Oecologia (2005) 144: 106–114
DOI 10.1007/s00442-005-0028-1

ECOSYSTEM ECOLOGY

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Examining the strength and possible causes of the relationship between fire history and Sudden Oak Death

Received: 11 June 2004 / Accepted: 26 January 2005 / Published online: 11 May 2005

Abstract Fire can be a dominant process in the ecology of forest vegetation and can also affect forest disease dynamics. Little is known about the relationship between fire and an emerging disease epidemic called Sudden Oak Death, which is caused by a new pathogen, *Phytophthora ramorum*. This disease has spread across a large, fire-prone portion of California, killing great numbers of oaks and tanoaks and infecting most associated woody plants. Suitable hosts cover a much broader geographic range, raising concern over where the disease may spread. To understand the strength and potential sensitivities of a fire–disease relationship, we examined geographic patterns of confirmed *P. ramorum* infections in relation to past fire history. We found these infections to be extremely rare within the perimeter of any area burned since 1950. This finding is not caused by spatial bias in sampling for the disease, and is robust to variation in host abundance scenarios and to aggregation of closely spaced sampling locations. We therefore investigated known fire-related factors that could result in significantly lower incidence of the disease in relatively recently burned landscapes. Chemical trends in post-fire environments can influence the success of pathogens like *P. ramorum*, either by increasing plant nutrient stress or by reducing the occurrence of chemicals antagonistic to *Phytophthoras*. Succession in the absence of fire leads to greater abundance of host species, which will provide increased habitat for *P. ramorum*; this will also increase intraspecific competition where these trees are abundant, and other density-dependent effects (e.g. shading) can reduce resource allocation to defenses. Despite these findings about a fire–disease relationship, a much deeper understanding is necessary before fire can be actively used as a tool in slowing the epidemic.

Keywords Epidemiology · Fire history · Landscape pattern · Plant disease ecology · *Phytophthora ramorum* · Spatial analysis

Communicated by Alan Knapp

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Introduction

Understanding the landscape ecology of a disease is challenging, due to complex interactions between pathogens and hosts at a variety of spatial and temporal scales (Holdenrieder et al. 2004). Sudden Oak Death is a new disease in forests and woodlands that has spread rapidly since 1995, reaching epidemic proportions along a ~300-km stretch of the central coast of California (Fig. 1). The disease was also recently found in Oregon (Goheen et al. 2002), and is a growing concern outside the United States (Brasier 2003; Ivors et al. 2004). It is caused by a newly discovered pathogen, *Phytophthora ramorum*, which has already caused the mortality of vast numbers of oaks (*Quercus* spp.) and tanoak or chestnut oak (*Lithocarpus densiflorus*) in California forests.

The level of mortality that will eventually occur due to Sudden Oak Death remains uncertain. Observations thus far, the nature of the pathogen, and the slow regeneration time for oak forests suggest that the ecological consequences of the disease will be substantial. *Phytophthoras* are considered the most devastating pathogens of dicotyledonous plants (Kamoun 2000), and they have been the cause of especially significant plant disease epidemics. For crop plants, the blight that resulted in the Irish potato famine is a most notable example. In many cases, natural vegetation has also been severely impacted. *P. cinnamomi* was introduced to Australia, causing an epidemic that has destroyed tens of

thousands of hectares of valuable jarrah (*Eucalyptus marginata*) forests (Weste and Marks 1987). In western North America, *P. lateralis* has decimated many stands of a forest-dominant, Port-Orford-cedar (Hansen et al. 2000). Phylogenetic research places *P. ramorum* very close to *P. lateralis* (Rizzo et al. 2002; Martin and Tooley 2003; Ivors et al. 2004), and their geographic origin may coincide (Rizzo et al. 2002). Genetic studies, the rapidly expanding epidemic, and the mortality of tanoaks all strongly suggest that *P. ramorum* is not native in western North America. Due to a lack of evolved defenses, non-native pathogens may have tragic long-term effects. The textbook example is the closest North American relative to tanoak, the American chestnut, once widespread and abundant but now virtually extinct due to an Asian fungus.

