Journal of Ecology

British Ecological Society

Journal of Ecology 2008, **96**, 766–776 doi: 10.1111/j.1365-2745.2008.01376.x

Multi-scale patterns of human activity and the incidence of an exotic forest pathogen

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Summary

1. Plant pathogens can have major impacts on diverse taxa and ecological systems world-wide, and some of the most conspicuous of these are invasive non-native species. Although many factors are known to influence the distribution and abundance of plant pathogens, the extent to which humans play a role is less well-known but still critical for understanding the dynamics of invasive pathogens in natural systems.

2. One invasive pathogen of great concern world-wide is *Phytophthora ramorum*, which causes Sudden Oak Death, an emerging forest disease. Here, we examined the influence of humans and a range of environmental factors on the distribution of *P. ramorum* at three distinct spatial scales in northern and central California.

3. At the local scale, *P. ramorum* more commonly occurred in soil on hiking trails used heavily by humans than in soil from adjacent areas off trails. These results support the hypothesis that humans dispersed the pathogen within already infected areas and into areas lacking local sources of inoculum.

4. At the landscape scale, using a network of 202 randomly located plots across a 275 km^2 area, we found that forests on public land open to recreation exhibited higher prevalence of disease in a critical infectious host tree (bay laurel, *Umbellularia californica*), than forests on private lands. Infection levels were also higher in plots surrounded by large amounts of forest with warm climatic conditions and greater potential soil moisture. Although prevalence of diseased canker hosts (*Quercus agrifolia, Q. kelloggii and Lithocarpus densiflora*) was positively associated with dominance of bay laurel and rainy season temperatures, it was not significantly related to public access, and we hypothesize that this occurred because our study area was in the early stages of infection.

5. At the regional scale, the probability of disease occurrence at 165 sites distributed across the geographic range of *P. ramorum* in California increased significantly as human population density increased in the surrounding area. Chances of infection also increased significantly with precipitation and presence of bay laurel.

6. *Synthesis*. Our results suggest that human activity – along with temperature, moisture and host composition – is associated with increased prevalence of an influential exotic forest pathogen. These results indicate that there may be conflicts between humans and disease, and that efforts to address this issue may require aggressive management of human activity.

Key-words: disease incidence, exotic forest pathogen, foliar and canker hosts, human activity, *Phytophthora ramorum*, Sudden Oak Death

Introduction

Pathogens are increasingly recognized to have large impacts on a wide range of species and ecological systems throughout the world. Either individually or in association with other factors, such as climate change, pathogens have been implicated in the decline, extirpation or global extinction of both terrestrial and marine species (Daszak *et al.* 2000; Harvell *et al.* 2002; Smith *et al.* 2005), and their effects can scale up to the community level (Dobson & Crawley 1994). Plant pathogens can have particularly dramatic effects on their host species and associated ecosystems, and in many cases the most destructive of these organisms are both non-native and highly invasive (Gilbert 2002; Anderson *et al.* 2004; Rizzo 2005; Burdon *et al.* 2006). For example, the chestnut blight fungus (*Cryphonectria parasitica*) was accidentally introduced into

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the United States from Asia via the nursery trade and rapidly decimated populations of the once dominant American chestnut (*Castanea dentata*) in eastern forests (Anagnostakis 1987). Another incident occurred in Western Australia, where the accidentally introduced root-feeding fungus *Phytophthora cinnamomi* caused massive dieback of Jarrah (*Eucalyptus marginata*) and substantially altered the structure and composition of these forests (Newhook & Podger 1972; Weste & Marks 1987).

Many factors influence the distribution and abundance of plant pathogens throughout their native and introduced ranges. Abiotic factors, such as temperature and moisture levels, have been shown repeatedly to be important drivers of disease establishment and spread (Burdon 1987; Davidson *et al*. 2005; Woods *et al*. 2005). Other work has demonstrated the importance of considering the abundance and spatial configuration of host and non-host vegetation in plant epidemiological studies (Burdon 1987; Burdon *et al*. 1995; Thrall *et al*. 2003; Condeso & Meentemeyer 2007). However, the extent to which humans influence the prevalence of plant pathogens – either by creating new transmission pathways for disease spread (e.g. Hansen *et al*. 2000; Jules *et al*. 2002) and/or by modifying habitat conditions that in turn affect pathogen performance (e.g. Meentemeyer *et al*. 2008a) – is less well-understood (Holdenrieder *et al*. 2004). In either case, given the continued and expanding growth, mobility and impact of human populations world-wide, it is imperative to understand how human land-use activities affect the dynamics of pathogen populations in natural communities and ecosystems (Anderson *et al*. 2004; Foley *et al*. 2005).

Currently, one of the plant pathogens of greatest concern world-wide is *P. ramorum*, a newly described (Werres *et al*. 2001) and highly invasive pathogen that causes a forest disease known as Sudden Oak Death (Rizzo *et al*. 2002, 2005; Rizzo & Garbelotto 2003). This exotic pathogen has reached epidemic levels in many coastal forests of California and south-western Oregon in the U.S., where it has already killed thousands of tanoaks (*Lithocarpus densiflora*) and trees in the red oak (*Quercus* sp.) group (Rizzo & Garbelotto 2003), and many more coastal forests face considerable risk of infection (Meentemeyer *et al*. 2004). The pathogen has also been found in Canada and 11 western European countries, where it occurs predominately on ornamental plant species in nurseries, managed gardens and semi-natural areas (Appiah *et al*. 2004; Henricot & Prior 2004).

The potential ecological and economic consequences of this invasion are considerable and potentially widespread. Extensive death of host trees in forested landscapes will result in the loss of habitat and food resources for wildlife, increased fire danger, accelerated runoff and associated soil erosion, and changes to the structure and composition of plant, animal and microbial communities (Rizzo & Garbelotto 2003; Rizzo *et al*. 2005). Local, state and federal agencies are also incurring high regulatory costs associated with monitoring, eradication and containment. Lastly, the horticultural industry has suffered significant economic losses due to quarantines and destruction of infected plants (Appiah *et al*. 2004).

Determining the factors that influence the success and prevalence of *P. ramorum* is critical for understanding the dynamics of this influential pathogen and for controlling the extent of its invasion. Susceptibility of forests to *P. ramorum* invasion is currently known to be affected by host composition (Maloney *et al*. 2005; Rizzo *et al*. 2005), landscape heterogeneity of host vegetation (Condeso & Meentemeyer 2007), historic land-cover change (Meentemeyer *et al*. 2008a), and suitability of abiotic conditions for growth and survival (Davidson *et al*. 2005). Humans are also known to play an important role in promoting the occurrence of other invasive *Phytophthora* species (Weste & Taylor 1971; Marks *et al*. 1975; Hansen *et al*. 2000; Jules *et al*. 2002), but the degree to which humans affect the prevalence of *P. ramorum* has received little attention (although see Davidson *et al*. 2005). Here, we evaluate questions at three different spatial scales that address the potential influence of humans and a range of additional factors on the distribution of *P. ramorum*: (i) is *P. ramorum* more frequently found in soil along hiking trails than in soil off trail?; (ii) do forests on public lands open to recreation exhibit more disease than private lands that are closed to the public?; and (iii) Does the probability of *P. ramorum* occurrence at sites increase with greater human population density in the surrounding forest? In examining the latter two questions, we have considered the importance of public access and human density relative to a range of environmental variables in facilitating disease establishment. Research addressing such questions is urgently needed to understand the dynamics of an influential exotic pathogen and to facilitate management efforts to control its spread.

