

Effects of individual, community, and landscape drivers on the dynamics of a wildland forest epidemic

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Abstract. The challenges posed by observing host–pathogen–environment interactions across large geographic extents and over meaningful time scales limit our ability to understand and manage wildland epidemics. We conducted a landscape-scale, longitudinal study designed to analyze the dynamics of sudden oak death (an emerging forest disease caused by *Phytophthora ramorum*) across hierarchical levels of ecological interactions, from individual hosts up to the community and across the broader landscape. From 2004 to 2011, we annually assessed disease status of 732 coast live oak, 271 black oak, and 122 canyon live oak trees in 202 plots across a 275-km² landscape in central California. The number of infected oak stems steadily increased during the eight-year study period. A survival analysis modeling framework was used to examine which level of ecological heterogeneity best predicted infection risk of susceptible oak species, considering variability at the level of individuals (species identity, stem size), the community (host density, inoculum load, and species richness), and the landscape (seasonal climate variability, habitat connectivity, and topographic gradients). After accounting for unobserved risk shared among oaks in the same plot, survival models incorporating heterogeneity across all three levels better predicted oak infection than did models focusing on only one level. We show that larger oak trees (especially coast live oak) were more susceptible, and that interannual variability in inoculum production by the highly infectious reservoir host, California bay laurel, more strongly influenced disease risk than simply the density of this important host. Concurrently, warmer and wetter rainy-season conditions in consecutive years intensified infection risk, presumably by creating a longer period of inoculum build-up and increased probability of pathogen spillover from bay laurel to oaks. Despite the presence of many alternate host species, we found evidence of pathogen dilution, where less competent hosts in species-rich communities reduce pathogen transmission and overall risk of oak infection. These results identify key parameters driving the dynamics of emerging infectious disease in California woodlands, while demonstrating how multiple levels of ecological heterogeneity jointly determine epidemic trajectories in wildland settings.

Key words: diversity-disease risk; emerging infectious disease; landscape epidemiology; pathogen spillover; *Phytophthora ramorum*; sudden oak death; seasonality; survival analysis; time-varying covariate.

INTRODUCTION

Risk of emerging infectious diseases in forest ecosystems continues to rise as worldwide changes in human mobility and land-use patterns promote the establishment and spread of nonnative plant pathogens (Lovett et al. 2006, Aukema et al. 2010). Once established, emerging diseases can significantly alter the structure and function of forest ecosystems when their

impacts on growth, reproduction, or survival reduce the abundance of ecologically important host species in the community (Paillet 2002, Ellison et al. 2005, Mordecai 2011). Given that novel plant diseases are predicted to increase worldwide (Jones 2009, Fisher et al. 2012), coupled with their potentially large economic and ecological impacts (Pimentel et al. 2005, Kovacs et al. 2011), it is crucial to identify the environmental factors driving epidemic outcomes in forest diseases. This is an especially challenging task in wildland ecosystems (i.e., those not planted, farmed, or intensively managed) as they are characterized by complex spatial and temporal heterogeneity of hosts and environmental conditions across broad geographical extents (Alexander 2010, Hatala et al. 2011,

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Meentemeyer et al. 2012). To date, we have little empirical understanding of how variability across multiple levels of individual hosts, their communities, and broader landscape contexts jointly influence disease dynamics in wildland settings.

At the level of individual hosts, intraspecific variation in disease susceptibility is a principal source of heterogeneity controlling epidemic trajectories (Cronin et al. 2010, Haavik and Stephen 2010, Jules et al. 2014). Such heterogeneity may stem from numerous sources including host genetics, morphology, size, or age (Garrett et al. 2009, McPherson et al. 2010, Bell et al. 2015). For example, emerging forest pathogens have often been shown to have greater impacts on large trees than small ones (Kauffman and Jules 2006, Cobb et al. 2012, Jules et al. 2014). Larger trees may be more susceptible to infection due to higher pathogen contact rates (e.g., larger surface area), higher likelihood of attack by pathogen vectors (e.g., more suitable bark texture or chemistry) or other life-history traits correlated with size and age (Smith and Hoffman 2001, Paillet 2002). In more complex multihost systems, interspecific variability in susceptibility must also be accounted for, as generalist pathogens can exhibit differential impacts on species that vary in their competence (the ability to maintain and transmit infections). In such situations, long-term monitoring of individuals from across the entire host species pool is needed to capture and understand interspecific differences in disease impacts, including differential rates and patterns of infection and disease-induced mortality (Cobb et al. 2010, Kueh et al. 2012).

Studies of disease ecology at the community level more broadly consider how variation in species composition, abundance and diversity alter epidemic trajectories (Mitchell et al. 2002, Keesing et al. 2006, Haavik and Stephen 2010). Host density is often regarded as a major factor driving disease epidemics, either through direct mechanisms (e.g., reduced inter-host distances) or indirect effects such as parasite-mediated competition (Burdon and Chilvers 1982). Generalist pathogens, however, may be decoupled from the density of any single host and instead respond to the joint population densities of several species (Freckleton and Lewis 2006, Beckstead et al. 2010), in which disease dynamics are regulated by asymmetries in the competency of each host species to maintain and transmit infection (Cobb et al. 2010; Mordecai 2011). Highly competent hosts that are abundant and widespread can function as pathogen reservoirs, amplifying inoculum that then spills over to less competent hosts (Power and Mitchell 2004). Frequent spillover can have dramatic and long-lasting consequences for community structure and function through the selective removal of hosts that are more sensitive to disease-induced mortality (Metz et al. 2012). In contrast, species-rich communities characterized by a higher relative abundance of less-competent hosts can exhibit reduced disease

risk due to diluted pathogen transmission events (Keesing et al. 2010, Ostfeld and Keesing 2012).