Declines of tanoaks and oaks due to *P. ramorum* will affect hundreds of vertebrates and invertebrates that consume en masse the rich source of food these dominant trees produce. For example, one study estimated annual acorn production of a single mature tanoak to be 455 kg (Tappeiner et al. 1990). Susceptible trees provide many other resources and may also be linked into diffuse mutualisms involving mycorrhizal fungi and other co-occurring plants (Egerton-Warburton and Allen 2001). Thus, Sudden Oak Death may cause a cascade of long-term ecological impacts in affected ecosystems (Rizzo and Garbelotto 2003). The disease has recently been identified as a serious potential threat to the habitat requirements for the Northern Spotted Owl (*Strix occidentalis caurina*), perhaps the most important endangered species in the Pacific Northwest in terms of conservation efforts (Courtney et al. 2004).

Fire influences many aspects of ecosystems, and past fire occurrence has been hypothesized to reduce the susceptibility of natural vegetation to diseases (e.g. Reaves et al. 1990; Schwartz et al. 1995). Fire suppression has been effective in areas where Sudden Oak Death emerged, suggesting that an absence of fire may have created conditions favoring the disease (Swiecki 1999; Rizzo et al. 2002; Rizzo and Garbelotto 2003). In addition, infections have been observed less frequently in previously burned areas (K. Fischer, E. Sanderson, S. Strindberg, G. Woolmer, C. Blomquist and N. M. Kelly, unpublished work), and prescribed fire has been credited with reducing the disease in certain locations (P. Robards, California State Parks, personal communication). The strength of the fire–disease relationship has not been analyzed, however, nor have fire-related mechanisms been considered thus far in the disease dynamics of Sudden Oak Death. The goal of our work has therefore been to examine in detail the spatial patterns of fire history and infection locations and to identify possible causes of a relationship. In our analyses, we addressed scenarios of varying host abundance and potential spatial dependence in sampling locations, and tested for possible sampling bias for infections occurring inside and outside burns. We also investigated known fire-related mechanisms to explain how past fires could be affecting the pathogen, its hosts, and the environment in which they interact.

Materials and methods

Phytophthora ramorum infections

P. ramorum causes large lesions to develop on the main stem of oaks, especially tanoak (Rizzo and Garbelotto 2003). When lesions appear on these trees, death often follows quickly. This disease may be compounded by opportunistic bark beetles, which can hasten mortality of infected trees and disperse to uninfected trees, potentially causing additional mortality through positive feedback effects (Švihra 1999). As of 2001, stand-level mortality had reached 22% in tanoak and 15% in coast live oak, the two species most susceptible to death, and many more trees were infected in these stands (Swiecki and Bernhardt 2002).

P. ramorum also causes foliar infections, which occur in tanoak and many of the woody plant species within the current range of the epidemic, but not in true oaks. Plants susceptible to foliar infections include such well-known and economically important trees as redwood (*Sequoia sempervirens*) and Douglas-fir (*Pseudotsuga menziesii*) (Davidson et al. 2002; Maloney et al. 2002). Foliar infections can apparently kill the forest dominant, Pacific madrone (*Arbutus menziesii*) and native rhododendrons (Goheen et al. 2002; Rizzo and Garbelotto 2003). Long-term, physiological stress caused by foliar infections could also threaten host growth rates, fecundity, and resistance to other diseases or insects (Brasier 1999). Infected foliage can produce and release inoculum in large quantities, strong evidence that foliar hosts may play an important role in the epidemiology of *P. ramorum* diseases (Rizzo and Garbelotto 2003; Davidson et al. 2005).

As a generalist pathogen, there is great concern that *P. ramorum* may spread and infect forests far beyond its current range. The distribution of known hosts in California and the Pacific Northwest suggests that suitable habitat exists over an extensive portion of the western US and beyond (Rizzo et al. 2002). The recent distribution of infected plants from California nurseries has, unfortunately, made a rapid range expansion a more realistic possibility (Stokstad 2004). Stand-level variables have generally not been found to be good predictors of disease occurrence (Swiecki and Bernhardt 2002), although factors such as the distance to forest edge and a topographic moisture index have been used to model disease patterns at the landscape scale (Kelly and Meentemeyer 2002). Recent predictions of the potential for spread based on climate and host data reflect that moister and more humid environments are at greatest risk (Meentemeyer et al. 2004; Guo et al. 2005). In addition, host susceptibility to infection does not appear to be ameliorated by favorable moisture status (Swiecki and Bernhardt 2002). A difficulty in associating

landscape-level factors with spread of the disease has been the known versus perceived distribution of the pathogen. Other pathogens can cause Sudden Oak Death symptoms, so it is necessary to isolate *P. ramorum* from hosts to confirm infections and their locations (Rizzo et al. 2002).