Study system

Phytophthora ramorum (water mold, Oomycota) is a generalist pathogen that infects over 40 plant genera, including ferns, gymnosperms, monocots and dicots, with the number of known host species increasing substantially each year (Rizzo & Garbelotto 2003). Because of its recent appearance, high virulence, genetic structure and geographic distribution, *P. ramorum* is thought to be non-native in both North America and Europe (Werres *et al*. 2001; Brasier 2003; Rizzo & Garbelotto 2003), although its exact region of origin is still unknown (D. M. Rizzo, pers. comm.).

The pathogen causes symptoms that are expressed in two distinct forms: non-lethal infections in foliar hosts that can lead to the production of large numbers of dispersal spores and lethal infections in canker hosts that do not produce infectious spores (Rizzo & Garbelotto 2003). Thus, transmission of the disease is driven primarily by infection among foliar hosts, whereas canker hosts do not contribute to pathogen spread (Garbelotto *et al*. 2003; Davidson *et al*. 2005; Rizzo *et al*. 2005). The canker hosts found in our study region are coast live oak (*Quercus agrifolia*) and California black oak (*Q. kelloggii*), whereas the most dominant foliar host is bay laurel (*Umbellularia californica*). The latter is an especially important foliar host because it produces large amounts of inoculum (Davidson *et al*. 2005) and is associated

Fig. 1. A map showing the location of 202 study plots within a 275 km^2 region of eastern Sonoma County used to assess the intensity of disease caused by *Phytophthora ramorum* on public and private land. The location of plots on public land open to recreation is indicated by circles, whereas those on private lands closed to the public are indicated by triangles. The border of Fairfield Osborn Preserve, where the hiking study occurred, is indicated by a solid black line.

with oak and tanoak mortality (Kelly & Meentemeyer 2002; Maloney *et al*. 2005). Unlike other host species, tanoak develops both non-lethal foliar and twig infections as well as lethal canker infections (Maloney *et al*. 2005).

Methods

HUMAN DISPERSAL OF A FOREST PATHOGEN

To address the ability of humans to disperse *P. ramorum* in soil along hiking trails, we conducted a study during March 2003 within different habitat types at Sonoma State University's Fairfield Osborn Preserve, 50 km north of San Francisco (Fig. 1). We selected 54 sample locations distributed equally among nine 6-m trail segments that passed through one of three habitat types differing greatly in *P. ramorum* prevalence: forests dominated by coast live oak and infected bay laurel, open grasslands lacking *P. ramorum* hosts, and stands of white oak (*Q. garryana*) that also lacked hosts. The nine trail segments were separated from each other by at least 2 km. For each of these trail segments, we collected six soil samples (top 3–5 cm), half of which were obtained from the trail surface, spaced 3 m apart, and the other half were obtained from adjacent locations 2 m off trail. We collected all soil samples after recent rainfall events to ensure that the soil was moist and optimal for *P. ramorum* survival. Within 24 h of collection, we assessed all soil samples for the presence of *P. ramorum* using standard culturing techniques employed with this pathogen (Davidson *et al*. 2005; Fichtner *et al*. 2007). In the laboratory at room temperature, we immersed each soil sample in a container of deionized water and then half submerged an unblemished green D'Anjou pear in this solution to serve as bait. To reduce pesticide residues, we washed each pear carefully prior to use. After 4–5 days, we removed the pears from the soil/water solutions and, after rinsing with deionized water, allowed them to sit uncovered on trays at room temperature for an additional 4–5 days. We then collected samples from lesions on the skin of each pear and plated this material on petri dishes containing pimaricin–ampicillin–rifampicin–pentachloronitrobenzene (PARP) agar placed in dark incubators at 17.5–20 °C. PARP agar includes two antibiotics and an antifungal agent to prevent growth of competitive species of saprophytic bacteria and fungi. After 7–14 days, we examined each of these cultures and identified all *Phytophthora* species based on morphological characters (Davidson *et al*. 2003). Only *P. ramorum* was detected in our soil samples.

To analyse the distribution of *P. ramorum* in these soil samples, we used a log-linear test of conditional independence, with habitat type (infected oak/bay laurel forest, white oak forest and grassland) and sample location (on vs. off trail) as predictor variables.

LAND ACCESS AND DISEASE SYMPTOMS

To examine the degree to which disease severity was associated with public access and 12 other variables, we studied patterns of *P. ramorum* infection level on private and public lands in a 275 km^2 region in eastern Sonoma County, approximately 70 km north of San Francisco, California (Fig. 1). This region consists of a mosaic of vegetation and land-cover types, including oak woodland, redwood–tanoak forest, chaparral shrubland, annual grassland, agricultural areas and scattered residential developments. Using a geographic information system (GIS), we established a network of 202

randomly located 15 × 15 m plots in areas mapped as oak woodland on public and private lands (see Meentemeyer *et al.* 2008 a for details on our methods for randomly selecting plots). We measured the incidence of *P. ramorum* within each of our plots, 96 of which occurred in forested landscapes that were open to the public and used heavily for recreation (parks and nature preserves), and 106 occurred on private lands not accessible to the public. For each plot, we sampled the following host trees between mid-April and mid-May 2004 for symptoms of infection by *P. ramorum*: coast live oak, black oak, tanoak and bay laurel. An understanding of factors that drive infection in dominant foliar hosts, such as bay laurel, is especially key because of their documented role in promoting disease occurrence (Davidson *et al.* 2005; Maloney *et al.* 2005).

We used symptoms of infection by *P. ramorum* as an indirect and non-destructive measure of disease incidence. Symptoms of infection in bay laurel consist of spotting and necrosis on leaves, usually at the tips. In contrast, symptomatic coast live oak and black oak exhibit cankers on the bark of the main trunk that produce dark red 'bleeding' exudates and discoloration (Davidson *et al.* 2003).

For bay laurel, we evaluated the validity of this approach by collecting 15 symptomatic leaves from three to five randomly selected trees per plot and assessing them for the presence of *P. ramorum* using standard baiting and culturing techniques (Davidson *et al.* 2003). For each leaf, we excised small amounts of material from the margin of the necrotic area. This material was then plated on PARP media for up to 7 days and, as before, examined for *P. ramorum* and other *Phytophthora* species that can occur in our study area. This culturing indicated that the majority of bay laurel trees categorized as symptomatic were positive for *P. ramorum* and no other *Phytophthora* species that cause similar symptoms were detected. Thus, we are confident that disease symptoms were a reliable measure of *P. ramorum* infection.

