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Ecosystem transformation by emerging infectious disease: loss of large tanoak from California forests

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Summary

1. Few pathogens are the sole or primary cause of species extinctions, but forest disease has caused spectacular declines in North American overstorey trees and restructured forest ecosystems at large spatial scales over the past 100 years. These events threaten biodiversity associated with impacted host trees and other resources valued by human societies even when they do not directly cause host extinction.

2. Invasion of *Phytophthora ramorum* and emergence of the forest disease sudden oak death has caused a large-scale decline of tanoak (*Notholithocarpus densiflorus*) in Californian coastal forests. Here, we describe structural changes to tanoak forests and develop predictive models of infection rates, mortality rates and changes in tanoak biomass and abundance by combining regionally extensive longitudinal field studies and mathematical modelling.

3. Pathogen-invaded stands had smaller average tanoak tree size and higher proportions of large dead tanoak trees compared with uninvaded stands. This pattern is caused in part by a positive relationship between tanoak size and mortality rate, as well as prolific basal sprouting from trees killed by the disease. Tanoak infection, mortality and biomass decline rates were positively related to the prevalence of infection in sporulation-supporting species, especially California bay laurel (*Umbellularia californica*).

4. We developed a stage-structured and spatially explicit mathematical model including species dynamics and *P. ramorum* transmission, where the long-term outcome of disease ranges from host extinction when densities of bay laurel are high to limited or no disease outbreak. Low densities of tanoak in a matrix of non-susceptible neighbouring species resulted in slow-enough transmission to retain overstorey tanoak, suggesting host-density thresholds may exist in real forests.

5. *Synthesis.* Tanoak is likely to persist in many disease-impacted forests via vegetative reproduction, but overstorey trees may be eliminated or greatly reduced in abundance, a pattern similar to other forest diseases that have emerged in the last century including chestnut blight and beech bark disease. Our results support a general model of disease-caused changes to forest trees useful for the analysis of emerging forest pathogens where vegetative reproduction, community-level epidemiology and stage-specific mortality rate interact to determine local disease intensity and host decline.

Key-words: community-driven transmission, disease ecology, forest disease, pathogen-caused extinction, *Phytophthora ramorum*, plant population and community dynamics, selective species removal, sudden oak death

Introduction

From a historical and epidemiological perspective, the lack of evidence for pathogen-induced extinction of tree-host species is surprising. Many novel forest pathogens have epidemiological characteristics associated with host endangerment (McCallum & Dobson 1995; Holt *et al.* 2003; de Castro & Bolker 2005) probably resulting from a lack of shared evolutionary history with their hosts. However, species extinction is typically caused by a combination of factors, which may include

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disease (cf. Pimm, Jones & Diamond 1988; Hanski 2005); this may explain why no pathogens have been confirmed as the sole cause of tree species extinction. Considering plant and animal diseases, pathogens have only been implicated in the permanent loss of host species that were also challenged by other factors commonly associated with extinction risk such as small population size, highly restricted habitat, extensive habitat loss or broad changes in the environment (Cunningham & Daszak 1998; de Castro & Bolker 2005; Smith, Sax & Lafferty 2006). Pathogens have increased the extinction risk for trees with restricted ranges, specialized habitats and small population sizes including butternut (*Juglans cinerea*; Loo 2009) and Florida nutmeg (*Torreya taxifolia*; Schwartz, Hermann & van Mantgem 2000; Smith *et al*. 2011). In contrast, diseases of widespread temperate trees such as chestnut blight, Dutch elm disease and beech bark disease have precipitated regional population declines by altering canopy distribution and biomass rather than causing extinction of their hosts (Swinton & Gilligan 1996; Paillet 2002; Garnas *et al*. 2011). In these examples, hosts often persist at local scales through basal sprouting from below-ground tissues that are not infected by the causative pathogens. This pattern suggests that understanding pathogen-caused reductions in host population size and biomass, and subsequent changes in community structure of widespread forest trees are more essential than forecasting host extinction risk *per se*, especially for pathogens that cause broad-scale shifts in size-class distributions. Many species are obligates on these overstorey trees, and the loss of these hosts poses a direct threat to local or regional biodiversity (Orwig 2002; Ellison *et al*. 2005; Rizzo, Garbelotto & Hansen 2005; Loo 2009; Wardle *et al*. 2011).

The emerging fungal-like pathogen *Phytophthora ramorum*, which causes the disease sudden oak death, has resulted in a rapid decline of tanoak (*Notholithocarpus densiflorus*), a canopy tree native to California and Oregon (Rizzo, Garbelotto & Hansen 2005). Dispersal of *P. ramorum* spores is primarily through local rain splash (< 20 m) with occasional dispersal events up to several kilometres (Davidson *et al*. 2005; Mascheretti *et al*. 2008). This bimodal dispersal pattern has led to rapid pathogen establishment throughout the coastal range of tanoak (Hansen *et al*. 2008; Meentemeyer *et al*. 2008; Filipe *et al*. 2012). Conservation-focused management strategies for sudden oak death are needed, especially in northern California where wide-spread disease is expected to emerge in forests with high tanoak density and biomass over the next 10–20 years (Lamsal *et al*. 2011; Meentemeyer *et al*. 2011). *Phytophthora ramorum* infects over 40 native coastal plant species, but varies in its ability to infect and sporulate on different plant parts and in its ability to kill different host plant species. Within trees, sporulation occurs on infected leaves, but not on infected woody stems. Sporulation is also dependent on environmental conditions in space and time; variation among years can reach an order of magnitude, with the highest sporulation rates during warm spring rain events and in cooler and wetter communities such as coast redwood forests (Davidson, Patterson & Rizzo 2008; Davidson *et al*. 2011, 2005). Sporulation is greatest on California bay laurel (*Umbellularia californica*), significantly lower on tanoak, and no sporulation has been documented on true oak species (*Quercus* spp.; Davidson *et al*. 2005, 2011). Community composition, especially the prevalence of bay laurel, is a critical driver of pathogen establishment and disease emergence from stand to landscape scales (Maloney *et al*. 2005; Rizzo, Garbelotto & Hansen 2005; Davidson *et al*. 2011; Meentemeyer *et al*. 2011; Haas *et al*. 2011). Although susceptibilities of bay laurel and tanoak are equivalent, infection in bay laurel causes no known deleterious impacts either physiologically (DiLeo, Bostock & Rizzo 2009) or at the population-level (Cobb, Meentemeyer & Rizzo 2010). In contrast, tanoak and oak are frequently killed following *P. ramorum* infection (Davidson *et al*. 2005; McPherson *et al*. 2010). These selective species removals lead to changes in forest community structure and composition, often increasing the relative dominance of bay laurel or redwood while decreasing the relative dominance of tanoak (Brown & Allen-Diaz 2009; Cobb, Meentemeyer & Rizzo 2010; Ramage, O'Hara & Forrestel 2011).

