlusion) may be affected by these secondary organisms; in the absence of SOD these secondary organisms accelerate the decomposition process and the onset of the final and visually discernible demise of the tree, it has yet to be proven that they actually increase the mortality rate. In general, most of the decay fungi and bark beetles involved are regarded as secondary agents, capable only of attacking severely compromised trees. One unexpected effect of these secondary attacks is that SOD-infected trees which are still green (but with significant cambial girdling and xylem occlusion) may be affected by these secondary organisms; in the absence of SOD these secondary organisms are normally observed only on trees that are visually dead, i.e., characterized by a dry canopy.

Both tanoaks and oaks have shown significant intraspecific variability in susceptibility. Tanoak mortality in infested sites in California has been observed at rates significantly greater than the baseline, at 5.5 to 6% year$^{-1}$ (78, 82, 83), with an average of 20% (85) to 25% (30) basal area dead at the landscape scale but with up
to 60% (78) to 100% (30) of mature stems at the level of the plot, with exponentially increasing mortality over time (15). Indeed, a model by Cobb et al. (17) suggests that the complete loss of tan-oaks as a codominant overstory species is likely to occur in large portions of the tanoak’s geographic range. Even if the species is lost from the overstory, there may be a long period during which the species persists as an understory shrub, due to its propensity for prolific stump sprouting—much as the American chestnut has been lost from the eastern U.S. overstory (17, 103). It is not clear, however, how this reduction in stature and the associated loss of wildlife food and habitat will affect the ecological characteristics of mixed redwood-tanoak stands, which are among the most productive in the world (103, 121).

Within the Cobb et al. model, the rate of extinction for large trees was sensitive to environmental factors as well as to differences in rates of infection, transmission, and mortality among even small size classes of tanoaks. Laboratory and field disease trials have documented variations in sapling infection and mortality rates among tanoak families and individuals; a study of inoculations of detached leaf from throughout the tree’s geographic range, documenting quantitative variation in resistance among individuals and populations, also highlighted intriguing interactions of environment and disease (62). Because this first study was focused on mature, wild trees, environmental and genetic effects could not be disentangled, but there was a significant correlation between lower average minimum daily temperatures and greater susceptibility to infection spread, even though inoculations and incubations occurred in the lab, after detached branches were equilibrated under common conditions for several days. This potential finding of increased susceptibility when grown at lower temperatures may fit with the observation of Davidson et al. (25–27) that tanoak infections begin earlier in the spring than do bay laurel infections, and it may also play a role in the tanoak’s much greater role in the spread of sudden oak death in Oregon than in the rest of its range (55, 65). The presence of heritable resistance has been detected with inoculation resistance assays of open-pollinated seed families, collected from sites spanning the tanoak’s range and with the trees grown together in a common nursery, as well as concurrently with a field disease trial, tracking disease and mortality in the nursery trees’ siblings planted out in an infested site (61, 63). The studies together have documented ecologically relevant genetic resistance in a largely susceptible host species; these differences may well play a large role in disease dynamics overall.

Oak mortality has been reported in the range of 3% to 5.5% year$^{-1}$, depending on site (12, 85), with the percentage of standing dead trees similar to that of tanoak at the landscape scale (85). Most of this variability may be explained by age of the infestation, by the amount of bay laurel intermixed with oaks (30, 71, 78, 82), and by the number of other tree species present, with a higher diversity of species leading to lower overall infection rates (53). Additionally, intraspecific variability in oaks’ susceptibility to $P. \ ramorum$ has been observed in inoculation studies performed on detached branches of coast live oaks (32, 33). Because these experiments were performed on excised plant parts of wild trees of unknown parentage, it has been impossible to determine whether the observed variation was genetically or environmentally determined, but it seems reasonable that both mechanisms may play a part. In a study by Dodd et al. (33), the average susceptibility of trees in populations with older infestations, where only survivors could be tested, was lower than the susceptibility of a nearby population in which the infestation was newer and had not yet reached high levels of infection, suggesting that more susceptible trees are indeed preferentially infected by the pathogen. The susceptibility of oaks was measured to be maximal when trees were physiologically active and experienced highest levels of evapotranspiration and translocation of nutrients, suggesting that tree phenology may play an important role in determining their susceptibility. Experimental evidence from a survey of disease symptoms in the field combined with a laboratory inoculation of bay laurels (65) points to a synchronism between times of maximum oak susceptibility and times of greatest sporulation potential on bay laurel leaves. The extent of oak mortality, despite the high levels of inoculum necessary for infection, suggests that the synchrony between pathogen sporulation and the susceptibility of both bay laurels and oaks may play a large role in driving disease.