Spatial data characteristics

We obtained the survey of confirmed *P. ramorum* infection points as of October 2002, reported through the California Oak Mortality Task Force (COMTF). In addition to the date of testing, the COMTF database contains the host species and the geographic coordinates of its location obtained from a global positioning system (Kelly and Tuxen 2003). To delimit our study area, we used the latitudes of the northern- and southern-most documented points in California and the distance of the farthest inland point (61 km) from the Pacific coast (Fig. 1).

The fire history data used in this study were acquired from the California Department of Forestry and Fire Protection (CDF). CDF maintains a database of wildfire perimeters in wildlands across California (http://frap.cdf.ca.gov/data/frapgisdata/select.asp). These data were merged with a supplemental coverage of known management burns also obtained from CDF. Although the available metadata description does not claim complete coverage of wildfires less than 121.5 ha (300 acres) on private and state lands and 4 ha (10 acres) on U.S. Forest Service lands, many mapped fires smaller than these thresholds are included. The omission of some smaller fires is unlikely to influence our analyses, as larger fires overwhelmingly dominate in terms of area burned in the study site. In addition, many unmapped small fires have occurred in areas that also burned in larger, mapped fires and were therefore accounted for in our analyses (Fig. 1).

The CDF data set is not complete for burning that occurred prior to 1950, so we restricted analyses to fires occurring after this point. Although somewhat arbitrary, this cutoff results in landscapes that are relatively recently burned versus the remaining areas, which are considered long unburned (i.e., at least 50 years). The most recent fires used in analyses were from the year 2000, the year before the first infection records from the COMTF database.

Statistical analyses

The COMTF infection database is the result of sampling for *P. ramorum* on an ad hoc basis by a variety of people and agencies, driven largely by observation of symptomatic plants at different locations. After symptomatic tissue samples were collected and sent to designated testing labs, those samples testing positive for *P. ramorum* typically became part of the COMTF database. It is

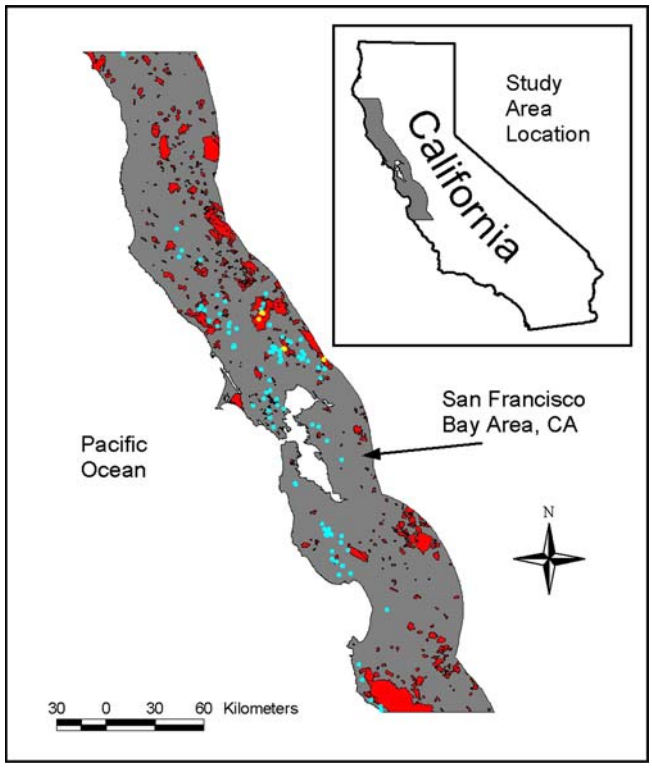

Fig. 1 Study area limits. The extent of the entire domain (~450 km in latitude by 61 km inland) is indicated by the *gray region*. The complete 1950–2000 mapped fire history is shown in *red*, and all confirmed infection points are in *blue*, except for those falling into past burns (shown in *yellow*; in a complex of 1964 fires). Only those infections and areas burned that were in susceptible vegetation types were included in the analyses

therefore possible that an observed relationship between infection locations and past fire history could be strongly influenced by artifacts of sampling or how one defines susceptible portions of the landscape for analyses. To address this concern, we examined the robustness of the relationship under two different sampling scenarios and three susceptible landscape definitions, and we also tested for spatial bias in the original sampling effort with respect to past fire locations.