Tanoak was harvested extensively in California from 1890 to 1950 when tannins extracted from bark supported a regional tanning industry (Bowcutt 2011). The advent of chemical tanning compounds eliminated the economic value of tanoak, but the legacy of bark extraction led to increased tanoak densities due to prolific basal sprouting from below-ground tissues following cutting. Tanoak is now a frequent target of herbicide applications because it reduces the growth of neighbouring economically valuable conifers (Bowcutt 2011). Similar to most species removals (Wardle *et al*. 2011), the potential loss of tanoak due to sudden oak death has unknown ramifications for biodiversity conservation and ecosystem function in many California forests. Tanoak often forms the mid- and lower canopy strata of redwood forests where it is an important component of canopy structure and diversity (Waring & O'Hara 2008). Additionally, tanoak is often the sole ectomycorrhizal host in redwood forests where it supports ectomycorrhizal fungal diversity comparable to oak-dominated communities (Rizzo, Garbelotto & Hansen 2005; Bergemann & Garbelotto 2006). Although tanoak insect communities are poorly studied, a survey of microlepidopteran communities on coast live oak (*Quercus agrifolia*) showed that 23% were monophagous (Opler 1974). These host traits, in combination with high pathogen virulence and generalism, suggest that loss of overstorey tanoak from redwood forests will threaten tanoak-obligate flora and fauna, reduce biodiversity and alter functional processes (McCallum & Dobson 1995; Power & Mitchell 2004; de Castro & Bolker 2005; Rizzo, Garbelotto & Hansen 2005; Wardle *et al*. 2011).

Given the regional decline of tanoak, it is important to understand the extinction risk posed by *P. ramorum*, forecast rates of population decline and changes in host biomass. However, many questions pertinent to biological conservation remain open for sudden oak death and other wide-spread forest diseases. When are forest diseases likely to result in local tree extinctions compared to the loss of overstorey trees? For management applications designed to reduce disease, which community conditions retain overstorey tanoak and how do these inform conservation-focused disease management

strategies? We address these questions for sudden oak death using three approaches: (i) We analyse factors that affect tanoak infection and mortality rates using a large, annually surveyed and geographically extensive population sample. (ii) We use the results of the empirical analysis to design and parameterize a dynamic epidemiological model. (iii) We use the epidemiological model to predict medium- to long-term effects of *P. ramorum* on tanoak populations, changes in forest structure, and the role of epidemiological and host characteristics on tanoak persistence within pathogen-invaded stands.

Materials and methods

STUDY DESIGN

This study employs two separately established plot networks; one invaded network where *P. ramorum* has been confirmed since 2002 (110 plots; Maloney *et al.* 2005; Cobb, Meentemeyer & Rizzo 2010) and an uninvaded network of sites that were *P. ramorum*-free during plot establishment between 2003 and 2005 (95 plots; Cobb, Meentemeyer & Rizzo 2010; Appendix S1, Fig. S1 in Supporting Information). All plots are circular, 500 m^2, randomly located within stands, and have a distance of at least 100 m between plot boundaries. Past logging and wildfire have occurred at most sites, but the dates of these events are typically unknown. For the majority of invaded study sites, precise arrival dates for the pathogen are also unavailable, but the disease was established at most sites by 1998 (Maloney *et al.* 2005).

FIELD SURVEYS AND LABORATORY METHODS

In both the pathogen-invaded and uninvaded plots, all stems >1 cm d.b.h. (diameter at breast height) were mapped, tagged, identified to species and measured for diameter. Canopy position of each stem was also recorded on a four-class scale (understorey, intermediate, co-dominant or dominant), which reflects the position of a tree in the canopy relative to its neighbours. For each tree, symptomatic tissue was collected when present and was returned to the laboratory for pathogen isolation on a *Phytophthora*-selective medium (PARP; Davidson *et al.* 2005). A full census of invaded plots was conducted in 2002, and partial follow-up censuses were conducted from 2003 to 2006 in which five previously uninfected and previously confirmed infected trees of all species were randomly chosen within each plot and surveyed between April and June. A final complete census was undertaken in 2007 between May and September. In this survey, newly symptomatic individuals were sampled between May and July (60 of 110 plots) to avoid biasing infection estimates with samples from late summer months when pathogen recovery in culture is poor. All trees in invaded plots were surveyed for mortality in 2007. In the invaded plot network, 1227 of 2029 tanoak had confirmed infections across 13 sites. Within uninvaded plots, 1583 tanoak were surveyed across 13 sites.