A different explanation of the various susceptibilities to infection by $P. \ ramorum$ may reside in the constitutive presence in some oaks of compounds with antimicrobial properties. Some phenolic compounds were found to increase in concentration in the tissue of both naturally and artificially infected oaks (95). The preliminary identification of compounds whose presence is associated with smaller lesions in infected mature oak trees is also promising in the effort to understand how oaks survive infections (21, 92).

**PATHOGEN VARIABILITY AND HISTORY OF INVASION IN U.S. FORESTS**

Although it is still unknown where $P. \ ramorum$ originated, it is clear that the major global spread pathway is associated with the movement of infected ornamental plants (reviewed in reference 51). All four known lineages are in fact present in nurseries or ornamental gardens worldwide, and three of the lineages are present in U.S. nurseries. Since the completion of the first two studies of the genetic structure of NA1 populations, it has become evident that the NA1 lineage in California forests went through a strong bottleneck and currently displays very limited genetic variability (49, 50, 80, 81, 101). It is reasonable to assume that the observed genetic bottleneck may have been caused by the transfer of $P. \ ramorum$ from its native environment to nurseries, followed by escapes from ornamental plants into the wild.

Based on the index of association, which compares the amount of linkage disequilibrium among markers to a random distribution, it appears that all California forest populations are reproducing exclusively clonally (66). Currently, the overall genetic depiction of California forest populations is that of a “network” of hundreds of individuals, all very closely related, and generated from 3 to 4 progenitors, which have become the most abundant genotypes at the center of the network. Genotypes with only 1 to 3 individuals represent more than half of the overall population and are only rarely present in multiple sites (22, 80, 81). Presumably, most of these singleton genotypes are the end products of unique and different local pathways of accumulation of successive mutations at the simple sequence repeat (SSR) loci used as markers. The presence of hundreds of genotypes originating in the absence of recombination implies populations of enormous sizes, a consequence of the great success of establishment of this exotic pathogen in California.

Using coalescent analysis and comparing migration levels among study sites by pairwise $PHI_{ST}$ estimates (an analog of the
index of fixation $F_{ST}$), the spread routes of the pathogen from initial introduction sites have been reconstructed with remarkable confidence (22, 80, 81). The picture that emerges from these analyses is that of an infestation originating from nursery populations in Santa Cruz and Marin Counties, with at least 6 additional instances of founder populations having clearly been introduced by humans, as indicated by the presence of genetically identical but spatially disjoint populations (80). The understanding of the almost simultaneous introduction of the pathogen in distant locations in California spanning from southern Humboldt County to the Big Sur region (Monterey County), followed by a relatively contained “natural” spread, well explains how the current range of distribution came to be and contradicts an alternative scenario of an incredibly fast spreading process from one or a very few founding sites.

A modified approach to coalescent analysis, with the age of the infestation used as a constraint, puts nurseries as the undisputed original source of the California infestation (22). Coalescence shows that as time progresses, a direct connection with nursery sources dissipated, and recent outbreaks find their source in naturalized wild populations. However, when attempting to place infestations caused by singleton genotypes, some appeared to have been directly derived from nurseries as late as 2005, potentially revealing a hard-to-detect and continuous trickle of nursery genotypes into wildlands.

Several lines of evidence point to one to four related genotypes as the founders of the California epidemic (22, 80, 81, 101). Interestingly, one of the four genotypes is ancestral to the rest and is almost exclusively found in outbreaks directly associated with a nursery source. The other three founder genotypes are clearly derived from it, and they appear to be significantly more widespread than their progenitor (22). Although genotyping is accomplished through neutral SSR markers and, hence, is not directly informative of genotypic fitness, it appears that the three derived genotypes have established themselves more successfully than the ancestral nursery genotype, potentially suggesting a quick adaptation of \textit{P. ramorum} to California ecosystems (Fig. 3). A comparative study of virulence of genotypes belonging to the four groups is needed to corroborate this likely hypothesis of adaptive microevolutionary processes.