Because plant diseases can result in clustered infections and samples were sometimes collected in close proximity, we aggregated infection locations to create an alternative subsampling scenario that addressed possible spatial dependence in observations. This was accomplished in a geographic information system (GIS) by generating a buffer around each point, merging any overlapping buffer regions together, and then generating the geographic centroid of the resulting area. Based on published findings about clustering of *P. ramorum* symptoms at a spatial scale of 100–300 m (Kelly and Meentemeyer 2002), we chose a buffer distance of 400 m. We refer to the aggregated scenario observations as infection sites, which actually reflect a conservative 800-m distance criterion. Excluding nurseries, $n = 259$ for the total number of positive infection points in the study area, and a greatly reduced $n = 90$ for sites.

To concentrate our analyses on landscapes in which natural fire–disease dynamics would be evident, we avoided areas dominated by human land uses (e.g., urban and agricultural). To create alternative host abundance scenarios, we restricted the domain of analysis based on the dominance of natural vegetation types using data from the California Gap Analysis Project (Davis et al. 1998). Gap vegetation data employed a minimum mapping unit of 100 ha and ranked up to three dominant vegetation types by their extent within each mapped unit. This classification allowed for varying scenarios of host abundance, based on the primary, secondary, and tertiary dominants of each mapped unit in the database. The vegetation types are from the Wildlife Habitat Relationship (WHR) classification developed for mapping California's vegetation (Parker and Matyas 1981). Mixed evergreen, redwood, and coastal oak forests and woodlands, as well as mixed oak/chaparral WHR types dominated the host abundance scenarios we assessed. We selected these WHR types because tanoak, coast live oak, and California bay are widespread and abundant in them. We also included blue oak woodlands, as coast live oak and other *P. ramorum* hosts are common in this vegetation type within the study area.

We used three levels of screening to generate landscape susceptibility scenarios: L1 specifies that susceptible vegetation dominates the primary, secondary, or tertiary cover types in mapped units; L2 requires that either the primary or secondary cover types are susceptible; and L3 means that the primary cover must be susceptible vegetation. This yields a gradient of increasingly restrictive screens based on host abundance (Fig. 2), progressively excluding locations that could fall in or near mixed urban and agricultural landscapes.

We tested the null hypothesis that the proportion of infections in previously burned areas is not lower than would be expected by chance. The expected value was based on the proportion of each landscape susceptibility scenario that had burned since 1950. We tested for differences in hypothesized proportions using the normal approximation to the binomial test, which is appropriate for large sample sizes (Zar 1999). The probability that an observed proportion is the same as expected is derived from the portion of a normal distribution more extreme than a normal deviate Z, given by the normal approximation; the test is equivalent to a chi-square goodness of fit test for hypothesized proportions (Zar 1999). We applied this test for both point and site observation scenarios and in each of the three landscape scenarios of host abundance, L1–L3.

To account for a possible effect of sampling bias resulting from accessibility to burned vs. unburned areas and other factors, we analyzed the spatial distribution of sampling locations, regardless of infection status (i.e., both positive and negative for *P. ramorum*). Samples testing negative were acquired from the California Department of Food and Agriculture (CDFA), whose data comprise a subset of the COMTF database, over