Studies of landscape-level disease dynamics often consider habitat and climatic heterogeneity across broader spatial and temporal scales (Holdenrieder et al. 2004, Meentemeyer et al. 2012). For many plant pathogens, particularly those exhibiting passive dispersal via wind and water flow, connectivity of host vegetation across the landscape facilitates long-distance dispersal of infectious propagules and hence contributes to the overall inoculum pressure experienced at a site (Perkins and Matlack 2002, Ellis et al. 2010). Simultaneously, landscape-level gradients in abiotic conditions, including weather and topographic variability, can greatly influence pathogen population dynamics and dispersal (Kauffman and Jules 2006, Condeso and Meentemeyer 2007, Moore and Borer 2012). For example, seasonal variation in temperature and rainfall has been shown to exert strong influences on infection patterns at both local and landscape scales due to changes in host phenology and abundance of pathogen propagules in the environment (Altizer et al. 2006). Nevertheless, conducting landscape-level disease studies across spatially heterogeneous wildland ecosystems often comes at the cost of ignoring individual- and community-level details of the disease system (Meentemeyer et al. 2012, Dillon et al. 2014).

In this paper, we describe results from a landscape-scale, longitudinal study designed to evaluate disease dynamics of sudden oak death (SOD, caused by *Phytophthora ramorum*) across multiple levels of host-pathogen-environment interactions, from individual hosts up to the community and across the broader landscape. First observed in North America during the mid 1990s (Rizzo et al. 2002), SOD has killed millions of oak (*Quercus* spp.) and tanoak (*Notholithocarpus densiflorus*) trees in coastal forests of California and Oregon (Meentemeyer et al. 2008b, Brown and Allen-Diaz 2009, McPherson et al. 2010), and is also affecting managed landscapes in Europe (Brasier and Webber 2010). Disease forecasts estimate a 10-fold increase in the spread of SOD in California by 2030 (Meentemeyer et al. 2011), potentially putting billions of oak and tanoak trees at risk of infection (Lamsal et al. 2011).

Several factors make SOD a valuable system for understanding how ecological heterogeneity at multiple levels influences wildland epidemics. First, *P. ramorum* is a generalist pathogen that infects dozens of native plant species in forests, each with different infection susceptibilities and competency for disease transmission (Rizzo et al. 2005). Broadly speaking, two types of symptoms occur across the pathogen's wide range of North American hosts: dead-end canker infections on the main stem of oaks and tanoaks that may be lethal, and nonlethal foliar infections on a long list of woody and herbaceous hosts that produce variable amounts of inoculum. These foliar infections can amplify the

pathogen, driving transmission between hosts and pathogen spillover to oaks and tanoak. For example, the widespread reservoir host, California bay laurel (*Umbellularia californica*), produces the most inoculum in coastal forests of central California (Davidson et al. 2005, 2008) and has been strongly correlated with disease risk in several landscape epidemiological studies (Condeso and Meentemeyer 2007, Haas et al. 2011, Dillon et al. 2014). Tanoak is a unique host that contracts both lethal stem and twig infections and may produce significant inoculum on its leaves (Rizzo et al. 2005). The extent to which other hosts play a role in pathogen transmission is less understood, although their presence has been shown to “dilute” disease risk in biologically diverse communities (Haas et al. 2011).

A second factor that makes *P. ramorum* valuable for studying disease dynamics in wildlands is that pathogen transmission occurs through several dispersal mechanisms that contribute to both short- and long-range spread, and which are highly sensitive to fluctuating weather conditions (Davidson et al. 2005, 2008, DiLeo et al. 2014). Pathogen sporulation in California is most prolific in late-winter and spring months (November–May), corresponding to the rainy season of the region’s Mediterranean type climate, when prolonged leaf wetness during warm rains can result in very high levels of inoculum production (Rizzo et al. 2005). During wet conditions, most pathogen dispersal occurs locally (<250 m) via inoculum spillover from infected leaves of foliar hosts to nearby oaks through splashed and windblown rain (Davidson et al. 2005). At larger geographic extents, *P. ramorum* may be transported long distance through streams (Hohl et al. 2013), outplanting of infected ornamental plants along wildland-urban interfaces (Rizzo et al. 2005), and via infested soil carried by wildlife, vehicles, and hikers (Cushman and Meentemeyer 2008).

Here we ask two interrelated questions using a survival analysis modeling framework that considers spatial and temporal heterogeneity across multiple scales of ecological organization: (1) which level (or combination) of heterogeneity—individual, community or landscape—best predicts risk of infection in susceptible oaks, and (2) does seasonal climate variability influence progression of oak disease severity, and if so, which temporal extents best explain infection patterns? In answering these questions, we aim to identify key parameters driving the spread of SOD in California, while shedding light on the role that multilevel environmental heterogeneity plays in wildland forest epidemics.

METHODS

Disease monitoring

To fit our multilevel survival model, we collected field data annually from 2004 to 2011 at 202 randomly

distributed, geospatially referenced plots (15 × 15 m) across a 275-km² region in eastern Sonoma County, California, USA (Appendix S1: Fig. S1). *P. ramorum* impacts were first observed in the region in 2000, and the pathogen has since become widely distributed across the landscape (Anacker et al. 2007, Cushman and Meentemeyer 2008, Meentemeyer et al. 2008a, Ellis et al. 2010). Across the study area, elevation ranged from 55 to 800 m, and plant communities were composed of distinct patches of mixed evergreen forest dominated by bay laurel and oak species, interspersed with annual grassland and chaparral. Upon plot establishment in 2004, we conducted detailed vegetation surveys to catalog the species identity of all woody plants in the understory and overstory of each plot. Tanoak occurrence was low across the study area and was observed in only nine plots.