Although the existence of four clusters within the NA1 lineage is a recent discovery (22), significant phenotypic differences have emerged not only among but also within lineages (8, 36, 64, 79). A study testing 12 NA1 genotypes (64) determined that those with greater virulence displayed that trait across hosts, without any sign of host specialization. However, when pooled together, isolates from oaks were significantly less virulent than isolates from foliar hosts, even when inoculated on oaks. In a follow-up study employing over 100 isolates, Kasuga et al. (70) confirmed that a sizable proportion of genotypes isolated from oaks showed a non-wild-type phenotype, consisting of (i) sectoring of colonies on agar, (ii) early senescence and death of colonies, and (iii) reduced virulence when inoculated on oaks (Fig. 4). These traits were permanently displayed in non-wild isolates. A screening of 16,000 gene products in non-wild-type genotypes identified upregulation of transposons and downregulation of Crinkler (CRN) genes compared to those in the wild type. When comparisons were performed using polymorphic SSR markers, no differences were found between oak and nonoak isolates or between wild-type and non-wild-type isolates, suggesting an epigenetic rather than a purely genetic regulatory mechanism. Previous studies employing
had vastly different dynamics from that of those in California, largely because of an eradication campaign enacting host removal and burning wherever *P. ramorum* has been discovered (48). The Oregon infestation is in Curry County near the California border, but genetic analyses have shown Oregon genotypes to have originated from a probable single introduction, separate from that leading to the California forest infestation and without a clear linkage with the current Oregon nursery population (100). Eradication efforts have slowed but not stopped the spread of the Oregon infestation; new infestations up to 5 km from the nearest known prior infestation have occurred yearly. Curiously, bay laurel trees (Oregon myrtle) are not reported to sustain heavy infection in Oregon, where tanoaks are thought to be the primary infectious host (55).

**OUTLOOK AND CONCLUSIONS**

Ten years after the publication of the seminal papers that first described *P. ramorum* as a new species (124) and as the causal agent of sudden oak death in California (104), significant progress has been made in the understanding of this new pathogen and of its pathogensystems. However, a complete understanding of the ecology and epidemiology of this species still eludes us. In part, our inability to fully understand the biology and epidemiology of this organism may be due to its complexity (four evolutionary lineages unequally present in two continents), to a novel epidemiology (an airborne rather than a soilborne forest *Phytophthora* species with distinct infectious and dead-end hosts), and finally to its encounters with novel hosts resulting in unpredictable outcomes (as the sudden larch death examples show). Different lines of evidence point to an astounding ability on the part of the pathogen to increase its populations in response to favorable environmental cues as a key point to its success. It additionally appears that mild summers (as on the coast of California) and mild winters (as in the western United Kingdom and Ireland) allow for portions of its populations to survive during unfavorable seasons, providing a base for successive outbreaks. It is to be hoped that where these mild conditions do not exist for oversummering or overwintering, outbreaks will be much less severe. In order to corroborate this hypothesis, it is pivotal that the scientific community focus on identifying the region of the world where *P. ramorum* first evolved. A study of *P. ramorum* in its native environment will allow us to determine how native plants have coevolved to cope with its attacks and may provide invaluable clues on how to identify approaches to slow down its progress as an exotic pathogen in North America and Europe.

**REFERENCES**

1. Anacker B, et al. 2008. Susceptibility to *Phytophthora ramorum* in a key infectious host: landscape variation in host genotype, host phenotype, and environmental factors. New Phytol. 177:756–766.

2. Animal and Plant Health Inspection Service, United States Department of Agriculture. 2012. Domestic quarantine notices, subpart *Phytophthora ramorum*. Title 7 CFR §301.92.

3. Antia R, Regoes RR, Koella JC, Bergstrom CT. 2003. The role of evolution in the emergence of infectious diseases. Nature 426:658–661.

4. Bimpong EG, Clerk GC. 1970. Moltitility and chemotaxis in zoospores of *Phytophthora palmivora*. Ann. Bot. 34:716–724.