In addition to public access, we also evaluated the importance of 12 other variables as predictors of forest disease. Given its role in inoculum production and disease transmission (Davidson *et al.* 2005), we estimated the dominance of bay laurel in each plot by determining the diameter breast height (DBH) of all bay laurel trees and calculating cumulative DBH. To account for the influence of landscape heterogeneity of host habitat (Condeso & Meentemeyer 2007), we mapped the distribution of forest habitat in a GIS using fine-scale multi-spectral imagery (ADAR, 1 m pixel resolution) and quantified the amount of forest within a 150 m area surrounding each plot as an index of habitat contiguity. We also determined the geographic location (x- and y-coordinates) of each plot to control for historical patterns of disease spread. To account for the influence of temperature and moisture conditions known to affect foliar pathogens (Woods *et al.* 2005), we used the parameter elevation regression on independent slopes model (PRISM; Daly *et al.* 1994) to estimate minimum and maximum daily temperature, relative humidity and precipitation between December and May (the time of year when *P. ramorum* is active – see Davidson *et al.* 2005). We also derived the following variables for each plot from a USGS 10 × 10 m digital elevation model using a GIS: elevation, potential solar radiation between December and May calculated using the cosine of illumination on slope model (Dubayah 1994), and the topographic moisture index (TMI) calculated as the natural log of the ratio between upslope drainage area and the slope gradient of a grid cell (Beven & Kirkby 1979; Moore *et al.* 1991). Lastly, because of its potential importance in disease establishment (Moritz & Odion 2005), we included each plot's fire history since 1950 (fire or no fire) from records compiled by the California Department of Forestry and Fire Protection. To determine the degree to which public access and other variables influenced disease severity, we used backward elimination stepwise multiple regression, with public access and the 12 environmental factors just described as predictor variables and proportion of symptomatic bay laurel and canker hosts per plot as response variables. As noted previously, tanoaks can serve as both foliar and canker hosts; here we focused only on tanoaks serving as canker hosts.

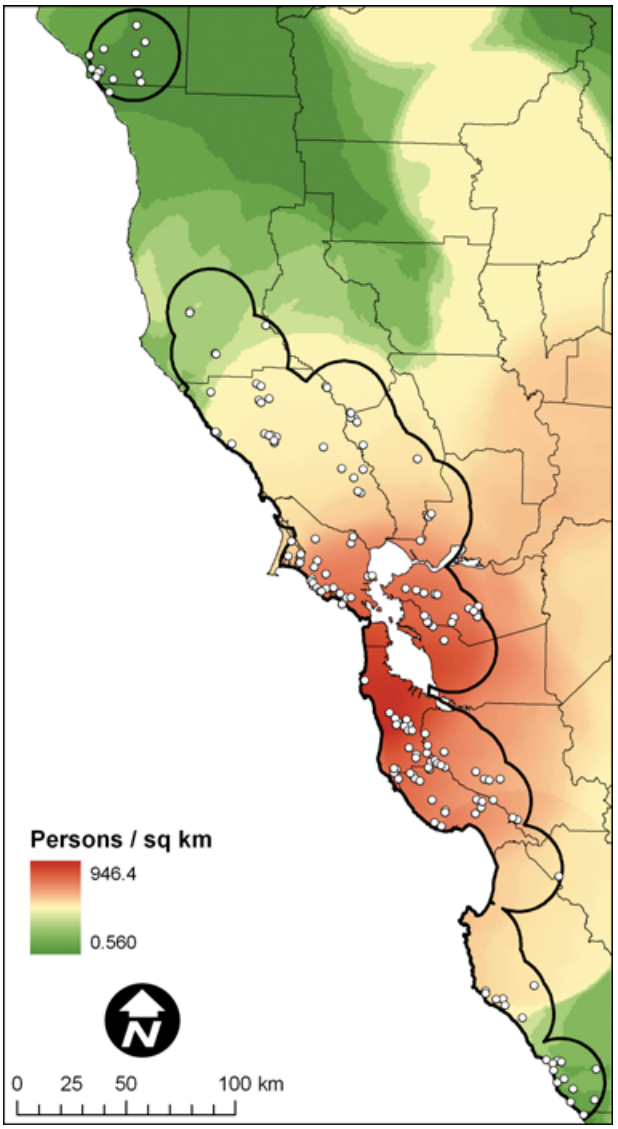

Fig. 2. A map illustrating the density of human populations across the zone of infestation in 14 counties of northern and central California where *Phytophthora ramorum* and Sudden Oak Death are known to occur. Points within this zone indicate the location of 165 sites used to assess predictors of disease incidence at the regional scale.

HUMAN POPULATION DENSITY AND DISEASE PRESENCE

To determine if disease occurrence was associated with human population density in the surrounding area or 12 other environmental variables, we first delineated a general zone of potential infestation in the 14 counties of northern and central California where *P. ramorum* and Sudden Oak Death are known to occur (Fig. 2). This was achieved by inscribing a 20 km buffer around the 275 sites officially confirmed by the

California Department of Food and Agriculture (CDFA) to contain host plants infected with *P. ramorum* as of May 2003. Isolated outbreaks of disease have rarely been detected further than 20 km from known CDFA-verified infections (Meentemeyer & Mark, unpubl. data).

Within this zone, we assessed the occurrence of *P. ramorum* at 165 randomly located forested sites on public land between May and August of 2003 and 2004. At each site, we established two 50 × 10 m intersecting belt transects (forming an 'L' shape) and visually inspected all foliar and canker host species for symptoms of *P. ramorum* infection. To test for the presence of *P. ramorum*, we collected up to 25 leaves showing symptoms of disease from five of the most symptomatic foliar host individuals. We sealed all leaf samples in plastic bags and stored them in coolers while in the field. We also assessed canker hosts for disease symptoms caused by *P. ramorum* but, to avoid injuring trees, collected canker samples only when symptomatic foliar host plants were not found. We used the same symptoms of infection for canker hosts as those outlined previously.

Symptomatic samples collected in known *P. ramorum*-infected counties were processed by the CDFA and samples collected in counties thought to be uninfected were processed at the University of California at Davis. In both laboratories, symptomatic leaf tissue was assessed for the presence of *P. ramorum* using the same standard baiting and culturing techniques (see Meentemeyer *et al*. 2008b for additional details). The identity of a subset of isolates was confirmed through sequencing the ITS region of the nuclear ribosomal DNA and comparing with voucher sequences of *P. ramorum* (Ivors *et al*. 2004). As an additional test, leaf samples that did not yield *Phytophthora* cultures were resampled with a PCR-based molecular assay using *P. ramorum*-specific primers designed to detect pathogen DNA in leaves (Hayden *et al*. 2004). We classified a site as infected by *P. ramorum* if at least one tissue sample yielded a positive culture or PCR detection of the pathogen. We classified a site as uninfected by *P. ramorum* if there was no positive isolation or PCR detection even though symptoms typical of *P. ramorum* were observed and sampled at the site, or if no symptoms typical of *P. ramorum* were observed at the site.

We quantified the human population density (persons/km^2) surrounding each site using a multi-scale nested approach to determine the spatial scale at which human population pressure might influence the presence of *P. ramorum*. To accomplish this, we used a GIS to measure the U.S. Census Bureau's 2000 block-level census in circular areas of increasing radii surrounding each of the 165 sites: 10, 30, 50 and 100 km. Since each scale resulted in significant relationships ($P < 0.05$), we chose to present the 50 km data as they yielded the strongest relationship.