During plot establishment, we tagged tree stems ≥2 cm diameter at breast height (DBH; 1.4 m height) for annual remeasurement of the reservoir host, bay laurel; the three dead-end hosts, including coast live oak (*Quercus agrifolia*), black oak (*Q. kelloggii*), and canyon live oak (*Q. chrysolepis*); and tanoak. Stems were defined as either the main trunk or as individual branches separating from the main trunk below breast height. Only oaks rooted in plots were surveyed, whereas bay laurel and tanoak were included as long as a portion of their canopy intersected the plot. Each year during peak symptom expression (March–May), tagged stems were assessed for health status (alive/dead) and the presence of *P. ramorum* symptoms. Infection on oaks was based on the presence of “bleeding” canker lesions (i.e., dark red exudates and discoloration of bark surface) on the main stem that are characteristic of *P. ramorum*. To relocate cankers across years, we recorded canker dimensions and location for each stem. A stem’s transition from uninfected to infected required that one or more cankers be found for at least two (not necessarily consecutive) years. Oak stems that died following infection were assumed to have died from SOD. Stem mortality (defined as death of all aboveground tissue) was based on examination of cambial damage to the basal tree trunk, drying and abscission of foliage, and presence of opportunistic organisms including ambrosia beetles (*Monarthrum scutellare* and *M. dentiger*), bark beetles (*Pseudopityophthorus pubipennis*), and a sapwood rotting fungus (*Annulohyphoxylon thouarsianum*) (McPherson et al. 2005).

Our assessment of foliar infection focused on the reservoir host bay laurel, the primary producer of inoculum in this landscape (Davidson et al. 2005, 2008, Anacker et al. 2007, Condeso and Meentemeyer 2007). Bay laurel is the most abundant species across the study system (present in 180 of 202 plots; 89%). Given that most bay laurel exhibit some level of *P. ramorum* infection in infested regions (Anacker et al. 2007; Ellis et al. 2010), assessing an individual as either infected

or uninfected was deemed too coarse to examine inter-plot variation in disease severity. Instead, we quantified disease severity in each plot by counting the number of symptomatic leaves on each bay laurel stem ≥ 2 cm DBH for 60 s each year (Anacker et al. 2007, Meentemeyer et al. 2008a). Infected leaves on bay laurel primarily occur in the lower, visible canopy where *P. ramorum* performs best in mild, wet, and shaded environments (with minimal UV exposure) (Davidson et al. 2005, DiLeo et al. 2014). Prior to sampling, a pilot study demonstrated no significant differences in the number of symptomatic leaves counted by different observers (Condeso and Meentemeyer 2007). The presence of *P. ramorum* in each plot was verified via diagnostic laboratory protocols using *Phytophthora*-selective media (Davidson et al. 2005).

Other *Phytophthora* species (*P. nemorosa*, *P. pseudosyringae*) are found in coastal California forests and can cause symptoms in oak and bay laurel that appear similar to those caused by *P. ramorum* (Wickland et al. 2008). False positives are possible at the individual tree level as we did not take destructive samples from every canker in a plot. However, our previous studies based on extensive culturing have indicated that these other *Phytophthora* species are very rare in our study area (Davidson et al. 2005, 2008, 2011, Anacker et al. 2007, Wickland et al. 2008, DiLeo et al. 2014).

Multilevel environmental covariates

We collected data on 11 environmental covariates to parameterize our survival analysis models. At the individual level, we included size (DBH) and species of each tagged oak stem. At the community level, we calculated species richness, bay laurel density, and inoculum load (sum of infected leaf counts) for each plot. Species richness was measured as the number of woody plant species rooted within or overhanging each plot during establishment. Bay laurel density was quantified as the number of living stems per plot, and could vary across years due to ingrowth, new stems entering the study upon reaching size requirements or exiting due to mortality. For our interannual estimates of inoculum load, we focused solely on bay laurel, as all other foliar hosts including tanoak have been shown to produce trivial amounts of inoculum compared to bay laurel in this region (Davidson et al. 2005, 2008, DiLeo et al. 2014). At the landscape scale, we used a GIS to calculate the following variables: host habitat connectivity, topographic moisture index (TMI), potential solar irradiation (PSI), and seasonal climate variability (i.e., monthly average values of minimum and maximum temperature and cumulative precipitation). To assess multiyear effects of climate variability, we averaged each of the three climate variables across four time intervals: (1) current year, (2) average of current and previous year, (3) average of current and previous two years, and (4) average of current and

previous three years. Additional details for each landscape level covariate can be found in Appendix S2.

Multilevel survival analysis

We used survival analysis to model effects of individual, community, and landscape characteristics on the time to infection of oak hosts (California black oak, canyon live oak, and coast live oak). Survival analysis is a collection of statistical procedures for data analysis in which the outcome variable of interest is time until an “event” (Kleinbaum and Klein 2012). We fit a shared frailty model consisting of three components: a frailty term with a gamma probability distribution, a baseline hazard function, and a term modeling the influence of observed covariates that are conditionally independent given the frailty. Frailty models extend the traditional Cox model when observations are clustered by groups or nested as levels (e.g., individuals within a household). In these models, the survival and hazard functions used to quantify the probability distribution of “time to event” data depend on an observable random quantity, a random effect or so-called “frailty,” that accounts for excess risk of distinct groups.