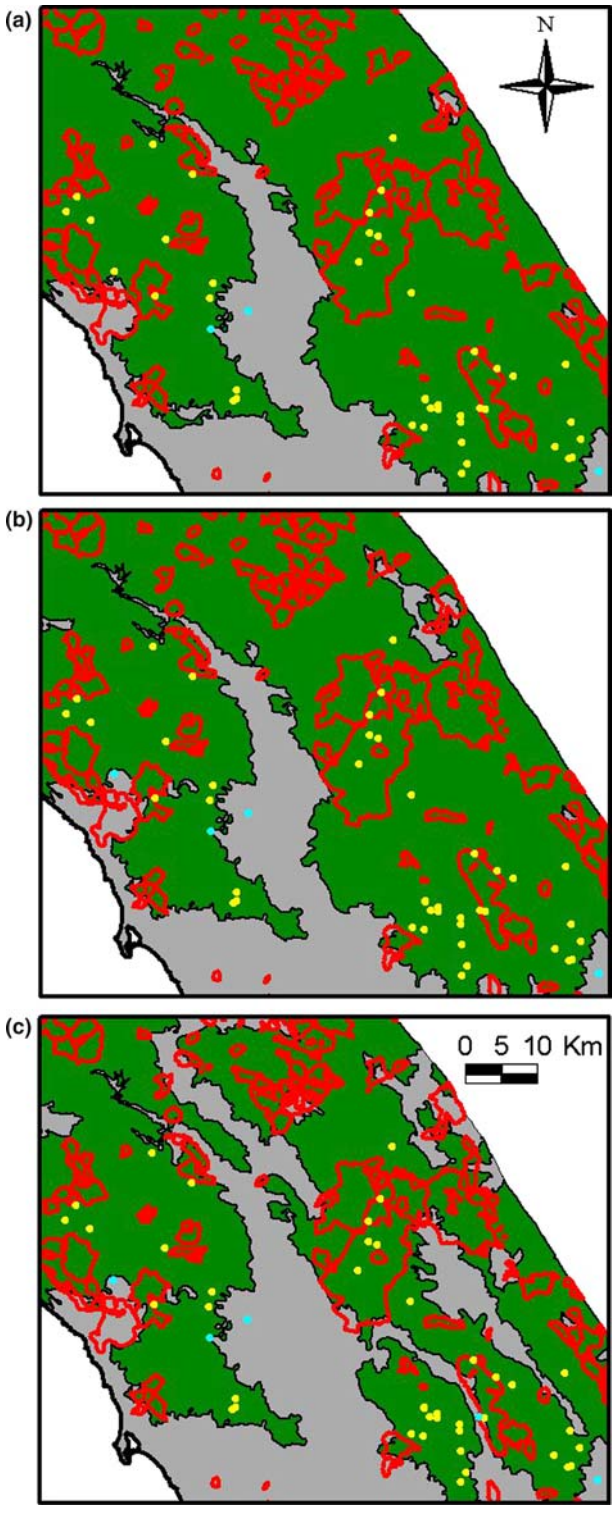

Fig. 2 Example of vegetation screens. The region shown here is in Sonoma County, north of the San Francisco Bay Area. These panels demonstrate how the gradient in host abundance generated different landscape susceptibility scenarios (in *green*). Increasingly restrictive vegetation screens in panels **a**, **b**, and **c** correspond to L1, L2, and L3, respectively. Whether infection locations are included (*yellow points*) or excluded (*blue points*) from analyses depends on the scenario; the same applies to burned areas (perimeters in *red*)

the same time period of analyses. While geographic coordinates were collected for all COMTF positive points, only a portion of the many CDFA negatives had coordinates ($n = 124$ out of 675 total, excluding nurseries). Thus, in order to determine the overall relative sampling intensity inside and outside burns without exaggerating the contribution of CDFA positive points ($n = 66$), we weighted their contribution according to their representation in the whole CDFA dataset. The resulting measure of sampling intensity for symptomatic plants inside and outside burns was then used to test the hypothesis that sampling inside and outside of burns was not different. This was done for each combination of observation scenarios (i.e., points and sites) and susceptible landscape scenarios (i.e., L1–L3), again using the normal approximation to the binomial test.

Results

Species composition of infection hosts was similar in both the COMTF and CDFA datasets, and the ratio of foliar to stem hosts was about three-to-two in each. California bay dominates the sampling database, including those locations from previously burned areas, followed by coast live oak and tanoak. Remaining species accounted for less than 10 percent of the total in each data set.

Based on the prevalence of susceptible hosts in the natural vegetation of the study area, the screening process resulted in the following reductions to the potential infection space for each susceptibility scenario: landscape scenarios L1, L2, and L3 represented 69 percent, 64 percent, and 49 percent of the entire study area, respectively. The amounts burned since 1950 in L1, L2, and L3 were 14.6 percent, 13.8 percent, and 13.1 percent, respectively, comprising the ''expected'' percentages used in statistical tests. These percentages resulted from 866 fires and ~367,000 ha that had burned at least once over the 1950–2000 period.

The number of COMTF positive infection samples found in burns was far fewer than expected by chance, for all scenario combinations (Fig. 3a). Moreover, the likelihood of obtaining such divergence from expected values is exceptionally low. Even for landscape L3, the most spatially restrictive scenario, the number of positive sample points found in burns was significantly fewer than expected by chance at $\alpha = 0.001$. Probabilities of observing an infection in past burns increased when adjacent points were merged into sites, reducing numbers of observations, but they were all still statistically significant at $\alpha = 0.05$ (Fig. 3a). It is also worth noting that these results were robust to further restrictions on the definition of a susceptible landscape (i.e., omitting blue oak woodlands, which could have lower host densities; data not shown). Results from the scenarios analyzed here are evidence for a strong and consistent negative relationship between infections and fire history.