In addition to human population density, we also evaluated the importance of 13 potentially influential variables as predictors of forest disease. These were elevation, geographic location (x and y-coordinates), sampling year (2004 vs. 2005), minimum and maximum daily temperature, relative humidity, precipitation, potential solar radiation, TMI and fire history. We also controlled for the presence or absence of the infectious hosts bay laurel and tanoak in each of our sites. To determine if the occurrence of *P. ramorum* was associated with human population density, we used logistic regression, with human population density and the 12 other factors as predictor variables and *P. ramorum* presence or absence as the response variable.

Results

HUMAN DISPERSAL OF A FOREST PATHOGEN

Our local-scale study indicated that presence of the pathogen in soil on and off hiking trails differed significantly among the three habitat types evaluated ($G^2 = 25.1$, d.f. = 2, $P < 0.0001$; Fig. 3). As expected, *P. ramorum* was equally common in soil on and off trail from infected coast live oak/bay laurel woodlands, where large amounts of pathogen inoculum can be produced. However, for grasslands and white oak forests, that both lack *P. ramorum* hosts, the pathogen was commonly found in soil samples collected from hiking trails, but was virtually absent from soil in adjacent locations off trail.

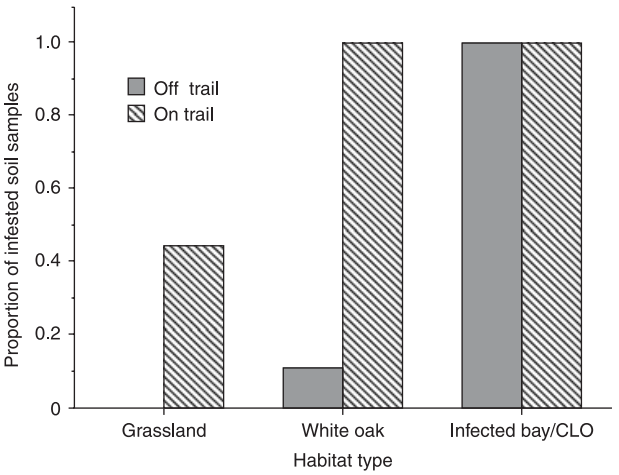

Fig. 3. Proportion of soil samples infested with the pathogen *Phytophthora ramorum* on hiking trails and 1-m off trail in three different habitat types (grassland, white oak forest, and bay laurel/coast live oak forests infected with *P. ramorum*).

LAND ACCESS AND DISEASE SYMPTOMS

Our landscape-level study indicated that the proportion of bay laurel trees exhibiting symptoms of infection by *P. ramorum* was significantly greater in plots on public lands that were open to recreation than in plots on private land that lacked public access (Table 1a; Fig. 4a). We also found that symptoms of infection were positively associated with TMI, maximum daily temperature and amount of forested area surrounding plots (Table 1a). In addition, we detected an underlying spatial pattern of infection across the landscape: the proportion of symptomatic bay laurel trees was greatest in the western and southern parts of our 275 km^2 study region (Table 1a). None of the other environmental variables considered – elevation, minimum daily temperature, relative humidity, precipitation, solar radiation, cumulative DBH of bay laurel and fire history – were significant predictors of symptomatic bay laurel trees and were thus excluded from the model.

We performed a similar landscape-scale analysis for canker hosts (coast live oak, black oak and tanoak) and found that the proportion of symptomatic canker hosts in each plot was positively associated with cumulative DBH of bay laurel and negatively associated with minimum daily temperature (Table 1b). In contrast, public access (Fig. 4b) and the other variables were not significant predictors of symptomatic bay laurel trees and were eliminated from the model.

Table 1. Models resulting from multiple regressions involving the proportion of symptomatic host trees (a, b) and presence of the pathogen *Phytophthora ramorum* (c)

Response variable	Predictor variable	Parameter estimate	F/χ^2	P	Model d.f.	Model R^2 (adjusted)
Landscape level (linear regression)						
(a) Proportion of symptomatic bay hosts per plot	Land status	–0.0540	5.31	0.0224	1, 170	0.23
	TMI	0.0425	11.40	0.0009		
	x Coordinate	–0.000021	11.53	0.0009		
	y Coordinate	–0.000023	18.15	< 0.0001		
	Maximum temperature	–0.0105	2.74	0.0998		
	Amount of forested area	0.000049	2.87	0.0920		
(b) Proportion of symptomatic canker hosts per plot	Cumulative DBH of bay	0.0003	8.90	0.0033	1, 167	0.11
	Minimum temperature	–0.0203	8.11	0.0050		
Regional level (logistic regression)						
(c) Presence of *P. ramorum*	Human population density	–0.0047	7.03	0.0080	1	0.34
	Presence of bay laurel	2.2064	34.14	< 0.0001		
	Precipitation	–0.0421	8.41	0.0037		

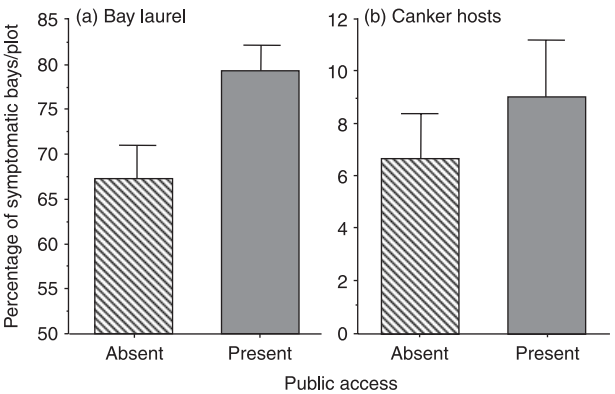

Fig. 4. Mean percentage (+1 SE) of bay laurel hosts (a) and canker hosts (b) exhibiting symptoms of infection by *Phytophthora ramorum* in forests located on public land, open to recreation, vs. private lands lacking public access.

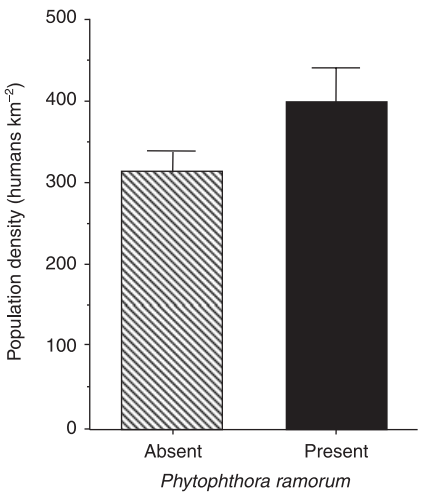

Fig. 5. Mean density (+1 SE) of human populations surrounding forested areas that have host trees infected with *Phytophthora ramorum* and those areas lacking infected host trees.

HUMAN POPULATION DENSITY AND DISEASE PRESENCE

Our regional-scale study indicated that the chances of *P. ramorum* being present at a forested site increased significantly as human population density within a 50 km radius area increased (Table 1c; Fig. 5). We also detected positive associations with precipitation and the presence of bay laurel (Table 1c). None of the other 11 variables – elevation, plot location, sampling year, minimum and maximum temperature, relative humidity, TMI, solar radiation, fire history and tanoak presence – were significant predictors of *P. ramorum* presence and were removed from the model.