Following Rondeau et al. (2012), we let T_{ij} denote the grouped event times under study, with the j th ($j = 1, \dots, n_i$) individual tree stem belonging to the i th plot ($i = 1, \dots, G$). The hazard function, which is the instantaneous risk of infection at time t conditional on surviving until t or longer is

$$\lambda_{ij}(t|v_i) = v_i \lambda_0(t) \exp(\beta_{I,C,L}^T X_{ij}) = v_i \lambda_{ij}(t)$$

where $\lambda_0(t)$ is the baseline hazard function, X_{ij} is the covariate vector associated with the regression parameters β ($\beta_{I,C,L}$ is a vector of the β parameters across the three hierarchical levels of data collection and $\beta_{I,C,L}^T$ is its transpose to make $\beta_{I,C,L}^T X_{ij}$ the linear combination of the dependent variables), and v_i is the random effect associated with the i th group. The model assumes that the v_i are independently and identically distributed from a gamma distribution with expected value $E(v_i) = 1$ and $\text{Var}(v_i) = \theta$, i.e., $v_i \sim \Gamma(\frac{1}{\theta}, \frac{1}{\theta})$. We used the “counting process” data format to incorporate time-dependent covariates (Kleinbaum and Klein 2012), whereby multiple observations correspond to the same individual stem monitored through time. That is, a stem’s total at-risk follow-up time is subdivided into smaller intervals, allowing the values for predictor variables to change over the course of the study period.

Here, “time” refers to the number of years elapsing since an oak stem entered the study (i.e., upon reaching ≥ 2 cm DBH) and was then infected by *P. ramorum* or censored. Censoring refers to situations in which a stem was never infected during the study or was removed prematurely (e.g., human cutting). We used field plot as our frailty term because oaks rooted within the same

plot likely share underlying risk factors that vary spatially yet are difficult or impossible to measure (e.g., invasion history). Models were partitioned into four levels based on the hierarchical structure of data collection: (1) individual effects, (2) community effects, (3) landscape effects, and (4) full models parameterized with significant covariates from each nested model. Because the 30-yr PRISM climate data were not available for 2011, we fit our survival analysis models using field data from 2004 to 2010 only, resulting in the omission of four infection events from 2011 (out of 186).

Models were run using the ‘frailtypack’ package v2.3 (Rondeau et al. 2012) in R v2.14.2 (R Development Core Team 2010). Parameter coefficients were estimated using maximization of the penalized log-likelihood, which involves a smooth estimation of the baseline hazard function using an approximation by splines (Rondeau et al. 2006). The frailtypack package uses an approximate version of likelihood cross-validation criterion (LCV_v) to guide model selection. In semiparametric models, such as the shared gamma frailty model, LCV_a is approximately equivalent to the Akaike information criterion (AIC), whereby lower values indicate better predictive performance of estimating the hazard function (i.e., time to oak infection; Rondeau et al. 2012). A modified Wald test was used to deduce the statistical significance of the frailty term included to account for shared risk among oaks in the same plot (Rondeau et al. 2012).

To minimize problems due to multicollinearity, we used a correlation coefficient threshold of $|r| < 0.60$ for variable inclusion into models. As such, we retained average minimum temperature and precipitation as the two climate covariates because maximum temperature and precipitation exceeded this threshold. Bay laurel density and inoculum load were also highly collinear (Appendix S1: Fig. S2), yet we fit separate models including each covariate because we were interested in examining different mechanisms through which this reservoir host influences oak infection risk. All numerical variables were standardized to a mean of zero and standard deviation of one to eliminate effects from differences in measurement scales on model outcomes. We applied a natural log ($x + 1$) transformation to four variables in order to make them as normally distributed as possible and thus increase the validity of the statistical analyses: DBH, bay laurel density, inoculum load, and TMI. We ensured that models did not violate the proportional hazards assumption of Cox models, requiring the hazard function is constant over time, by examining model output using the ‘cox.zph’ call in the ‘survival’ R-package (Therneau 2014).

RESULTS

Disease progression

We monitored 1201 oak stems across 172 plots for signs of *P. ramorum* infection and SOD-induced

mortality across the 8-yr study period. The remaining 30 plots did not contain oak hosts. During plot establishment, we found 76 dead oak stems that we excluded from analyses because we could not reliably assess prior infection status (or species identity in many cases). Of the remaining 1125 living oak stems, 732 (65%) were coast live oak, 271 (24%) were California black oak, and 122 (11%) were canyon live oak. Coast live oak was the third most prevalent species overall (of host and non-host woody plant species), found in 124 plots (Appendix S1: Fig. S3). California black oak occurred in 75 plots while canyon live oak was found in 19 plots. As of 2011, 151 (21%) coast live oak and 35 (13%) black oak exhibited *P. ramorum* symptoms. We never observed symptoms of *P. ramorum* in canyon live oak. Background mortality was higher on a percentage basis in California black oak ($n = 56$; 21%) than coast live oak ($n = 58$; 8%). However, coast live oak had a significantly greater number of SOD-dead stems ($n = 49$; 7%) compared to black oak ($n = 8$; 3%) by the end of the study period ($t = 2.704$, $P = 0.007$). In addition, the number of coast live oak becoming infected as well as those dying following infection exhibited a continual increase across the study period, whereas infection and mortality were less severe over time in California black oak (Fig. 1). When size effects were analyzed, we found that larger stems of both oak hosts were more prone to infection than smaller stems (Fig. 2).