DATA ANALYSIS

We used the Weibull accelerated failure time model (Kleinbaum & Klein 2005) to estimate tanoak infection and mortality rates in invaded plots. This approach to survival modelling allowed us to assess effects of individual and community variables on disease progression and to estimate survival times for tanoak. The survival function has the form $S(t_1,t_2) = \exp[-\lambda(t_2 - t_1)^\rho]$ where t_1 is the last time an individual was observed alive or uninfected, t_2 is the first time an individual was observed dead or infected, the scale parameter λ is a function of the covariates of interest, and the shape parameter ρ determines the time dependency of the hazard. The shape parameter (ρ) provides additional insight into changes in baseline rates: when $\rho > 1$, the rate accelerates; when $\rho < 1$, the rate slows; and when $\rho = 1$, the rate is constant over time. We emphasize that each infection and mortality event is known to occur over an interval introduced by the partial surveys from 2003 to 2006 (interval censoring). Censoring intervals in our data are of random duration for each tree, which allowed us to use a conventional survival model. Survival models can accommodate frailties (random effects); we include 'study site' as a frailty to address correlation between plot response and unmeasured factors, especially invasion history (Kleinbaum & Klein 2005). We used maximum likelihood to estimate parameter values by fitting the model to observed infection and mortality times. We quantified the impact of several plot-level variables on tanoak infection and mortality rates, including tree size (d.b.h.), infected tanoak prevalence, infected bay laurel prevalence and redwood density. At the landscape scale, we also included an estimate of inoculum external to each plot using a published data set of plot-level force of infection (Meentemeyer *et al.* 2008). Here, force of infection is the sum of the inverse distances to all known *P. ramorum* infections derived from a spatially extensive, independent data set of pathogen distribution. The proportional hazards and Weibull distribution assumptions were evaluated following Kleinbaum & Klein (2005; Appendix S1, Figs S2 and S3). The survival models were validated by fitting the data without the final year of records (2007; hold-out data method) and regressing expected failure times (infection and mortality respectively) against a binomial variable indicating tree status in 2007. Model adequacy was evaluated using the receiver operator characteristic (ROC). If ROC values were <0.7, the model was rejected. Mortality models were subjected to a second validation with an independent data set from the Big Sur region (66 plots, 1712 tanoak stems; Metz *et al.* 2011) using the same ROC criteria but substituting mortality from all sources as the dependent variable and tree vigour as a frailty. These model adjustments were necessitated by differences in pathogen surveys, which also preclude validation of infection models with Big Sur data. We also calculated ROC values of the predicted values from models fitted with the entire data set against tree status in 2007.

EPIDEMIOLOGICAL MODEL

To gain insight into future trends in tanoak population decline and changes in canopy structure and stand composition, we developed a spatially explicit, density-dependent, stage-structured model of tanoak population dynamics under an emerging epidemic of *P. ramorum*. Model parameters were estimated using our plot-level measurements. The model is defined on a 20-ha lattice consisting of 400 cells, each with an area of 500 m^2 (the size of our study plots). Each cell is populated with tanoak, bay laurel and epidemiologically unimportant species among which redwood is predominant in this plot network (Maloney *et al.* 2005). On this lattice, we superimposed pathogen transmission dynamics (within and between cells), disease-independent mortality and seedling establishment in unoccupied space. The infection dynamics of each species were defined using standard SIS compartments: *Susceptible* (S) and *Infected* (I) for tanoak and bay laurel. Like many hardwood trees, disease-killed tanoak develops basal sprouts from below-ground tissues (Cobb, Meentemeyer & Rizzo 2010; Ramage, O'Hara & Forrestel 2011), and we include this process in the model.

The dynamics governing the proportions of each species (1, 2 and 3 represent tanoak, bay laurel and redwood, respectively), in each

epidemiological state (Susceptible (S) or Infected (I)) and tanoak size class ($i = 1,..,4$), and in each cell location (x), are described by the system of differential equations:

$$\begin{cases} \frac{dS_{1,i,x}}{dt} = \delta_{1,i}[B_{1,x}E_x + r\alpha_{1,i}I_{1,i,x}] - d_{1,i}S_{1,i,x} - \Lambda_{1,i,x}S_{1,i,x} + \mu_1 I_{1,i,x} \\ \qquad + a_{i-1}S_{1,i-1,x} - a_i S_{1,i,x} \\ \frac{dI_{1,i,x}}{dt} = -\alpha_{1,i}I_{1,i,x} - d_{1,i}I_{1,i,x} + \Lambda_{1,i,x}S_{1,i,x} - \mu_1 I_{1,i,x} + a_{i-1}I_{1,i-1,x} - a_i I_{1,i,x} \end{cases}$$

$$\begin{cases} \frac{dS_{2,x}}{dt} = b_2(S_{2,x} + I_{2,x})E_x - d_2 S_{2,x} - \Lambda_{2,x}S_{2,x} + \mu_2 I_{2,x} \\ \frac{dI_{2,x}}{dt} = -d_2 S_{2,x} + \Lambda_{2,x}S_{2,x} - \mu_2 I_{2,x} \end{cases}$$