Unlike the COMTF positive samples, the percentage of all CDFA samples (i.e., both positive and negative) in past burns was not lower than expected. In fact, the percentage of all CDFA sample points from burned areas was somewhat *more* than expected, and significantly more so in the case of landscape L3 (Fig. 3b). Sampling sites were distributed in almost the exact proportions expected by random placement on the landscapes (Fig. 3b). This indicates that the overall sampling effort was generally unbiased with respect to fire history, or tended to be somewhat biased toward burned areas, and that under-sampling in previously burned areas does not explain the lack of infections found there.

Remarkably, the few positive samples that were found inside the 1950–2000 burn perimeters were all in a

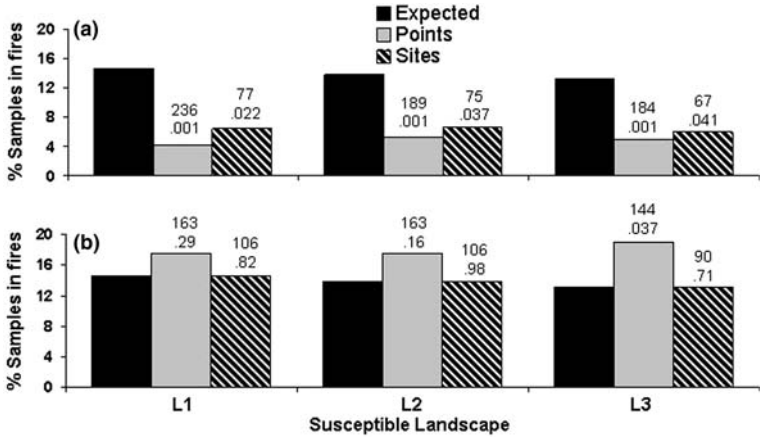

Fig. 3 Proportion of samples observed in past burns. Results for points and sites in previously burned areas are tested against the amount expected, based on the proportion of each landscape burned from **a** COMTF samples testing positive and **b** CDFA samples regardless of infection status. The total number of observations and the *P*-value for the hypotheses tested are shown above each bar. **a** demonstrates that there are significantly fewer infections in past burns than expected by chance ($\alpha = 0.05$) in all scenarios; **b** demonstrates that sampling in general was not biased away from past burns

complex of fires that occurred in 1964, in a relatively limited inland area (Fig. 1). Despite more area being burned after 1964 (~259,000 ha) compared to 1951–1964 (~149,000 ha), we found no samples in burns that occurred during the later period. Findings would therefore be even stronger if pre-1965 fire history data were considered "long unburned" (i.e., at least 35 years). Regardless of whether the areas burned in 1964 were anomalous, the strength of the overall fire–disease pattern is striking. The robustness of this relationship suggests that the lack of infections in previously burned areas is the result of direct or indirect fire-related factors. Given the present data, however, the almost total lack of *P. ramorum* infections in past burned areas precludes more detailed statistical analyses (e.g., trends with time since fire or between different burned plant communities).

Discussion

Our analyses demonstrate a strong and consistent negative relationship between the locations of confirmed *P. ramorum* infections and areas that have burned since 1950. Before discussing the most likely fire-related mechanisms that might cause this pattern, it is worth mentioning some of the possible limitations of our study. For example, we do not address the possible differences in timing of pathogen introduction or levels of pathogen exposure that may vary across our study area (D. Rizzo, personal communication). Another issue not investigated here is variation in the risk of infection, based on climatic factors (e.g., Meentemeyer et al. 2004; Guo et al. 2005), within our landscape susceptibility scenarios. Further research will be required to identify the specific mechanism(s) causing the pattern, to quantify how the relationship may vary along moisture gradients, and to pursue other fundamental questions that these data cannot address. Nonetheless, results appear to be insensitive to removal of potentially spatially dependent infection samples and to variation in host abundance scenarios. Moreover, the pattern is not due to under-sampling for *P. ramorum* in previously burned areas, which could have been the case for sampling that is strongly affected by the road network (e.g., Hijmans et al. 2000) or other confounding factors. Although the disease was rarely found in burns, proportionally more samples were taken from within past burns, suggesting that *P. ramorum*-like symptoms may be common in burns even when the disease is not.