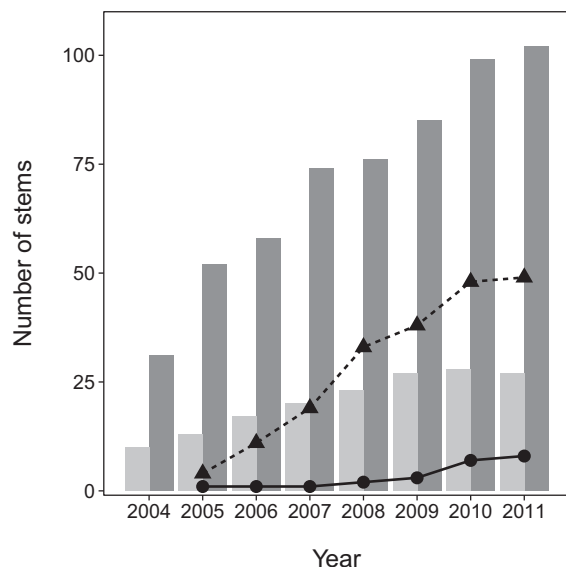


FIG. 1. The number of stems infected with *Phytophthora ramorum* for coast live oak (dark gray bars) and California black oak (light gray bars) across the 8-yr study period. Lines represent the number of stems dying following infection for coast live oak (triangles) and California black oak (circles). These raw counts have not been weighted by differences in the relative abundance of each species.

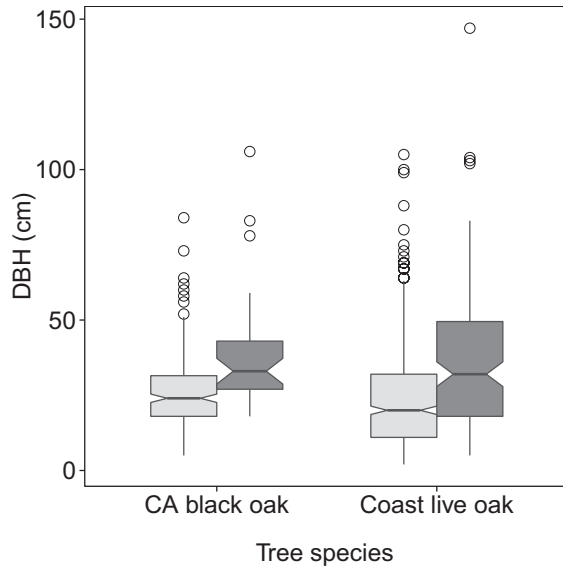


FIG. 2. Notched boxplots showing the relationship between *Phytophthora ramorum* infection status and tree diameter at breast height (DBH) for California black oak and coast live oak stems (infected stems shown in dark gray). Rectangles show the interquartile range (IQR), heavy lines show the median (50th percentile), and whiskers extend to ± 1.5 IQR to show variability outside the upper and lower quantiles. Data points beyond the end of the whiskers are outliers (open circles). Notches display the 95% confidence interval around the median (± 1.5 IQR/ \sqrt{n}). If the two boxes' notches do not overlap there is strong evidence (95% confidence) their medians differ (Chambers et al. 1983).

Environmental variables

For the survival analysis models, we discarded nine additional plots because they did not contain coast live oak or California black oak (163 plots). The number of coast live oak and California black oak stems per plot ranged from 1 to 29 (mean = 6.2, median = 5), while the number of infected stems ranged from 1 to 16 (mean = 2.4, median = 2) for both species. Across the entire plot network (202 plots), 54 woody plant species were observed during the 2004 vegetation surveys (Appendix S1: Fig. S3A). In plots containing coast live oak and/or California black oak, the average number of woody plant species was 7.6 (median = 7, range = 2–17) Appendix S1: Fig. S3B. The two plots exhibiting the lowest species richness each contained only bay laurel and coast live oak.

Bay laurel was the most prevalent host species across the plot network, occurring in 89% ($n = 180$) of the 202 plots and in 87% ($n = 141$) of the plots with oak hosts. Bay laurel density varied little across the study period, with mean stem density per plot ranging from 11.6 to 12.4 across years (with an overall range of 0–76 stems monitored per plot; Appendix S1: Fig. S4A). In contrast, bay laurel inoculum load exhibited greater interannual variability, with average symptomatic leaf count per plot ranging from 267 leaves in 2005 to 983 leaves in 2011 (Appendix S1: Fig. S4B). We found an

increasingly strong positive correlation between bay laurel density and inoculum load through time (range of r^2 across years, 0.49–0.80; Appendix S1: Fig. S2).

During the study, the region experienced interannual drought conditions (2001, 2007–2009) as well as above-average precipitation in some years (2003–2006; Fig. 3a). Average minimum temperature was less variable, although the 2004–2006 rainy seasons were slightly warmer overall (Fig. 3b). Interannual fluctuations in inoculum load were positively correlated with precipitation patterns across three time periods: (1) in 2006 when an abnormally wet rainy season corresponded