$$\frac{dS_{3,x}}{dt} = b_3 S_{3,x}E_x - d_3 S_{3,x} \qquad \text{eqn 1}$$

As tanoak density among tree size classes is of central interest in our study, the model includes size classes for tanoak, but not for other species. This simplification avoids assumptions about life cycle parameters for poorly studied species such as bay laurel. For tanoak, $\delta_{1,i}$ in (1) ensures that recruitment and resprouting contribute to the smallest size class ($\delta_{1,i} = 1$ for $i = 1$ and $\delta_{1,i} = 0$ for $i \neq 1$) and a_i controls transition (growth) of tanoak from class i to the next larger-size class $i + 1$ ($a_0 = 0, a_4 = 0$). The parameter $\alpha_{1,i}$ is the rate of disease-caused mortality of tanoak of size class i, and r is the probability that tanoak killed by disease develops basal sprouts. This formulation of resprouting is parsimonious, but allows tanoak with dead aboveground biomass to persist through basal sprouts while ensuring that individuals that do not resprout eventually die (at rate $(1 - r)\alpha_{1,i}$). For tanoak and bay laurel, the parameters μ_1 and μ_2 are the rates of recovery from infection to the susceptible state. For each species, we include the parameter d_j, representing rates of disease-independent mortality and b_j for the rates of recruitment from seed per tree ($j = 1,2,3$). Tanoak recruitment from seed is described by:

$$B_{1,x} = \sum_{i=1}^{4} b_{1,i}(S_{1,i,x} + I_{1,i,x}); \qquad \text{eqn 2}$$

The establishment of species in each cell is a density-dependent process conditioned by the proportion of unoccupied space available for recruitment:

$$E_x = 1 - W_1 \sum_{i=1}^{4} \omega_{1,i}(S_{1,i,x} + I_{1,i,x}) - W_2(S_{2,x} + I_{2,x}) - W_3 S_{3,x}; \qquad \text{eqn 3}$$

where W_j is a relative measure of per-capita space used by species j (a ratio of actual space to mean space occupied) and $\omega_{1,i}$ is a similar measure for size classes within the tanoak population. Therefore, species recruitment is a simplified competitive process based on available space for establishment, and we define our species such that there is no explicit difference in species colonization (under equal densities) by taking $W_j = 1$. Lastly, species also interact via the force of infection on tanoak and bay laurel from infections in the same cell and in adjacent cells (Filipe & Gibson 2001):

$$\Lambda_{1,i,x} = f_{wth}\left[\beta_{1,i}\sum_{j=1}^{4} I_{1,j,x} + \beta_{12}I_{2,x}\right] + f_{btw}\sum_{y\in N(x)}\left[\beta_{1,i}\sum_{j=1}^{4} I_{1,j,y} + \beta_{12}I_{2,y}\right],$$

$$\Lambda_{2,x} = f_{wth}\left[\beta_{21}\sum_{j=1}^{4} I_{1,j,x} + \beta_2 I_{2,x}\right] + f_{btw}\sum_{y\in N(x)}\left[\beta_{21}\sum_{j=1}^{4} I_{1,j,y} + \beta_2 I_{2,y}\right]; \qquad \text{eqn 4}$$

where $\beta_{1,i}$ and β_2 are the rates of infection within species and β_{12} and β_{21} the rates of infection between species. Tanoak size classes have different susceptibility, but equal infectivity (Davidson, Patterson & Rizzo 2008). Transmission of infection within cells and between adjacent cells (where $N(x)$ is the set of cells adjacent to cell x) is determined by f_{wth} and f_{btw} (where $f_{wth} + f_{btw} = 1$), which are the proportions of spores deposited within and between adjacent cells, respectively.

The initial condition of (1) is a forest in dynamic equilibrium in terms of species composition and distribution of tanoak size classes. The pathogen is introduced to a single infected cell at the centre of the lattice. The simplifying assumption of dynamic equilibrium before pathogen introduction allowed us to estimate several parameters (d, b and E) and to use observed mean stem densities as one scenario of initial species composition. Briefly, we estimated the death rates (d) from observations in the uninfected plots and set to zero the transmission parameters (β_i) and initial infections. We then used the model to estimate seed recruitment rates (b) and available space (E) that maintained a constant tanoak population size over a 1000 year model run, with the additional condition that seed production increases with tanoak tree size. We further simplified the model by assuming that the system is not exposed to external inoculum, a non-significant covariate in the survival analysis. We ran the model up to 100 years following pathogen introduction in three scenarios of initial composition: (i) observed mean densities of each species; (ii) high tanoak density, but no bay laurel; and (ii) low tanoak density, high redwood density and no bay laurel. We emphasize to the reader that these three initial sets of composition correspond to two epidemiological scenarios; in the first, two species transmit the pathogen, but in the latter two scenarios, transmission is from tanoak only. See Appendix S1 for a derivation of the model using a sequence of simpler literature-related models. Exact parameter values can be found in Table S1 from which the model results can be reproduced.

Results

STAND STRUCTURE IN *PHYTOPHTHORA RAMORUM*-INVADED STANDS

Mortality levels were low for tanoak in uninvaded plots and generally decreased with tree size (Fig. 1a). This pattern was reversed in *P. ramorum*-invaded stands where the proportion of dead individuals increased with increasing size class. Consequently, the relationship between stem density and stem size of living trees differed substantially between pathogen-invaded and uninvaded stands, with greater frequency of large tanoak stems in uninvaded stands (Fig. 1b). Although the frequency of dead trees was much higher in invaded stands, particularly for large trees, the densities of living trees < 6 cm d.b.h. were similar between the two stand types.