It is interesting that the negative relationship between the disease and fire history holds for fires that occurred decades ago, long before *P. ramorum* is thought to have been present. Our findings therefore indicate that past fires, or something that varies closely with fire over long time periods, may strongly limit landscape-scale patterns of this expanding epidemic. Although antagonistic relationships between fire and forest diseases have been established or hypothesized in many areas (e.g., Froelich et al. 1978; Baker 1988; Reaves et al. 1990; Brennan and Hermann 1994), identification of the actual fire-related mechanisms responsible for disease inhibition in nature has remained challenging. Regardless, certain aspects of the host environments studied here and the biology of *Phytophthora* species point to specific fire-related mechanisms that can influence host susceptibility and landscape pathology (Holdenrieder et al. 2004) of Sudden Oak Death.

Post-fire chemical changes

The potential for fire to influence the growth of spores and mycelia of fungal pathogens through direct effects of heat and/or smoke has long been known (e.g., see review of Parmeter 1977). Spore and mycelial growth of *Phytophthoras* may be similarly influenced. Chemicals in smoke may persist after deposition, and they have been shown to have dramatic effects on plant pathogens. For example, smoke can inhibit spore germination and also stimulate germination when conditions are unsuitable, both of which lead to fungal pathogen mortality (Parmeter 1977). Growth of fungal hyphae can also be inhibited on substrata exposed to smoke (Parmeter 1977; Schwartz et al. 1995), and that of *Phytophthora* has been found to be sensitive to the chemicals encountered (Morris et al. 1998). Despite these effects, it does not seem likely that they would be persistent long enough after fire (i.e., for decades) to fully explain our findings.

Fire causes many direct and long-term changes in soil chemistry, particularly in calcium (Ca), magnesium (Mg), phosphorous (P), and pH (Viro 1974; Wells et al. 1979; Agee 1993). For example, there can be increases in exchangeable Ca for a few decades after a fire (Viro 1974), and Ca availability is crucial for plant resistance to parasitic diseases (Marschner 1995). Uptake of P, a macronutrient integral to virtually all biochemical and developmental processes in plants, will also decrease with time since fire, as available forms diminish in soils (Wells et al. 1979). Plant growth may become limited in very long undisturbed environments, due to a lack of P in relation to other nutrients (e.g., nitrogen; Wardle et al. 2004). Plants exhibit a variety of symptoms as a result of P deficiency, and it has been described as the cause of "senescence" of some California vegetation in the absence of fire (Rundel and Parsons 1980). Increased nutrient stresses in the long absence of fire may therefore raise susceptibility of hosts to *P. ramorum* infections.

Given fire effects on pH and nutrients, post-fire changes in soils could be limiting to the pathogen. *P. ramorum* is not currently thought to travel through soil to infect plant roots; however, it may still spread via the soil surface, and recent research indicates that the disease can occur from infected potting soil (Lewis et al. 2004). Post-fire soil environments support a variety of

microbial communities antagonistic to forest pathogens (e.g., Reaves et al. 1990), and inhibition of *Phytophthoras* could occur via changes in competition, antibiosis, or parasitism between soil-borne microorganisms (Erwin and Ribeiro 1996). Phosphate, a naturally occurring P compound that is relatively abundant after fire, has also been shown to inhibit growth of *Phytophthoras* in laboratory media (Grant et al. 1992).

An intriguing possibility is that post-fire changes in other P compounds inhibit infection success of the pathogen. Phosphite, an isostere of phosphate, is readily taken up by plants, and phosphite-based compounds have long been used as systemic fungicides to control *Phytophthoras* (Guest and Grant 1991). In fact, phosphite treatments now show promise for increasing host resistance to *P. ramorum* (Garbelotto et al. 2002). Examination of the fungicidal role of phosphites has focused on synthetic products, but natural sources can exist. Phosphite is considered too unstable to persist in soil environments; however, the ability of some plant roots to cause localized extreme changes in pH and CO_2 concentrations (Smith et al. 2003) indicates that plant rhizospheres can be chemically quite unusual. In addition, analogous to biological nitrogen cycling, microorganisms and pathways responsible for biological P cycling have recently been identified (Ohtake et al. 1996; Buckel 2001; McDonald et al. 2001). The existence of natural phosphonates (i.e., with a C–P bond) at a lower oxidation state than phosphite, in conjunction with microorganisms specialized to utilize these compounds (Ohtake et al. 1996; Buckel 2001; McDonald et al. 2001), raises questions about the natural availability of phosphite and other P compounds that can be antagonistic to *Phytophthoras*.