5. Boutet X, Vercauteren A, Heungens K, Laurent F, Chandelier A. 2010. Oospores progenies from *Phytophthora ramorum*. Fungal Biol. 114:369–378.

6. Brasier C, et al. 2004. *Phytophthora alni* sp. nov. and its variants: designation of emerging heteroploid hybrid pathogens spreading on *Alnus* trees. Mycol. Res. 108:1172–1184.

7. Brasier CM, Vettraino AM, Chang TT, Vannini A. 2010. *Phytophthora lateralis* discovered in an old growth *Chamaecyparis* forest in Taiwan. Plant Pathol. 59:595–603.

8. Brasier C. 2003. Sudden oak death: *Phytophthora ramorum* exhibits transatlantic differences. Mycol. Res. 107:258–259.

9. Brasier C, Denman S, Brown A, Webber J. 2004. Sudden oak death (*Phytophthora ramorum*) discovered on trees in Europe. Mycol. Res. 108:1108–1110.

10. Brasier C, Kirk S. 2004. Production of gametangia by *Phytophthora ramorum* in vitro. Mycol. Res. 108:823–827.

11. Brasier C, Webber J. 2010. Plant pathology: sudden larch death. Nature 466:824–825.

12. Brown LB, Allen-Diaz B. 2009. Forest stand dynamics and sudden oak death: mortality in mixed-evergreen forests dominated by coast live oak. For. Ecol. Manage. 257:1271–1280.

13. Cherubini P, Dobbertin M, Innes JL. 1998. Potential sampling bias in long-term forest growth trends reconstructed from tree rings: a case study from the Italian Alps. For. Ecol. Manage. 109:103–118.

14. Chimento A, Cacciola SO, Garbelotto M. 2012. Detection of mRNA by reverse-transcription PCR as an indicator of viability in *Phytophthora ramorum*. For. Pathol. 42:14–21.

15. Cobb R, Lynch SC, Meentemeyer R, Rizzo DM. 2008. Five years of monitoring infection and mortality in redwood tanoak forests, p 215–218. In Frankel SJ, Kliejunas JT, Palmieri KM (ed), Proceedings of the sudden oak death third science symposium. USDA-Forest Service Pacific Southwest Research Station, Albany, CA.

16. Cobb R, Meentemeyer R, Rizzo D. 2010. Apparent competition in canopy trees determined by pathogen transmission rather than susceptibility. Ecology 91:327–333.

17. Cobb RC, Filipe JAN, Meentemeyer RK, Gilligan CA, Rizzo DM. 2012. Ecosystem transformation by emerging infectious disease: loss of large tanoak from California forests. J. Ecol. 712–722.

18. Cobb R, Chan M, Meentemeyer R, Rizzo D. 2012. Common factors drive disease and coarse woody debris dynamics in forests impacted by sudden oak death. Ecosystems 15:242–255.

19. Collins B, Parke J, Lachenbruch B, Hansen E. 2009. The effects of *Phytophthora ramorum* infection on hydraulic conductivity and tylosis formation in tanoak sapwood. Can. J. For. Res. 39:1766–1776.

20. COMTF. 2007. California Oak Mortality Task Force report, November 2007. University of California, Berkeley, Center for Forestry, Berkeley, CA, http://www.suddenoakdeath.org/.

21. Conrad A, Bonello P, Mcpherson BA, Wood D, Opiyo S, Mori S. Metabolite profiling to predict resistance to *Phytophthora ramorum* in natural populations of coast live oak. In Frankel SJ, Palmieri KM, Alexander J (ed), Proceedings of the Fifth Sudden Oak Death Science Symposium, 19 to 22 June 2012, Petaluma, CA, USDA-Forest Service, Albany, CA, in press.

22. Croucher P, Mascheretti S, Garbelotto M. Combining field epidemiological information and genetic data to comprehensively reconstruct the invasion genetics of the sudden oak death agent *Phytophthora ramorum* (Stramenopila: Oomycetes) in California. In Frankel SJ, Palmieri KM, Alexander J (ed), Proceedings of the Fifth Sudden Oak Death Science Symposium, 19 to 22 June 2012, Petaluma, CA, USDA-Forest Service, Albany, CA, in press.