INFECTION, MORTALITY AND BIOMASS DECLINE RATES

Tree size and community composition affected tanoak infection and mortality rates (Table 1). Tanoak infection rates accelerated moderately over the course of measurements ($\rho = 1.27$; $P < 0.001$) with expected median time to infection for the entire sample of 2.5 years (2.7 mean). Understory tanoak, generally trees < 10 cm d.b.h., had greater risk of infection compared with tanoak in the overstorey (Table 1 and

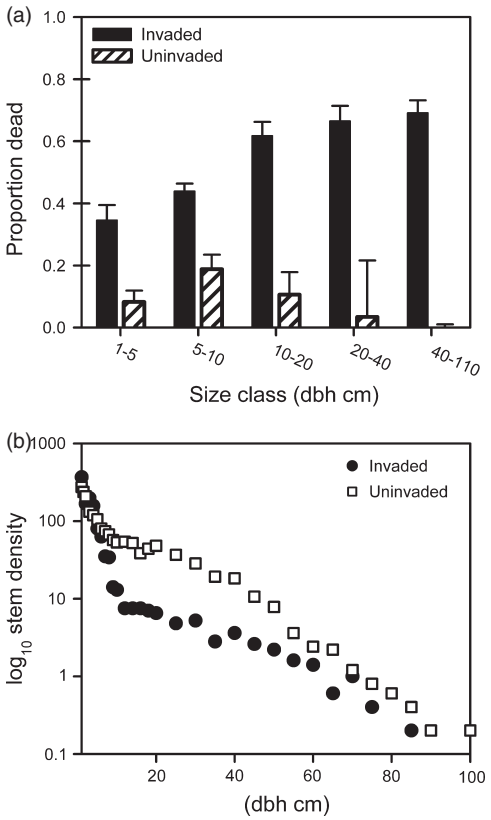

Fig. 1. A comparison of size distribution of dead and live tanoak (*Notholithocarpus densiflorus*) trees in pathogen-free (uninvaded) and invaded stands. Mean proportion of dead trees are shown in (a) with one binomial standard error. In (b), the size (d.b.h.) distribution of live trees in pathogen-uninvaded and pathogen-invaded plots are shown on a log-linear scale.

Fig. 2a). Within plot densities of infected tanoak and bay laurel were also negatively associated with time to tanoak infection (Fig. 2), suggesting that pathogen prevalence in both species leads to higher tanoak infection rates. Notably, a set of trees from the McWay Canyon site (Fig. S1) have long expected times to infection and are apparent in Fig. 1a as the set of trees above the 75th percentile and distinct from the rest of the sample population. This site is notable for low pathogen prevalence, low densities of tanoak and bay laurel and low per-capita infection rates.

Mortality rates of infected tanoak were significantly affected by individual and community-level factors (Table 1). Pathogen-caused mortality rates were constant from 2002 to 2007 after adjusting for the temporal increase in infected hosts ($\rho = 1.06$; $P = 0.28$). The median time to mortality of infected tanoak was 11.4 years (12.8 years mean). Expected time to mortality was longer, more variable and more strongly influenced by community-level factors and tree size than median times to infection. The expected time to mortality of tanoak with confirmed *P. ramorum* infections decreased with increasing tree size. For example, expected survival time was ~14 years following infection for the first quartile of tree size (1–2 cm d.b.h.), but only ~2 years following confirmation of infection in the fourth quartile (10.4–107 cm d.b.h.). Similar to infection rate, mortality rate also increased with prevalence of infection in tanoak and bay laurel (Table 1 and Fig. 2). The external inoculum variable did not significantly influence tanoak infection or mortality rates (Table 1), indicating that local conditions were more important than regional pathogen prevalence for these processes.

When the estimates of times to infection and mortality were combined, declines in tanoak basal area were sigmoidal with 50% basal area loss predicted 9 years after pathogen establishment, but 99% mortality after 63 years (Fig. 3). The yearly cumulative dead basal area estimated from the survival analysis was in close agreement with observed cumulative dead basal area in invaded plots (observed = 0.923*estimated; $r^2 = 0.885$, $N = 6$). In our network of field plots, 50% loss of tanoak basal area was reached in 2006, about 11 years following pathogen establishment. The asymmetry between times to 50% and 99% loss of initial tanoak basal area demonstrates the importance of tree size and community factors in determining infection and mortality rates. Tanoak trees with the longest expected survival times were small trees in stands with low densities of tanoak and bay laurel.

MODEL RESULTS

The epidemiological model suggests that tanoak is likely to persist as a component of many stands, but that tanoak canopy structure, biomass and density will shift substantially. When stands are composed of bay laurel and tanoak, and thus include two sources of pathogen transmission, the tanoak population declines towards extinction. This extinction event occurs substantially later than the 100-year temporal context of the (deterministic) model runs (Fig. 4a). When tanoak is the sole pathogen-transmitting species, *P. ramorum* does not cause a deterministic trend towards extinction, but large trees are lost from the modelled stand (Fig. 4b). In either transmission scenario (one or two sporulating species), disease increases the density of small trees and removes large trees in the model stands. Rates of disease emergence were highly sensitive to the initial prevalence of pathogen-transmitting species relative to the conglomeration of redwood and other species, which have no epidemiological role in the model. The most extreme example is found when the initial stand composition is $< 8\%$ tanoak (no bay laurel). In this case, disease does not emerge in the model, tanoak populations are constant for the entire simulation, and large tanoak is retained in the stand (Fig. 4c). In the model, pathogen establishment exhibits threshold behaviour when the initial tanoak composition is near 8% of stems, such that slightly higher tanoak prevalence or a small increase in transmission from a second species results in disease emergence and eliminates large tanoak trees.