Discussion

A growing number of invasive plant pathogens are having substantial effects on a diverse range of host species and ecological systems throughout the world. Although many factors are known to affect the distribution and abundance of these exotic invaders, the extent to which human activities affect their dynamics in nature is poorly understood. Our study has focused on relationships between human activity and the distribution of *P. ramorum* at three spatial scales. At the local scale, we found that there was greater incidence of the pathogen in soil on hiking trails than in adjacent areas off trail. At the landscape scale, our data indicate that forests on public land open to recreation experienced greater disease severity than forests on private land closed to the public. And at the regional scale, our results indicate that the chances of *P. ramorum* occurring at a forested site increased with human population density in the surrounding area. Although experimental approaches in our system were not feasible, our correlative studies tried to identify and control for factors other than human activity that were important in disease spread. In addition, our results at three different spatial scales each independently suggested that humans are one of numerous factors that promote the incidence of this influential forest pathogen.

HUMAN DISPERSAL OF A FOREST PATHOGEN

Our results clearly show that *P. ramorum* more commonly occurred in soil on hiking trails than in soil from adjacent areas off trail, and that the pathogen occurred on trail in locations that lack local sources of inoculum, such as grassland and non-host forest area. We hypothesize that humans drive this pattern by inadvertently tracking *P. ramorum* on the soles of their shoes and dispersing the pathogen along hiking trails into areas that lack host vegetation. Fairfield Osborn Preserve has an active educational program and many people hike its network of trails each week during the time of year when *P. ramorum* is sporulating. Thus, there is ample opportunity for the pathogen to be dispersed by hikers along trails, and possibly contribute to long-distance spread.

An alternative explanation for our hiking trail results is that animals, other than humans, are the primary force dispersing the pathogen. The two most likely non-human dispersers of *P. ramorum* at this site are native black-tailed deer (*Odocoileus hemionus columbianus*) and exotic feral pigs (*Sus scofa domesticus*). Although one of us has studied these species extensively in northern California (e.g. Cushman *et al*. 2004; McNeil & Cushman 2005), we do not feel their activities explain the results shown in Fig. 3. First, feral pigs do not commonly use hiking trails and do not occur at Fairfield Osborn Preserve (J. H. Cushman, personal observation). Thus, they can not be implicated in the dispersal of *P. ramorum* along trails at this location, although may be important off trail dispersers at other sites. Second, black-tailed deer create their own elaborate network of trails throughout Fairfield Osborn Preserve and do not regularly use hiking trails (J. H. Cushman, personal observation).

Further support for our conclusions is provided by the work of Davidson *et al*. (2005), who published the only other study exploring the potential for human transport of *P. ramorum* along hiking trails. In their study, also conducted at Fairfield Osborn Preserve, individuals walked along segments of hiking trail in forests dominated by infected bay laurel hosts and 40% of them had *P. ramorum* in soil on their shoes after traveling 2.4 km. Although these data could not be analysed statistically, Davidson *et al*.'s results clearly show that hikers can pick up *P. ramorum* on the soles of their shoes and lend further support to the hypothesis that humans are dispersal agents of this pathogen.

At least two other *Phytophthora* species have been shown to be dispersed by human activities after their initial introduction. Both Weste & Taylor (1971) and Marks *et al*. (1975) hypothesized that vehicles and logging machinery facilitated the incidence of *P. cinnamomi* in Australian forests because they observed that host trees infected with the pathogen occurred most commonly along the sides of roads and in recently logged areas. In another system, research on the fatal root disease of Port Orford cedar (*Chamaecyparis lawsoniana*) in southern Oregon and northern California showed that *P. lateralis* inoculum was commonly transported in mud and organic matter by vehicles and logging equipment along roads, by hikers and workers on and off trails, and by cattle and other wildlife species (Hansen *et al*. 2000; Jules *et al*. 2002).

Because *P. cinnamoni* and *P. lateralis* are root diseases, it is not surprising that human-dispersed soil infected with these pathogens can lead to disease in host trees. However, *P. ramorum* is an aerial pathogen and not thought to infect host trees via soil-root contact (although see Parke & Lewis 2007). Therefore, the mechanism by which human-dispersed infected soil leads to foliar and canker infection is not as clear. However, Davidson *et al*. (2005) showed that *P. ramorum* can be spread from infected soil to green leaf litter of bay laurel, which can then infect bay laurel seedlings via rain splash, thus suggesting that infected soil has the potential to be an important driver of *P. ramorum* transmission between host plants. Splash dispersal has also been shown to be an important mechanism of spread for other species in the genus *Phytophthora* (Madden *et al*. 1992, Ristaino & Gumpertz 2000). An additional soil-to-plant transmission pathway in our system may involve birds and small mammals. Western scrub-jays (*Aphelocoma californica*), wild turkeys (*Meleagris gallopavo*) and western grey squirrels (*Sciurus griseus*) all spend substantial time moving back and forth between the soil surface and bay laurel canopies (J. H. Cushman, personal observations). Such frequent movements between infected soil and foliar host vegetation suggest that there is abundant opportunity for these animals to also serve as key dispersal agents of *P. ramorum*.

LAND ACCESS AND DISEASE SYMPTOMS

Landscape-level data from our 202 study plots in eastern Sonoma County indicated that six variables were significant predictors of disease symptoms caused by *P. ramorum*. First, we found that forests on public land open to recreation were associated with increased symptoms of infection in the most dominant foliar host, bay laurel, even after taking into account the potentially confounding effects of 12 environmental factors. We hypothesize that the increased symptoms of disease in forests on public land occurred because these areas experience greater amounts of recreational activity (i.e. hiking, mountain biking, horseback riding), which in turn results in a greater influx of *P. ramorum* infested soil to these areas. Increased infection of bay laurel is important because it is one of the most heavily infected carriers of the disease in California forests (Davidson *et al*. 2005) and its presence is correlated with oak and tanoak mortality (Kelly & Meentemeyer 2002; Maloney *et al*. 2005; Swiecki & Bernhardt 2006).

An alternative explanation for our landscape-level results is that forests on public land differ fundamentally in some way from those on private land – other than the degree of public access. For example, the dominance of bay, amount of surrounding forest, geographical location and/or microclimate of plots on public and private land could all differ substantially. If such variation existed, and was not corrected for, one might falsely conclude that public access was a key factor rather than these other variables. We were cognizant of this possibility at the outset of our study and purposely accounted for these and other variables in our multivariate statistical analysis.

Thus, we feel justified concluding that our findings for public access are real and not an artefact.

Public access was not the only significant predictor of disease symptoms on bay laurel. First, we found that greater values of the TMI in plots were associated strongly with increased levels of disease symptoms. TMI estimates the ratio of upslope drainage area to slope for a site and greater values indicate higher potential soil wetness. Greater soil wetness may (i) allow the pathogen to persist longer in the soil, (ii) facilitate the ability of humans to track infested soil on the soles of their shoes, and/or (iii) increase evapotranspiration cooling, creating more suitable understorey microclimate conditions for transmission and survival of *P. ramorum*. In addition, we found that disease symptoms were most severe in southern and western portions of our 275 km^2 study area. These results probably reflect the historical pattern of disease spread across the northern San Francisco bay area. The first observed infections caused by *P. ramorum* occurred in the Mill Valley area of Marin County in the mid-1990s (Rizzo & Garbelotto 2003). Because of this, we hypothesize that the south-western portion of our study area was infected first, providing a greater amount of time for inoculum to accumulate in these forests relative to those to the north and east.