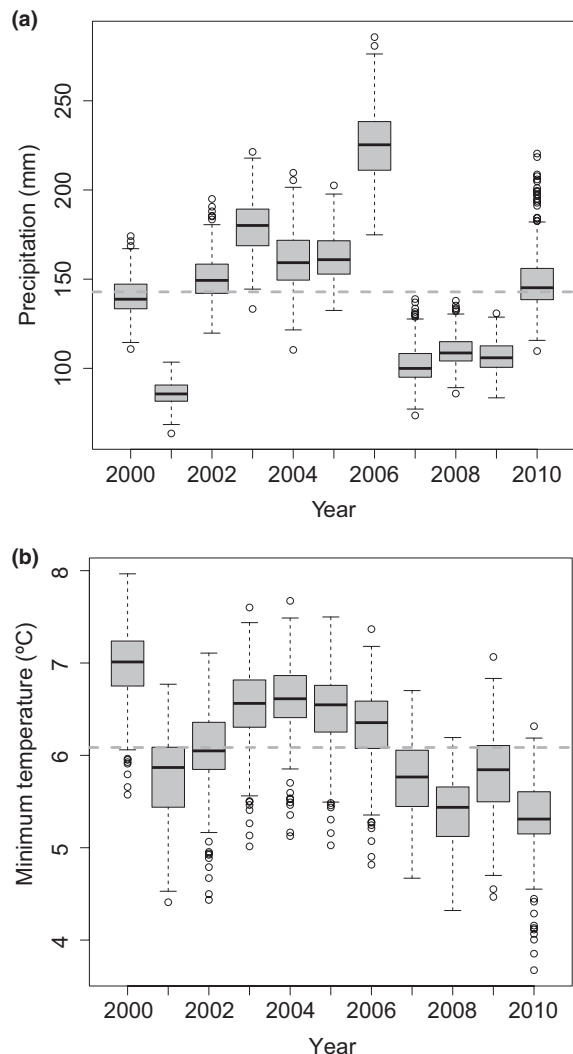


FIG. 3. Plot-level interannual variation in (a) mean cumulative rainfall (mm) and (b) mean minimum temperature $^{\circ}\text{C}$ during the 2000–2011 rainy seasons for plots containing coast live oak and/or California black oak ($n = 163$ plots). Dashed horizontal lines display the overall mean value across years. Rectangles show the interquartile range (IQR), heavy lines show the median (50th percentile), and whiskers extend to ± 1.5 IQR to show variability outside the upper and lower quantiles. Data points beyond the end of the whiskers are outliers (open circles).

to an increase in inoculum load; (2) during a three-year drought from 2007 to 2009 that corresponded to reduced inoculum load; and (3) from 2010 to 2011 when a return to average precipitation conditions led to an increase in inoculum load (Fig. 3a; Fig. S4B). We did not find a relationship between inoculum load and average rainy season minimum temperature.

Survival analysis models

Comparison of models (based on LCV_a) with climate effects calculated across the four time intervals revealed that average rainy season conditions during the current and previous year had the greatest influence on the progression of oak infection (Appendix S1; Table S1). At the community level, models parameterized with bay laurel inoculum load exhibited modest improvement compared to bay laurel density (see Appendix S1: Table S2 for results using bay laurel density). As such, we present results for models parameterized with these specific climate and community-level covariates.

The estimated coefficients in Table 1 give the linear additive effects of covariates on the log-hazard scale. Although the signs of coefficients are simple to

interpret (e.g., larger stem size increases infection risk), the magnitudes of effects are not easily translated. In contrast, exponentiated coefficients, referred to as hazard ratios, are more intuitive in that they are interpreted as a proportional shift in the hazard function due to a unit change in the associated covariate. Similar to an odds ratio in logistic regression, a hazard ratio of 1 (or a 95% confidence interval containing 1) indicates that there is no statistically discernible effect on infection risk from the covariate (Kleinbaum and Klein 2012). In survival analysis models without random effects, parameters describe the population-level relative risk. In our frailty models, however, the parameters are interpreted as a relative infection risk at the plot level, whereby the hazard ratios refer to comparisons within plots where individual stems share the same frailty. Across all hierarchical models (individual, community, landscape, and full), we found that the frailty term was statistically significant, implying greater unobserved heterogeneity in infection risk between plots and higher correlation of hazard risks for oaks within the same plot (Table 1).

After accounting for this latent heterogeneity, the individual-level model revealed that species identity and

TABLE 1. Shared gamma frailty models for time to *Phytophthora ramorum* infection in coast live oak and California black oak from 2004 to 2010 ($n = 163$ plots): (A) individual effects, (B) community effects, (C) landscape effects, and (D) the full model containing only statistically significant covariates from the three nested models.

Covariate	Coef.	SE	<i>P</i>	Hazard ratio	95% CI
(A) Individual effects					
Species identity†	-0.66	0.27	0.01	0.52	0.31–0.87
log(DBH)	0.82	0.11	<0.001	2.27	1.83–2.83
(B) Community effects					
Species richness	-0.56	0.14	<0.001	0.57	0.44–0.75
log(inoculum load) ‡	0.41	0.12	<0.001	1.51	1.19–1.92
(C) Landscape effects					
Host habitat connectivity	-0.18	0.17	0.27	0.83	0.60–1.15
log(topographic moisture index)	0.14	0.15	0.35	1.15	0.85–1.55
Potential solar irradiation	0.30	0.16	0.06	1.35	0.99–1.85
RS precipitation (2-yr mean)‡	1.58	0.13	<0.001	4.86	3.73–6.32
RS minimum temperature (2-yr mean)‡	0.33	0.17	0.06	1.39	0.99–1.94
(D) Full model					
Species identity†	-0.80	0.30	0.01	0.45	0.25–0.81
log(DBH)	0.81	0.12	<0.001	2.25	1.78–2.84
Species richness	-0.35	0.17	0.04	0.70	0.50–0.98
log(inoculum load)‡	0.37	0.14	0.01	1.45	1.10–1.91
Potential solar irradiation	0.28	0.17	0.10	1.32	0.94–1.85
RS precipitation (2-yr mean)‡	0.90	0.15	<0.001	2.46	1.81–3.33
RS minimum temperature (2-yr mean)‡	0.80	0.19	<0.001	2.24	1.53–3.27

Notes: Coef. is the penalized marginal log-likelihood estimate of each fixed regression coefficient (conditional on the frailty term); SE gives the standard errors of the estimated regression coefficients; *P* is the two-sided *P* value for the null hypothesis tests that each regression coefficient is 0 (or each hazard ratio is 1); Hazard ratio is the exponentiated coefficient for each covariate; and 95% CI represents the 95% confidence interval of each hazard ratio. DBH was measured in centimeters. RS stands for rainy season. (A) Approximate likelihood cross-validation criterion (LCV_a) = 0.106476, frailty parameter (θ) = 1.8392, $SE(\theta)$ = 0.393413. (B) LCV_a = 0.109573, θ = 1.28922, $SE(\theta)$ = 0.311402. (C) LCV_a = 0.106617, θ = 1.87317, $SE(\theta)$ = 0.414698. (D) LCV_a = 0.101869, θ = 1.98714, $SE(\theta)$ = 0.471247.