Model population dynamics were sensitive to the probability of basal sprouting (r). When stands include bay laurel, a reduction in basal sprouting accelerates tanoak population decline, but slightly increases the time to loss of overstorey tanoak. When $r = 0$ and tanoak is the sole transmitting species, large trees remain in the stand up to initial tanoak densities of 15%. When resprouting rate is set to very high levels

Table 1. Estimated coefficients (c_i) and validation for the Weibull accelerated failure time survival model with scale (λ) and shape (ρ) parameters for tanoak infection and mortality from *Phytophthora ramorum*. Expected median times (t_m) to tanoak infection and mortality are given by $t_m = [\ln(2)/\lambda]^{(1/\rho)} = [\ln(2)\exp(\Sigma c_i X_i)]^{(1/\rho)}$. Significant coefficients c_i ($P \leq 0.05$) are reported with standard error in parenthesis

Parameter/coefficient	Infection	Mortality
ρ	1.27 (0.04)	1.00 (0.05)
Intercept	1.097 (1.21)	3.75 (0.18)
Tree size (d.b.h. cm)	0.005 (0.002)	−0.031 (0.003)
Bay laurel infection*	−0.0012 (0.0002)	−0.0037 (0.0004)
Tanoak infection*	−0.0013 (0.0001)	−0.0027 (0.0003)
Redwood density	ns	ns
Primary infection†	ns	ns
Frailty effects		
Site (variance)†	0.609	0.035
Validation† (receiver operator characteristic)		
Full data set	0.700	0.844
Hold-out data	0.700	0.811
Independent data	na	0.839

na, parameter not applicable to model; ns, coefficient not significant.
*The number of individuals per ha with confirmed *P. ramorum* infections at the time of observed infection/mortality.
†See Data Analysis for further details.

($r > 0.9$), disease increases tanoak dominance although most tanoak remain in small size classes (< 10 cm d.b.h.).

Discussion

Our results show that sudden oak death will remove large tanoak trees from the overstorey of many coastal California forests, but tanoak extinction is unlikely to occur solely through mortality caused by *P. ramorum*. These results fit the general patterns of meta-analyses focusing on the role of pathogens in host extinction risk (de Castro & Bolker 2005; Smith, Sax & Lafferty 2006). The immediate conservation threat from sudden oak death and similar forest diseases is the rapid and extensive loss of overstorey trees, and the subsequent impacts to flora and fauna following shifts in community composition and canopy structure (Ellison *et al.* 2005). Sudden oak death is likely to reduce average tree size of tanoak, shift community composition and restructure the canopy of impacted forests (compare with Swinton & Gilligan 1996; Paillet 2002; Garnas *et al.* 2011). Loss of overstorey tanoak is likely within 30 years of pathogen establishment in many forests (Figs 3 and 4) due to the high competency of tanoak and bay laurel in transmitting *P. ramorum* and the positive relationship between tanoak tree size and mortality rate (Fig. 2; McPherson *et al.* 2010; Ramage *et al.* 2012).

Emerging forest pathogens frequently have greater impacts on large trees. Large trees may be more susceptible due to higher contact rates, higher likelihood of attack by pathogen vectors or a positive relationship between pathogen growth and tree size (Swinton & Gilligan 1996; Paillet 2002; Kauffman & Jules 2006). While several studies have shown that bay laurel and tanoak strongly influence *P. ramorum* establishment and disease emergence (Maloney *et al.* 2005; Meentemeyer *et al.* 2008; Cobb, Meentemeyer & Rizzo 2010; Haas *et al.* 2011), our survival analysis suggests higher rates of mortality in large tanoak are independent of vegetation composition. The cause of more rapid mortality rates in larger trees is unknown, although bark fissures and surface area increase with tree size, which may increase the likelihood of bole infection (Swiecki & Bernhardt 2006). Parke *et al.* (2007) documented decreased sap flow in infected tanoak stems and suggested that multiple infections on an individual are likely to increase physiological stress. Multiple infections within an individual tanoak or on a single tanoak bole become more likely as sources of infection increase, and we found that the effect of infection prevalence in bay laurel on per-tanoak mortality rate is greater than that of infection prevalence in tanoak (Table 1). Given that bay laurel supports much greater *P. ramorum* sporulation, these are the expected patterns if multiple infections increase with tree size and subsequently increase mortality rate.

This is the first study to estimate tree level *P. ramorum* infection and mortality rates across a wide range of community conditions and over a large geographical area. Also, our study is one of the few that estimate forest disease impacts using temporally intensive measurements (see also Kauffman & Jules 2006; Garnas *et al.* 2011). Our estimated survival times for tanoak are consistent with survival times of overstorey trees (> 25 cm) estimated from annual aerial surveys over large areas (Filipe *et al.* 2012), but longer than estimates reported by McPherson *et al.* (2010). The discrepancy between the present study and McPherson *et al.* is likely due to our inclusion of small trees (1–5 cm d.b.h.), which have the longest survival time following infection. We suggest that average survival time is a misleading measure of pathogen impacts when mortality rates are heterogeneous within a population because individuals that are highly susceptible or have especially low contact rates will skew estimates of the mean (Kauffman & Jules 2006). Predictions of mortality rates and progression of sudden oak death at the stand level will be most accurate when they account for tree size distribution and the densities of infected tanoak and bay laurel.

Our epidemiological model is designed to illustrate biotic factors affecting decline in tanoak populations invaded by *P. ramorum* and not to predict precise time scales for extinction. Therefore, several caveats must be recognized when interpreting the results or extending the model to other systems. The quantitative predictions of the model are sensitive to parameter values of mortality and transmission rates. However, the qualitative predictions of tanoak persistence, changes in size distribution and the dynamics introduced by a second sporulation-supporting species are robust to changes in these parameter values across a plausible range implied by the data and independent work (e.g. Fig. 1; Davidson *et al.* 2005; Cobb, Meentemeyer & Rizzo 2010). Additionally, while the realism of the model is greatly improved by its spatially explicit formulation (Filipe & Gibson 2001; Park, Gubbins & Gilligan 2001), the model does not incorporate individual-level stochasticity and thus is not adequate to forecast extinction when host