We detected two other strong trends that also warrant discussion. First, our data revealed a positive association between the intensity of disease symptoms in bay laurel and maximum daily temperature from December to May (the time of year when *P. ramorum* is most active and abundant). As long as there is ample rainfall and soil wetness, warm climate conditions at this time should facilitate growth of *P. ramorum* and promote host infection (Davidson *et al*. 2005). Second, we also detected a positive association between the intensity of disease symptoms in bay laurel and the amount of forested area surrounding plots. This result suggests that the ability of *P. ramorum* to infect bay laurel may be enhanced by large amounts of fairly contiguous host vegetation. This pattern is consistent with results from two other landscape-scale studies of *P. ramorum* (Condeso & Meentemeyer 2007; Meentemeyer *et al*. 2008a) as well as other pathogen species (Burdon *et al*. 1995, Thrall *et al*. 2003). Greater amounts of forest area may promote the success of *P. ramorum* by increasing landscape connectivity of host vegetation (Burdon 1987; Condeso & Meentemeyer 2007). Larger forested areas may also better sustain larger populations of *P. ramorum* during periods of unproductive climatic conditions (Burdon 1987).

Public access and 10 other environmental variables were not significant predictors of disease symptoms in canker hosts. The insignificance of public access and other variables is not surprising given that the pathogen spreads among bay laurel trees far in advance of oak infection (Davidson *et al*. 2005) and our study area is in an earlier stage of invasion by *P. ramorum* than other more publicized regions (e.g. Marin County and Big Sur). As a result, a large fraction of the bay laurel trees in our plots exhibited symptoms of infection by *P. ramorum* (Fig. 4a), whereas symptomatic canker hosts were substantially less common (Fig. 4b). As *P. ramorum* infection intensifies in bay laurel over time, we expect that more canker hosts will become infected and that patterns similar to those found for bay laurel will emerge.

Although public access was not yet an important predictor of disease for canker hosts, we found that both cumulative DBH of bay laurel and minimum daily temperature were significant. As hypothesized, symptoms of disease in canker hosts were more likely to occur as bay laurel became more dominant in plots (as measured by cumulative DBH). This result is consistent with the findings of other studies and further corroborates the importance of bay laurel in promoting disease in oaks and tanoaks (Kelly & Meentemeyer 2002; Davidson *et al*. 2005; Maloney *et al*. 2005). Higher minimum daily temperatures in December through May were also associated with greater disease symptoms in canker hosts. This seems reasonable given that locations with higher minimum temperatures should provide *P. ramorum* with more suitable growing conditions and more frequent canker host infections.

HUMAN POPULATION DENSITY AND DISEASE PRESENCE

Our regional-scale analysis of 165 sites in 14 counties of northern and central California indicated that human population density and two other predictor variables were significantly associated with disease presence. Two mechanisms are likely to explain the positive association between *P. ramorum* occurrence and human population density. First, because forests surrounded by high-density populations typically experience greater amounts of recreational activity, greater amounts of pathogen inoculum may be introduced, thereby increasing the likelihood of infections. Second, infected ornamental plants may be more frequently planted in residential areas adjacent to forests surrounded by high-density populations (Rizzo *et al*. 2005). A growing number of ornamental species (including azalea, camellia, grand fir and rhododendron) have been found to serve as foliar hosts for *P. ramorum* and infected plants are regularly found in nurseries (Stokstad 2004). Over numerous years, introduction of infected ornamental plants at urban–wildland interfaces may have served as primary infections, which subsequently led to greater amounts of *P. ramorum* inoculum and infection of host plants in these more populated regions. In summary, regardless of which mechanisms were operating, our data suggest that increased human density can inadvertently lead to increased *P. ramorum* occurrence and the disease it causes in adjacent forests.

In addition to the associations involving human population density, our analysis revealed that the chances of *P. ramorum* occurrence increased significantly with both the presence of bay laurel and precipitation. The bay laurel result is consistent with our previous analysis and the findings of other research, further implicating the pivotal role of this host in disease establishment. Higher precipitation may increase chances of *P. ramorum* occurrence in two ways: (i) by providing sufficient moisture for growth and transmission and (ii) by creating wetter soils that facilitate the ability of humans to track infested soil.

As with our landscape-level study discussed previously, there may be alternative explanations for our regional-scale results that warrant discussion. Specifically, if areas of high human density tend to occur in locations with specific characteristics – such as similar ranges in temperature or precipitation, for example – then it may be possible to detect an association between disease and density that is actually due to one or more of these characteristics, and not human density. If such situations exist and are not accounted for, one could again falsely conclude that human density was important, when in fact some other factor or combination of factors were driving the relationship. However, through our multivariate statistical analysis, we carefully examined 13 other variables that we suspected might also predict disease occurrence. Having taken this approach, we conclude that the significance of human density is real and not an artefact of correlations with other linked variables.

Fire is well-known to have played an important role in structuring forests of California (Sugihara *et al*. 2006) and there has been considerable interest in the degree to which it might affect the success of *P. ramorum* and Sudden Oak Death. For example, Moritz & Odion (2005) also examined large-scale patterns of *P. ramorum* occurrence in relation to mapped fire history since 1950. Using the same fire-history records that we used in our analyses, they found that infected locations were significantly less common in burned areas relative to unburned ones. In light of these results, we were surprised that fire history was an insignificant predictor of disease occurrence in our study. We suspect that the differing results stem from how the two studies delineated their focal study areas. Moritz and Odion defined a study area that ranged from the northern-most latitude in California where *P. ramorum* is known to infect host trees at an isolated outbreak in Humboldt County to the southern-most latitude in Monterey County, and then 61 km inland throughout this area, which corresponds to the farthest distance from the coast that infection is known to occur. In contrast, as described previously, our study area consisted of a 20 km region surrounding each of the 275 sites in California known to contain infected host plants. As a result, Moritz and Odion's study area was much larger than ours and may have included many more inland forested regions that burn regularly but have less favourable climatic conditions and less foliar host vegetation for use by *P. ramorum* (see Meentemeyer *et al*. 2004). Thus, we hypothesize that the consequence of defining a larger study area that contained many low-risk but fire-prone forests was to detect an association between fire and an absence of disease.