Vegetation succession

Because time-since-fire is a strong driver of ecosystem structure and species composition in the study area, it is likely that certain successional stages are more prone to infection than others. Succession in the absence of fire leads to greater abundance of host species, which could result in increased habitat for *P. ramorum*. As stated earlier, this trend is difficult to assess with current data, due to the almost complete lack of *P. ramorum* infections in stands of vegetation that burned at different times in recorded history. Regardless, tradeoffs over time between allocations to growth and chemical defenses can affect plant susceptibility to disease (Herms and Mattson 1992), which could contribute to the predominance of infections in long unburned areas. Many phenolic compounds play a role in pathogen resistance, and their production is known to vary with plant age (Levin 1971). The leaves of California bay trees, which can disperse large amounts of *P. ramorum* inoculum, contain a variety of phenolics (Lawrence et al. 1974). Although concentrations of these compounds in California bay leaves do not consistently decrease with plant age, some do show a downward trend (Goralka and Langenheim 1996). In addition, higher light levels can result in increased accumulation of such defensive compounds (Gershenzon and Croteau 1991), meaning that their production would tend to decrease with time-since-fire and increased shading from surrounding vegetation.

A known successional trend is also that of increased competitive stress in plants that quickly colonize or resprout in after forest disturbances (Connell 1978). For example, impacts from *P. cinnamomi* have been found to be greater with increasing vegetation height and age associated with the absence of fire (Brown et al. 2002). The covers of tanoak and coast live oak, both susceptible to *P. ramorum*, have also been found to increase with time-since-fire (Davis et al. 1988; Hunter 1997). Increased competitive stress with succession could therefore be a factor in the spread of Sudden Oak Death, as has been hypothesized for declines of Pacific madrone to the north of the study area (Elliott 2003).

Fire management

Our results suggest that areas burned in recent decades will be much less susceptible to the disease than those that have not. While fire may eventually be used in an attempt to manage the epidemic, much needs to be learned before specific recommendations can be made. The effect of fire on susceptibility to initial infection probably differs greatly from its influence on disease dynamics within already infected areas. It is also possible that deliberate fires under conditions providing easy containment will not have the same effect as past wildfires (Moritz and Odion 2004). For these reasons, and without knowledge of the causal mechanism(s) linking past fires to the absence of the disease, it is premature to suggest programs of deliberate burning.

It should be stressed that *P. ramorum* is currently thought to be a non-native pathogen (Rizzo et al. 2002), and our findings do not indicate that the epidemic is somehow caused by recent fire suppression. In fact, increased burning to limit the spread of *P. ramorum* could conflict with maintenance of natural fire regimes or compromise ecological goals (Whelan and Muston 1991; Spencer et al. 2003). In the Klamath region just north of where *P. ramorum* is concentrated in California, tanoak is especially common. Amounts of fire in this region have already been found to be increasing in recent decades (Odion et al. 2004). To the south of the range of *P. ramorum*, fire suppression has not produced fewer and larger wildfires in most coastal areas (Moritz 2003), as many assume. Instead, the region has probably seen an increase in fire frequency (Keeley et al. 1999a). There are a number of species in both regions that may be threatened by too frequent fire. Obligate seeding shrubs and serotinous conifers, in particular, can require several decades to accumulate the seed banks needed to regenerate following a fire (Zedler 1995; Keeley et al. 1999b;

Odion and Tyler 2002). Exotic species invasions following fire in these Mediterranean-climate regions are also a serious concern. Thus, increasing use of fire to combat spread of *P. ramorum* must be weighed against these and other potential ecological impacts of prescribed burning (Moritz and Odion 2004).

Acknowledgements We gratefully acknowledge financial support from the National Science Foundation (Award#EF-0341729); MAM was also partially supported by a grant from the James S. McDonnell Foundation during preparation of this manuscript. The input of K. Fischer, E. Sanderson, and others at WCS was helpful in formulating the research questions addressed here. We thank A. Schaffner for statistical advice, and C. Blomquist, T. Bruns, S. Cole, S. Frankel, M. Garbelotto, J. Keeley, A. McDonald, and D. Rizzo for reviewing drafts of the manuscript. The work performed here complies with the current laws of the United States of America.

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