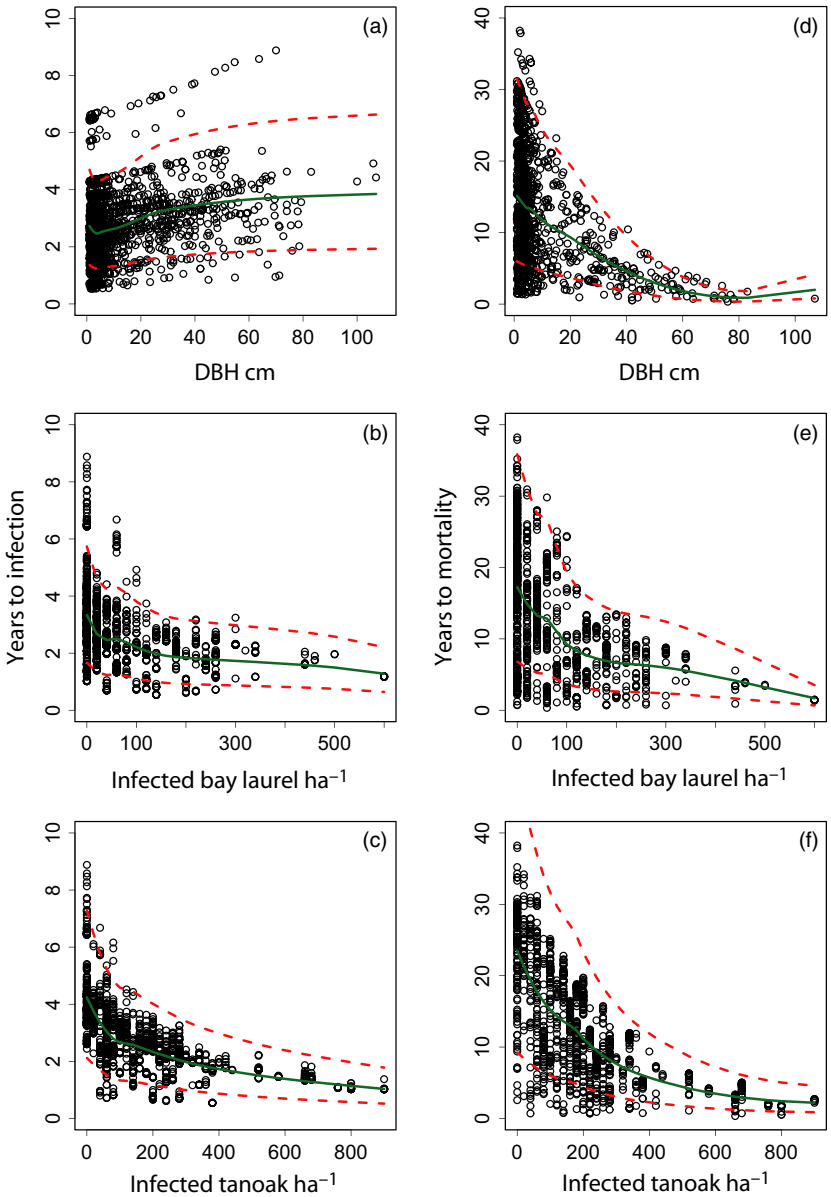

Fig. 2. Expected times to tanoak infection (a–c) and mortality (d–f) from *Phytophthora ramorum*. The top panels show tree-size effects, and the middle and bottom panels show community-level effects from prevalence of infection in bay laurel and tanoak, respectively. Points are the maximum likelihood estimates of survival times shown with the median (solid line), 25th and 75th percentiles (dashed lines).

population sizes become small (Hanski 2005). For example, Engen, Saether & Møller (2001) showed that deterministic models tend to overestimate the time to extinction of barn swallow (*Hirundo rustica*) populations due to a lack of stochasticity. Therefore, we expect that natural forests may experience slower or faster tanoak population declines than predicted by the epidemiological model due to uncertainty in our parameter estimates, the influence of stochastic events and unidentified epidemiological drivers (Engen, Saether & Møller 2001; Hanski 2005).

Individual-level variation is an important source of uncertainty in our models, but also a potential resource for managing sudden oak death and reducing its impacts in California landscapes. For example, Hayden *et al.* (2011) showed that rates of *in planta* pathogen growth vary among tanoak individuals within populations. Genetically associated variation in transmission and susceptibility may also occur among tanoak populations and could be a source of unexplained variation in the infection and mortality models (Fig. 2). Davidson *et al.* (2011) showed that infected bay laurel leaves are much more likely to be abscised than uninfected leaves and that abscission rate is variable across forest type; these processes suggest a finite and environmentally driven infectious period in bay laurel. Susceptibility and infectious period are likely affected by genetic variation within tanoak and bay laurel, and the model results suggest factors that decrease in β or increase in μ (the respective parameters) would slow disease progression and reduce the likelihood of tanoak extinction in real forests.

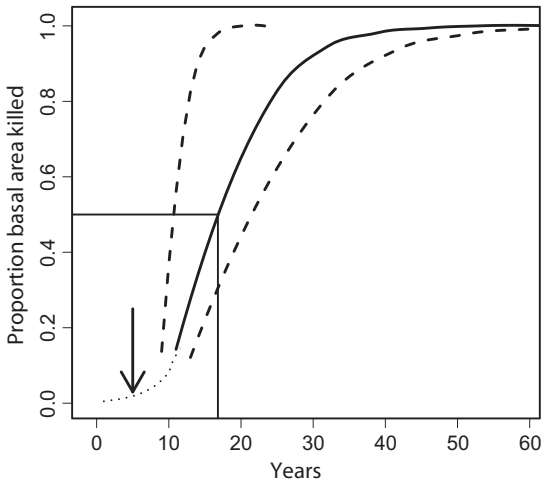

Fig. 3. Expected changes in tanoak basal area following establishment of *Phytophthora ramorum* in our invaded plot network. Data are the sum of estimated times to infection and subsequent mortality for tanoak in infected study plots. The initial year of disease emergence (1998) is indicated by the vertical arrow. Data are median expected times (solid line and points) presented with the 25th and 75th percentiles of the combined estimates (dashed lines). The dotted line represents interpolated disease dynamics assuming the pathogen arrived in 1995. The vertical line indicates 50% basal area loss.