23. Cushman J, Meentemeyer R. 2008. Multi-scale patterns of human activity and the incidence of an exotic forest pathogen. J. Ecol. 96:766–776.

24. Dart NL, Chastagner GA, Rugarter EF, Riley KL. 2007. Recovery frequency of *Phytophthora ramorum* and other *Phytophthora* spp. in the soil profile of ornamental retail nurseries. Plant Dis. 91:1419–1422.

25. Davidson JM, Wickland AC, Patterson HA, Falk KR, Rizzo DM. 2005. Transmission of *Phytophthora ramorum* in mixed-evergreen forest in California. Phytopathology 95:587–596.

26. Davidson JM, Patterson HA, Wickland AC, Fichtner EJ, Rizzo DM.
2011. Forest type influences transmission of *Phytophthora ramorum* in California oak woodlands. Phytopathology 101:492–501.

27. Davidson J, Patterson H, Rizzo D. 2008. Sources of inoculum for *Phytophthora ramorum* in a redwood forest. Phytopathology 98:860–866.

28. Davidson J, et al. 2004. Evidence for aerial transmission of *Phytophthora ramorum* among *Quercus* and *Lithocarpus* in California woodlands, p 108–114. In McComb JA, Hardy GE, Tommerup IC (ed), Phytophthora in forests and natural ecosystems. Proceedings of 2nd International IUFRO Working Party 7.02.09 Meeting, Albany, W. Australia. 20th Sept.–5th Oct. 2001. Murdoch University Print, Perth, Australia.

29. Davidson J, Werres S, Garbelotto M, Hansen E, Rizzo D. 2003. Sudden oak death and associated diseases caused by *Phytophthora ramorum*. PHP doi:10.1094/PHP-2003-0707-01-DG.

30. Davis F, Borchert M, Meentemeyer R, Flint A, Rizzo D. 2010. Pre-impact forest composition and ongoing tree mortality associated with sudden oak death in the Big Sur region, California. For. Ecol. Manage. 259:2342–2354.

31. Dileo M, Bostock R, Rizzo D. 2009. *Phytophthora ramorum* does not cause physiologically significant systemic injury to California bay laurel, its primary reservoir host. Phytopathology 99:1307–1311.

32. Dodd R, et al. 2005. Is variation in susceptibility to *Phytophthora ramorum* correlated with population genetic structure in coast live oak (*Quercus agrifolia*)? New Phytol. 165:203–214.

33. Dodd R, et al. 2008. Evidence for the role of synchronicity between host phenology and pathogen activity in the distribution of sudden oak death canker disease. New Phytol. 179:505–514.

34. Dybicz B, Kleczkowski A, Gilligan CA. 2009. Modelling control of epidemics spreading by long-range interactions. J. R. Soc. Interface 6:941–950.

35. EFSA Panel on Plant Health. 2011. Scientific opinion on the pest risk analysis on *Phytophthora ramorum* prepared by the FP6 project RAFFRA. EFSA J. 9:2186–2292. doi:10.2903/j.efsa.2011.2186.

36. Elliott M, et al. 2011. Phenotypic differences among three clonal lineages of *Phytophthora ramorum*. For. Pathol. 41:7–14.

37. Englander L, Tooley P. 2003. Plant hosts in the nursery industry - PLANTS MOVE! How might the movement of plants in the nursery industry contribute to the spread of *Phytophthora ramorum* to new areas? Sudden Oak Death Online Symposium. www.scientificsocieties.org/aps/proceedings/sodplengmails.pdf.

38. Erwin DC, Ribeiro OK. 1996. *Phytophthora* diseases worldwide. APS Press, St. Paul, MN.

39. Eyer C, Kozanitas M, Garbelotto M. Population dynamics of aerial and terrestrial populations of *Phytophthora ramorum* in a California watershed under different climatic conditions. In Frankel SJ, Palmieri KM, Alexander J (ed), Proceedings of the Fifth Sudden Oak Death Science Symposium, 19 to 22 June 2012, Petaluma, CA. USDA-Forest Service, Albany, CA, in press.