MANAGEMENT IMPLICATIONS

Coastal forests in California and Oregon are experiencing high levels of tree disease and mortality as a result of infection by *P. ramorum*. Although this pathogen has not reached epidemic levels in other regions of the world, it occurs in many areas of western Europe and there is considerable international concern that the disease will spread rapidly and cause severe economic and ecological damage (Rizzo & Garbelotto 2003; Appiah *et al*. 2004). Clearly, humans can unintentionally transport the pathogen long distances – among counties, states and countries – via the nursery trade and there is great apprehension about this possibility, especially since such activity has been implicated in the initial introduction of *P. ramorum* into North America (Rizzo *et al*. 2005, 2005). Our research offers multiple lines of evidence which each indicate that human activity in the form of recreation leads to increased incidence of disease, after the initial introduction. These results suggest that there may be conflicts between recreation and disease, and that efforts to control the disease may require aggressive management. One strategy for minimizing dispersal of infested soil would be to establish a collection of stations for cleaning hiking shoes and mountain bike tires in high-risk areas during wet, mild times of the year (March–May would probably be most critical). Shoe and tire cleaning procedures are known to be effective (Davidson *et al*. 2005; J. M. Davidson, pers. comm.) and several programs have been implemented on a small scale by state and federal agencies. However, these programs may need to be implemented on a much larger scale to be effective, which would require considerable commitment from agency officials as well as extensive community outreach and education. An alternative approach would be to restrict access to high-risk areas during periods of peak inoculum production. However, we suspect this approach be unpopular with the public and would also require considerable vigilance and resources from agencies to implement and enforce. The logistical and political challenges posed by these management strategies highlight the increasingly dire situation that public lands confront. With the increasing human use and decreasing funding that most protected natural areas face (Cole & Landres 1996), mitigating the impacts of recreation and the success of *P. ramorum* will continue to create enormously complex problems.

Conclusions

Identifying the suite of factors that mediate the distribution and abundance of plant pathogens in nature is critical for understanding the dynamics of these influential players, and ultimately the effects they have on communities and ecosystems. Our research has suggested that human activity, along with other key variables, is an important factor affecting the prevalence of an extremely influential exotic pathogen. We suspect that our results for humans and *P. ramorum* are not unique and believe that considerable insight will be gained by addressing the potential roles of human activities in the transmission of disease in other pathogen systems.

Acknowledgements

We thank Josh Amaris, Shelly Benson, Cheyenne Boylen, Kelly Buja, Emiko Condeso, Michelle Cooper, Greg Garner, Andre Gauthier, Eric Gordon, Rich Hunter, Amy Jirka, Sean King, Melina Kozanitas, Eric McCowan, Walter Mark, Leonard Miller, Steve Moyle, Gary Neargarner, Deanne Pietro, Heather Torpin, Kirsten Ward and James Zanzot for assistance with field work. We are grateful to Ian Smith for providing references on *P. cinnamomi* and to Brian

Anaker, Rich Hunter and Elizabeth Lotz for logistical support and assistance with GIS mapping. Comments from Jeremy Burdon, Caroline Christian, Barney Davies, David Gibson, Everett Hansen, Max Moritz, Karina Nielsen, Nathan Rank, David Rizzo, Tom Whitham, Richard Whitkus and two anonymous referees greatly improved the manuscript. Special thanks go to the landowners and public agencies that granted us access to their properties. This research was generously supported by grants from the National Science Foundation, USDA Forest Service (Pacific South-west Research Station), CSU Agricultural Research Initiative and the California Department of Forestry and Fire Protection.

References

Anagnostakis, S. (1987) Chestnut blight: the classical problem of an introduced pathogen. *Mycologia*, **79**, 23–37.

Anderson, P.K., Cunningham, A.A., Patel, N.G., Morales, F.J., Epstein, P.R. & Daszak, P. (2004) Emerging infectious diseases in plants: pathogen pollution, climate change and agrotechnology drivers. *Trends in Ecology and Evolution*, **19**, 535–544.

Appiah, A.A., Jennings, P. & Turner, J.A. (2004) *Phytophthora ramorum*: one pathogen and many diseases, an emerging threat to forest ecosystems and ornamental plant life. *Mycologia*, **18**, 145–150.

Beven, K.J. & Kirkby, M.J. (1979) A physically based, variable contributing area model of basin hydrology. *Hydrological Sciences Bulletin*, **24**, 43–69.

Brasier, C.M. (2003) Sudden Oak Death; *Phytophthora ramorum* exhibits transatlantic differences. *Mycological Research*, **107**, 257–259.

Burdon, J.J. (1987) *Diseases and Plant Population Biology*. Cambridge University Press, New York.

Burdon, J.J., Ericson, L. & Müller, W.J. (1995) Temporal and spatial changes in a metapopulation of the rust pathogen *Triphragmium ulmariae* and its host, *Filipendula ulmaria*. *Journal of Ecology*, **83**, 979–989.

Burdon, J.J., Thrall, P.H. & Ericson, L. (2006) The current and future dynamics of disease in plant communities. *Annual Review of Phytopathology*, **44**, 19–39.

Cole, D.N. & Landres, P.B. (1996) Threats to wilderness ecosystems: impacts and research needs. *Ecological Applications*, **6**, 168–184.

Condeso, T.E. & Meentemeyer, R.K. (2007) Effects of landscape heterogeneity on the emerging disease Sudden Oak Death. *Journal of Ecology*, **95**, 364–375.

Cushman, J.H., Tierney, T.A. & Hinds, J.M. (2004) Variable effects of feral pig disturbances on native and exotic plants in a California grassland. *Ecological Applications*, **14**, 1746–1756.

Daly, C., Neilson, P.P. & Phillips, D.L. (1994) A statistical-topographic model for mapping climatological precipitation over mountainous terrain. *Journal of Applied Meteorology*, **33**, 140–158.

Daszak, P., Cunningham, A.A. & Hyatt, A.D. (2000) Emerging infectious diseases of wildlife – threats to biodiversity and human health. *Science*, **287**, 443–449.

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

Davidson, J.M., Wickland, A.C., Patterson, H.A., Falk, K.R. & Rizzo, D.M. (2005) Transmission of *Phytophthora ramorum* in mixed-evergreen forest in California. *Phytopathology*, **95**, 587–596.

Dobson, A., Crawley, M. (1994) Pathogens and the structure of plant communities. *Trends in Ecology and Evolution*. **9**, 393–398.

Dubayah, R. (1994) A solar radiation topoclimatology for the Rio Grande river basin. *Journal of Vegetation Science*, **5**, 627–640.

Fichtner, E.J., Lynch, S.C. & Rizzo, D.M. (2007) Detection, distribution, sporulation and survival of *Phytophthora ramorum* in California redwood-tanoak forest soil. *Phytopathology* **97**, 1366–1375.

Foley, J.A., DeFries, R., Asner, G.P., Barford, C., Bonan, G., Carpenter, S.R., *et al*. (2005) Global consequences of land use. *Science*, **309**, 570–574.

Garbelotto, M., Davidsin, J.M., Ivors, K., Maloney, P.E., Huberli, D., Koike, S.T. & Rizzo, D.M. (2003) Non-oak native plants are the main hosts for the Sudden Oak Death pathogen in California. *California Agriculture*, **57**, 18–23.

Gilbert, G.S. (2002) Evolutionary ecology of plant diseases in natural ecosystems. *Annual Review of Phytopathology*, **40**, 13–43.

Hansen, E.M., Goheen, D.J., Jules, E.S. & Ullian, B. (2000) Managing Port-Orford-cedar and the introduced pathogen *Phytophthora lateralis*. *Plant Disease*, **84**, 4–14.

Harvell, C.D., Mitchell, C.E., Ward, J.R., Altizer, S., Dobson, A.P., Ostfeld, R.S. & Samuel, M.D. (2002) Climate warming and disease risks in terrestrial and marine biota. *Science*, **296**, 2158–2162.