†Parameter estimate is for California black oak.

‡Time-dependent variables.

stem size were significant predictors of infection risk (Table 1A). At the community level, higher species richness and inoculum load were also significantly associated with oak infection patterns (Table 1B). In contrast, rainy season precipitation (averaged over two years) was the only landscape-level covariate that had a statistically significant effect on oak hazard risk. Mean monthly minimum temperature and PSI were marginally significant ($P = 0.06$), whereas host habitat connectivity and TMI were not predictive of infection risk (Table 1C).

Based on comparison of model LCV_a scores, we found that the full model—parameterized with significant covariates from the nested models including average minimum temperature and PSI—had the best predictive performance of estimating oak infection risk (Table 1D). The model revealed that *P. ramorum* infection risk is higher for coast live oak compared to California black oak. That is, holding other covariates constant, California black oaks had a reduced hazard risk on average per year by a factor of $e^{-0.80} = 0.45$. Moreover, larger stems of both oak species were more prone to infection than smaller stems (also see Fig. 2). The model also indicated that oaks in plots characterized by high bay laurel inoculum production had increased infection risk, suggesting the occurrence of pathogen spillover from bay laurels to oaks. Yet, after accounting for the effects of inoculum load (and bay laurel density; see Appendix S1: Table S2), we found evidence of a dilution effect, whereby oaks located in plots with higher plant species richness had reduced infection risk. After adjusting the hazard risk for these individual and community level effects, results further revealed that above-average warmer and wetter rainy season conditions during the current and previous years substantially increased the hazard rate of oak hosts. Although PSI was marginally significant in the landscape level model, we did not find an effect of solar radiation on risk in the full model. All other covariates that were significant in the nested models remained so in the full model.

DISCUSSION

Invasive plant pathogens are important agents of environmental change in forest ecosystems, yet their spread and impacts are often difficult to study due to the logistical challenges of data collection across sufficiently broad geographical regions and over meaningful time scales (Holdenrieder et al. 2004, Meentemeyer et al. 2012, Dillon et al. 2014). Multihost disease systems, such as sudden oak death, present additional complexity where species vary in key epidemiological traits (Power and Mitchell 2004, Mordecai 2011). The longitudinal, landscape-scale study described here allowed us to evaluate dynamics of a multihost disease system with asymmetric transmission and impacts among several host species. By incorporating ecological heterogeneity across multiple levels of organization—the

individual, community, and landscape—we determined the joint roles that individual hosts (species, size), their communities (reservoir host inoculum load, species richness), and their surrounding landscape contexts (seasonally varying climate conditions) play in the infection dynamics of sudden oak death.

Theoretical work in disease ecology points to the need for understanding the role of individual host heterogeneity in controlling susceptibility, which can drive both the rate and extent of pathogen invasions (Lloyd-Smith et al. 2005, Hawley and Altizer 2011). Host susceptibility is influenced by multiple factors that vary within a population, including genetic variation and physical host traits such as size and age (Garrett et al. 2009, McPherson et al. 2010, Cobb et al. 2012). However, few investigations of forest pathogens report variation in susceptibility that can be related to host tree characteristics (Bell et al. 2015). Most studies instead focus on describing mean infection rates for a given population (Hatala et al. 2011, Larson 2011, Preisler et al. 2012). In our study, repeated monitoring of individual oak stems from 2004 to 2011 revealed that *P. ramorum* infection risk was highest in larger coast live oak (followed secondarily by larger California black oak), whereas no symptomatic canyon live oak were observed. These findings incorporating long-term monitoring of three oak species across a broad environmental gradient are consistent with previous SOD research (McPherson et al. 2005, 2010, Brown and Allen-Diaz 2009, Swiecki and Bernhardt 2010, Metz et al. 2012). Numerous studies on other wildland forest pathogens have also found disease disproportionately killing larger trees, including comandra blister rust (*Cronartium comandrae*) on lodgepole pine (Jacobi et al. 1993), white pine blister rust (*Cronartium ribicola*) on white pine species (Smith and Hoffman 2001), and Port-Orford-cedar root disease (*Phytophthora lateralis*) on Port Orford cedar (Kauffman and Jules 2006). The mechanisms underlying size-susceptibility relationships will vary among disease systems and site conditions, but may arise through direct effects in which larger-sized hosts provide a greater surface area for pathogen contact, or indirectly through traits correlated with stem size, such as larger stems embodying a greater diversity of microhabitats suitable for pathogen colonization, including larger bark fissures or injuries that facilitate inoculum entry into the cambium (Smith and Hoffman 2001, Swiecki and Bernhardt 2006). For example, Jules et al. (2014) found that invasion of Port Orford cedar by *P. lateralis* had disproportionate impacts on larger trees (≥ 20 cm DBH) because pathogen spores transported downstream in water were more likely to encounter the well-developed root systems of larger trees. In SOD, oaks < 10 cm DBH are rarely found to exhibit *P. ramorum* symptoms (Kliejunas 2010). The cause of greater infection risk in larger stems is unknown, although bark fissures and surface area

increase with tree size, which may increase the likelihood of bole infection (Swiecki and Bernhardt 2006, Cobb et al. 2012).