40. Fichtner E, Lynch S, Rizzo D. 2009. Survival, dispersal, and potential soil-mediated suppression of *Phytophthora ramorum* in a California redwood-tanoak forest. Phytopathology 99:608–619.

41. Fichtner EJ, Lynch SC, Rizzo DM. 2007. Detection, distribution, sporation, and survival of *Phytophthora ramorum* in a California redwood-tanoak forest soil. Phytopathology 97:1366–1375.

42. Fry WE, Goodwin SB. 1997. Re-emergence of potato and tomato late blight in the United States. Plant Dis. 81:1349–1357.

43. Garbelotto M, et al. 2002. Non-oak native plants are main hosts for sudden oak death pathogen in California. Calif. Agric. 57:18–23.

44. Garbelotto M, Hayden K, Swain S, Schmidt D. Long-term monitoring of *P. ramorum* inoculum identifies spatio-temporal patterns of pathogen sporation and proves that selective bay removal reduces risk of oak infection. In Frankel SJ, Palmieri KM, Alexander J (ed), Proceedings of the Fifth Sudden Oak Death Science Symposium, 19 to 22 June 2012, Petaluma, CA. USDA-Forest Service, Albany, CA, in press.

45. Garbelotto M, Rizzo D. 2005. A California-based chronological review (1995–2004) of research on *Phytophthora ramorum*, the causal agent of sudden oak death. Phytopathol. Mediterr. 44:127–143.

46. Garbelotto M, Schmidt D. 2009. Phosphonates controls sudden oak death pathogen for up to 2 years. Calif. Agric. 63:10–17.

47. Garbelotto M, Sivira P, Rizzo D. 2001. Sudden oak death syndrome kills 3 oak species. Calif. Agric. 55:9–19.

48. Goheen E, et al. 2004. An eradication strategy for *Phytophthora ramorum* in Oregon coastal forests. Phytopathology 94:535.
Minireview

73. Kroon LPNM, Brouwer H, de Cock AWAM, Govers F. 2012. The genus Phytophthora. Pac. Northw. Sci. 82:110–114.

74. Kullman H, Varner JM. 2010. The effects of sudden oak death on foliar moisture content and crown fire potential in tanoak. For. Ecol. Manage. 259:2103–2110.

75. Lane CR, et al. 2003. First outbreak of Phytophthora ramorum in England, on Viburnum tinus. Plant Pathol. 52:314.

76. Linzer R, Rizzo D, Cacciola S, Garbelotto M. 2009. AFLPs detect low genetic diversity for Phytophthora ramoromora and P. pseudozymogena in the US and Europe. Mycol. Res. 113:298–307.

77. Liu D, Kelly M, Gong P, Guo Q. 2007. Characterizing spatial–temporal tree mortality patterns associated with a new forest disease. For. Ecol. Manage. 253:220–231.

78. Maloney P, Lynch S, Kane S, Jensen C, Rizzo D. 2005. Establishment of an emerging generalist pathogen in redwood forest communities. J. Ecol. 93:899–905.

79. Manter DK, Kolody EH, Hansen EM, Parke JL. 1990–2030. Ecosphere 2:art17. doi:10.1890/ES10-00192.1.

80. Meentemeyer RK, et al. 2008. Migration patterns of the emerging plant pathogen Phytophthora ramorum on the West Coast of the United States of America. Phytopathology 99:739–749.

81. Prospero S, Hansen E, Grunwald NJ, Winton L. 2005. Survival of sudden oak death pathogen Phytophthora ramorum in Oregon from 2001 to 2004. Mol. Ecol. 16:2958–2973.

82. Prospero S, Grunwald NJ, Winton LM, Hansen EM. 1999. Migration patterns of the emerging plant pathogen Phytophthora ramorum on the West Coast of the United States of America. Phytopathology 89:231–238.

83. Ramage BS, Forrestel AB, Moritz MA, O’Hara KL. 2012. Sudden oak death disease progression across two forest types and spatial scales. J. Veg. Sci. 23:151–163.

84. Ramage BS, O’Hara KL, Forrestel AB. 2011. Forest transformation resulting from an exotic pathogen: regeneration and tanoak mortality in coast redwood stands affected by sudden oak death. Can. J. For. Res. 41:763–772.