Hayden, K.J., Rizzo, D.M., Tse, J. & Garbelotto, M. (2004) Detection and quantification of *Phytophthora ramorum* from California forests using a real-time polymerase chain reaction assay. *Phytopathology*, **94**, 1075–1083.

Henricot, B. & Prior, C. (2004) *Phytophthora ramorum*, the cause of sudden oak death or *ramorum* leaf blight and dieback. *Mycologist*, **18**, 151–156.

Holdenrieder, O., Pautasso, M., Weisberg, P.J. & Lonsdale, D. (2004) Tree diseases and landscape processes: the challenge of landscape pathology. *Trends in Ecology and Evolution*, 19, 446–452.

Ivors, K., Hayden, K.J., Bonants, P.J.M., Rizzo, D.M. & Garbelotto, M. (2004) AFLP and phylogenetic analyses of North American and European populations of *Phytophthora ramorum*. *Mycological Research*, **108**, 378–392.

Jules, E.S., Kauffman, M.J., Ritts, W.D. & Carroll, A.L. (2002) Spread of an invasive pathogen over a variable landscape: a nonnative root rot on the Port Orford cedar. *Ecology*, **83**, 3167–3181.

Kelly, N.M. & Meentemeyer, R.K. (2002) Landscape dynamics of the spread of Sudden Oak Death. *Photogrammetric Engineering & Remote Sensing*, **68**, 1001–1009.

Madden, L.V., Wilson, L.L., Yang, X. & Ellis, M.A. (1992) Splash dispersal of *Colletotrichum acutatum* and *Phytophthora cactorum* by short-duration simulated rains. *Plant Pathology*, **41**, 427–436.

Maloney, P.E., Lynch, S.C., Kane, S.F., Jensen, C.E. & Rizzo, D.M. (2005) Establishment of an emerging generalist pathogen in redwood forest communities. *Journal of Ecology*, **93**, 899–905.

Marks, G.C., Kassaby, F.Y. & Fagg, F.Y. (1975) Variation in population levels of *Phytophthora cinnamomi* in *Eucalyptus* forest soils in eastern Victoria. *Australian Journal of Botany*, **23**, 435–439.

McNeil, S.G. & Cushman, J.H. (2005) Indirect effects of deer herbivory on local nitrogen availability in a coastal dune ecosystem. *Oikos*, **110**, 124–132.

Meentemeyer, R.K., Anaker, B.J., Marks, W. & Rizzo, D.M. (2008b) Early detection of emerging forest disease using dispersal estimation and ecological niche modeling. *Ecological Applications*, in press.

Meentemeyer, R.K., Rank, N.E, Anaker, B.J., Rizzo, D.M. & Cushman, J.H. (2008a) Influence of land-cover change on the spread of an invasive forest pathogen. *Ecological Applications*, **18**, 159–171.

Meentemeyer, R.K., Rizzo, D.M., Mark, W. & Lotz, E. (2004) Mapping the risk of establishment and spread of Sudden Oak Death in California. *Forest Ecology and Management*, **200**, 195–214.

Moore, I.D., Grayson, R.B. & Ladson, A.R. (1991) Digital terrain modeling–a review of hydrological, geomorphological, and biological applications. *Hydrological Processes*, **5**, 3–30.

Moritz, M.A. & Odion, D.C. (2005) Examining the strength and possible causes of the relationship between fire and Sudden Oak Death. *Oecologia*, **144**, 106–114.

Newhook, F.J. & Podger, F.D. (1972) The role of *Phytophthora cinnamomi* in Australian and New Zealand forests. *Annual Review of Phytopathology*, **10**, 299–326.

Parke, J.L. & Lewis, C. (2007) Root and stem infection of rhododendron from potting medium infested with *Phytophthora ramorum*. *Plant Disease*, **91**, 1265–1270.

Ristaino, J.B. & Gumpertz, M.L. (2000) New frontiers in the study of dispersal and spatial analysis of epidemics caused by species in the genus *Phytophthora*. *Annual Review of Phytopathology*, **38**, 541–576.

Rizzo, D.M. (2005) Exotic species and fungi: interactions with fungal, plant and animal communities. *The Fungal Community*, 3rd edn (eds J. Dighton, P. Oudemans & J. White), pp. 857–877. CRC Press, Boca Raton.

Rizzo, D.M. & Garbelotto, M. (2003) Sudden oak death: endangering California and Oregon forest ecosystems. *Frontiers in Ecology & the Environment*, **1**, 197–204.

Rizzo, D.M., Garbelotto, M. & Hansen, E.M. (2005) *Phytophthora ramorum*: Integrative research and management of an emerging pathogen in California and Oregon forests. *Annual Review of Phytopathology*, **43**, 309–335.

Rizzo, D.M., Garbelotto, M., Davidson, J.M. & Slaughter, G.W. (2002) *Phytophthora ramorum* as the cause of extensive mortality of *Quercus* sp. and *Lithocarpus densiflora* in California. *Plant Disease*, **86**, 205–214.

Smith, K.F., Sax, D.F. & Lafferty, K.D. (2005) Evidence for the role of disease in species extinction and endangerment. *Conservation Biology*, **20**, 1349–1357.

Stokstad, E. (2004) Nurseries may have shipped Sudden Oak Death pathogen nationwide. *Science*, **26**, 1959.

Sugihara, N.G., Van Wagtendonk, J.W., Shaffer, K.E., Fites-Kaufman, J. & Thode, A.E., eds (2006) *Fire in California's Ecosystems*. University of California Press, Berkeley.

Swiecki, T.J. & Bernhardt, E. (2006) Disease risk factors and disease progression in coast live oak and tanoak affected by *Phytophthora ramorum* canker (Sudden Oak Death). *Proceedings of the 2nd Sudden Oak Death Science Symposium*. 2005 January 18–21; Monterey, CA. Gen. Tech. Rep. PSW-

GTR-196. Pacific Southwest Research Station, Forest Service, U.S. Department of Agriculture, Albany, California.

Thrall, P., Godfree, R. & Burdon, J.J. (2003) Influence of spatial structure on pathogen colonization and extinction: a test using an experimental metapopulation. *Plant Pathology*, **52**, 350–361.

Werres, S., Marwitz, R., Man in 't Veld, W.A., De Cock, A.W., Bonants, P.J.M., De Weerdt, M., Themann, K., Ilieva, E. & Baayen, R.P. (2001) *Phytophthora ramorum sp. nov*: a new pathogen on Rhododendron and viburnum. *Mycological Research*, **105**, 1155–1165.

Weste, G. & Marks, G.C. (1987) The biology of *Phytophthora cinnamomi* in Australasian forests. *Annual Review of Phytopathology*, **25**, 207–230.

Weste, G. & Taylor, P. (1971) The invasion of native forest by *Phytophthora cinnamomi*. I. Brisbane Ranges, Victoria. *Australian Journal of Botany*, **19**, 281–294.

Woods, A., Coates, K.D. & Hamann, A. (2005) Is an unprecedented Dothistroma needle blight epidemic related to climate change? *Bioscience*, **55**, 761–769.

Received 24 August 2007; accepted 10 March 2008
Handling Editor: Jeremy Burdon