Host–pathogen interactions in wildland ecosystems are influenced by complex spatial arrangements of tens to hundreds of plant species. As such, characterization of plant species assemblages—including species richness, identity, and density—is needed to better understand the role of biotic interactions in wildland epidemics (Burdon and Chilvers 1982, Power and Mitchell 2004, Hantsch et al. 2013). *P. ramorum* infects over 40 species native to California and Oregon coastal forests, yet most hosts are believed or documented to have low reservoir competence (Rizzo et al. 2005, Davidson et al. 2008). In communities with a single, highly competent host that is also the community dominant, pathogen transmission may be closely tied to population fluctuations of this species (Ostfeld and Keesing 2000, Mordecai 2011). Our finding that oak infection risk was higher in plots with greater bay laurel density and inoculum load estimates suggests that oak transmission pathways in the study region are driven primarily by spore production from foliar bay laurel infections. However, we also observed a negative association between oak infection risk and plant species richness in a community, even after accounting for the potentially confounding effects of bay laurel density. Haas et al. (2011) hypothesized that increased species diversity may reduce *P. ramorum* transmission by increasing the distance inoculum must traverse between bay laurel and oak hosts, whereby less competent hosts in the community act as physical barriers that intercept aerially dispersed inoculum and thus hinder pathogen establishment (Keesing et al. 2006). Biodiversity might also affect the average susceptibility of individuals in the community by influencing interspecific competition for limiting resources, thereby constraining the relative abundance of bay laurel in species-rich communities (Keesing et al. 2006). These findings suggest that although many plants in coastal California forests are hosts of *P. ramorum* (Rizzo et al. 2005), the presence of less competent, alternate hosts in the community may actually protect oaks by diluting impacts of the more competent, sporulating host bay laurel. In SOD-impacted forests, loss of biodiversity due to ongoing mortality of susceptible species in the community, combined with a lack of negative impacts by *P. ramorum* on bay laurel, may result in positive feedbacks between bay laurel abundance and pathogen population growth, precipitating further decline of oak and tanoak populations (Cobb et al. 2010, 2012, Metz et al. 2012).

Because natural plant communities are embedded within heterogeneous landscapes, we should expect that processes occurring at larger spatial extents play a key role in influencing disease dynamics at local scales (Holdenrieder et al. 2004, Meentemeyer et al. 2012, Dillon et al. 2014). For instance, the spread of pathogens and disease expression has been shown to be

influenced by landscape features including the spatial structure of host populations (Sullivan et al. 2011), as well as spatial heterogeneity in the abiotic environment including topographic and climate effects (Hatala et al. 2011). We found that warmer and wetter rainy season conditions over a two-year period substantially increased *P. ramorum* infection risk in oaks. Previous studies on SOD also observed that individual susceptibility to lethal bole cankers was greatest in years with frequent spring rains and that disease risk was especially severe when these conditions occurred two or more years in a row (Kanaskie et al. 2008, Swiecki and Bernhardt 2013). Literature reviews suggest that projected changes in climate will increase pathogen damage to forests through mechanisms altering pathogen development and survival rates, disease transmission, and host susceptibility (Desprez-Loustau et al. 2007, Dukes et al. 2009, Sturrock et al. 2011). In particular, foliar diseases caused by fungi and water molds (e.g., *Phytophthora* spp.) may be especially responsive to climate change because their ability to sporulate, disseminate, and infect is strongly associated with changes in temperature and precipitation (Desprez-Loustau et al. 2007, Kliejunas 2011). In the SOD system, consecutive years of favorable weather conditions, characterized by warmer temperatures coupled with rainfall events that are more frequent, longer in duration, or extend into the warmer spring months, may trigger an earlier onset of *P. ramorum* reproduction on bay laurel leaves, creating a longer period of inoculum buildup and increased probability of spillover to oaks (Davidson et al. 2005, 2008, Eyre et al. 2013, DiLeo et al. 2014). Our findings, which held after accounting for individual, community, and landscape-level heterogeneity, signal the potential for climate change to influence the establishment, spread, and severity of *P. ramorum* infection in California. Projected climate changes vary by geographic area for the state, but regional climate models generally agree that temperatures will increase statewide (Kliejunas 2011). Models differ considerably in their projections of precipitation, yet many indicate that the vast majority of precipitation will continue to fall during winter storms from the North Pacific (Kliejunas 2011).

Nonnative plant pathogens can cause dramatic and long-lasting changes to the composition of forests, especially when disease causes mortality of abundant tree species or species that play a foundational role in ecosystem functioning (Ellison et al. 2005, Lovett et al. 2006). The widespread mortality caused by *P. ramorum* in California and Oregon is a disconcerting example of a nonnative, virulent pathogen causing species-specific impacts to dominant trees, with the potential to substantially transform the composition of forests across a large and diverse region. To date, the challenge of observing, over meaningful times scales, the numerous and often complex interactions between forest pathogens, their hosts, and environmental

heterogeneity has limited our ability to understand and manage wildland disease epidemics (Meentemeyer et al. 2012, Dillon et al. 2014). In our longitudinal, landscape-scale study, we demonstrated how ecological heterogeneity across hierarchical levels of host–pathogen–environment associations collectively influences infection dynamics in a wildland forest disease. We found that larger individuals (especially coast live oak) are particularly susceptible to infection, that warmer and wetter landscapes in consecutive years increase disease risk, likely by facilitating pathogen spillover from the most competent reservoir host species, and that communities with richer species diversity have a buffering effect against infection risk. Our results point toward a future where many California oak woodlands may be dominated by the reservoir host bay laurel. We encourage spatially extensive, repeat measurement of disease progress in additional forest ecosystems, along with landscape simulation, to model dynamic feedbacks between changes in community composition and structure, increased inoculum availability, and altered weather conditions due to climate change.

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