85. Rizzo DM, Garbelotto M, Davidson JM, Slaughter GW, Koike ST. 2002. Phytophthora ramorum as the cause of extensive mortality of Quercus spp. and Lithocarpus densiflorus in California. Plant Dis. 86:205–214.

86. Rizzo DM, Garbelotto M, Hansen EM. 2005. Phytophthora ramorum: integrative research and management of an emerging pathogen in California and Oregon forests. Annu. Rev. Phytopathol. 43:309–335.

87. Rizzo D, Garbelotto M. 2003. Sudden oak death: endangering California and Oregon forest ecosystems. Front. Ecol. Environ. 1:197–204.

88. Shishkoff N. 2007. Persistence of Phytophthora ramorum in soil mix and roots of nursery ornamentals. Plant Dis. 91:1245–1249.

89. Stamm E, Parke J. The effect of Phytophthora ramorum on the physiology and xylem function of young tanoak trees. In Frankel SJ, Palmieri KM, Alexander J (ed), Proceedings of the Fifth Sudden Oak Death Science Symposium, 19 to 22 June 2012, Petaluma, CA. USDA-Forest Service, Pacific Southwest Research Station, Albany, CA. in press.

90. Swain S, et al. 2006. Composting is an effective treatment option for sanitization of Phytophthora ramorum-infected plant material. J. Appl. Microbiol. 101:815–827.

91. Swiecki TJ, Bernhardt E. Diagnosis and management of Phytophthora ramorum in California forests. For. Ecol. Manage. 259:2248–2255.

92. Tooley P, Kyde K, Englander L. 2004. Susceptibility of selected ericaeaceous ornamental host species to Phytophthora ramorum. Plant Dis. 88:993–999.

93. Tyler B, et al. 2006. Phytophthora genome sequences uncover evolutionary origins and mechanisms of pathogenesis. Science 313:1261–1266.

94. Van Poucke K, Heungens K, Franceschini S, Webber J, Brasier C. Phytophthora ramorum in Europe: a research update. In Frankel SJ, Palmieri KM, Alexander J (ed), Proceedings of the Fifth Sudden Oak Death Science Symposium, 19 to 22 June 2012, Petaluma, CA. USDA-Forest Service, Pacific Southwest Research Station, Albany, CA. in press.

95. Vercauteren A, et al. 2010. Clonal expansion of the Belgian Phytophthora ramorum populations based on new microsatellite markers. Mol. Ecol. 19:92–107.

96. Vercauteren A, et al. 2011. Aberrant genome size and instability of Phytophthora ramorum oospore progeny. Fungal Genet. Biol. 48:437–443.
120. Vettraino AM, Hüberli D, Garbelotto M. 2008. Phytophthora ramorum infection of coast live oak leaves in Californian forests and its capacity to sporulate in vitro. Australas. Plant Pathol. 37:72–73.

121. Waring K, O’Hara K. 2008. Redwood/tanoak stand development and response to tanoak mortality caused by Phytophthora ramorum. For. Ecol. Manage. 255:2650–2658.

122. Webber JF, Mullett M, Brasier CM. 2010. Dieback and mortality of plantation Japanese larch (Larix kaempfieri) associated with infection by Phytophthora ramorum. New Dis. Rep. 22:19.

123. Werres S, Marwitz R. 1997. Unbekannte Phytophthora. Deutscher Gartenbau 21:1166–1168.

124. Werres S, et al. 2001. Phytophthora ramorum sp. nov., a new pathogen on Rhododendron and Viburnum. Mycol. Res. 105:1155–1165.

125. Werres S, Zielke B. 2003. First studies on the pairing of Phytophthora ramorum. Z. Pflanzenk. Pflanzen. 110:129–130.

126. Werres S, De Merlier D. 2003. First detection of Phytophthora ramorum mating type A2 in Europe. Plant Dis. 87:1266.

127. Wickland A, Jensen C, Rizzo D. 2008. Geographic distribution, disease symptoms and pathogenicity of Phytophthora nemorosa and Phytophthora pseudosyringae in California, U. S. A. Forest Pathol. 38:288–298.

128. Widmer TL. 2009. Infective potential of sporangia and zoospores of Phytophthora ramorum. Plant Dis. 93:30–35.