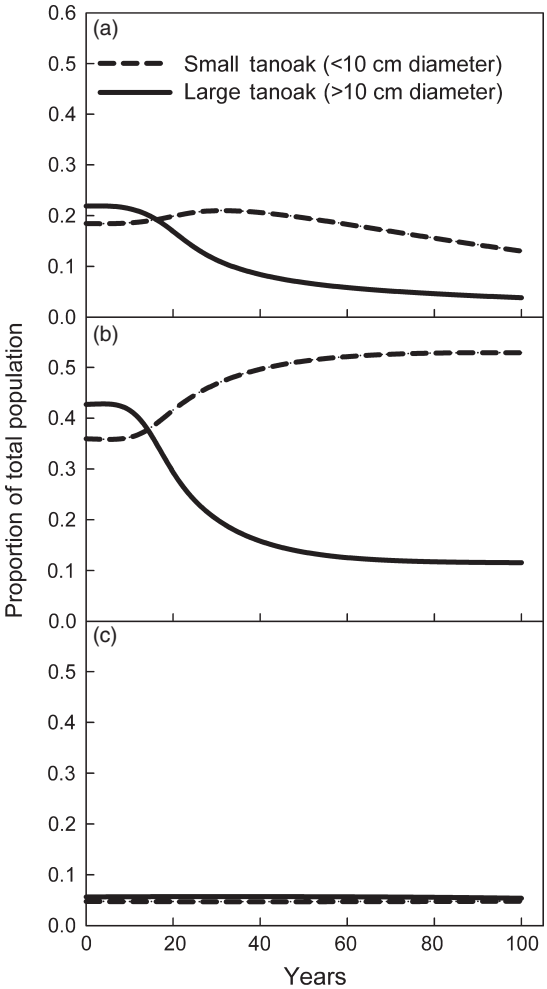

Fig. 4. Model simulations of tanoak stand structure and population changes for three initial stand conditions. (a) *Phytophthora ramorum* transmission occurs from tanoak and bay laurel whose initial proportions correspond to average numbers of stems in the plot network. (b, c) Transmission is from tanoak only, and the stands are dominated either by tanoak (b; tanoak = 80% of initial composition) or redwood (c; tanoak = 8% of initial composition). Panel (c) is the only model scenario where disease outbreak does not occur.

These results suggest that further research on these processes is warranted and could be useful for managing the disease and conserving biodiversity in at risk forests.

Our model predictions illustrate the importance of resprouting for tanoak persistence and suggest that low densities of tanoak occurring in a matrix of species that do not support *P. ramorum* sporulation will remain part of the forest overstorey. Tanoak frequently occurs in stands without bay laurel or other species, which support significant levels of *P. ramorum* sporulation (Meentemeyer *et al.* 2008; Václavík *et al.* 2010), and a well-developed body of theory suggests that host-density thresholds for pathogen establishment might occur in these stands (Park, Gubbins & Gilligan 2001; Deredec & Courchamp 2003; Holt *et al.* 2003; Gilligan & van den Bosch 2008). Scaling the model threshold of ~8% initial tanoak by the mean tanoak density of our plot network (561 ha^{-1}) suggests that treatments or stand conditions with <60 tanoak ha^{-1} (±9 SE) can retain overstorey tanoak when a second sporulation-supporting species is absent. Experimental treatments to retain overstorey tanoak are needed to identify and quantify host-density thresholds in the field. Using tanoak infection and mortality rate estimates corresponding to the 25th and 75th percentiles of the data (Fig. 1) did not change the qualitative dynamics of the model, but the threshold for disease outbreak in the low-density tanoak scenario increased with slower infection rate and faster mortality rate as both changes slow the rate of pathogen spread within a host population. Regardless of the actual value of the host-density threshold, treatments will only be effective when they shift stands to low densities of a single species transmitting the pathogen. Although we combined redwood with other species that do not support sporulation in our model, a recent study by Haas *et al.* (2011) showed that diverse forests have lower infection incidence compared with less diverse stands. The Haas *et al.* study suggests alternatives to simply increasing redwood dominance such as creating diverse mixtures of non-transmissive species, which may be more appropriate for some conservation-focused management goals. Broadly, the most infectious species (bay laurel) should be targeted for removal (Ndeffo Mabah & Gilligan 2010), and tanoak tree spacing should be increased to exceed 20 m, the distance within which local spore dispersal is most common (Davidson *et al.* 2005). Stands with low densities of tanoak that do not contain bay laurel may be protected from disease outbreak and should be targeted for tanoak conservation, especially where tanoak resistance levels are higher (*sensu* Hayden *et al.* 2011). Worldwide, understanding the epidemiological role of community members is central to predicting where *P. ramorum* will result in disease emergence and the extent of disease impacts.

The success of our recommended treatments could be thwarted by common species with relatively poor ability to transmit *P. ramorum* (Holt *et al.* 2003). Our model was sensitive to transmission among species suggesting that even relatively small sources of inoculum from rhododendron species, such as *Rhododendron macrophyllum*, could maintain pathogen populations and lead to disease emergence even when tanoak densities are low. *Rhododendron* and other low-sporulation-supporting species are more prevalent in the northern range of tanoak and are frequently targeted for removal in disease treatments (Hansen *et al.* 2008; Václavík *et al.* 2010). Additional epidemiological scenarios where one or more species are minor sources of sporulation are not considered in our model structure, but may underlie sudden oak death dynamics in Northern California, Oregon and recent outbreaks in Western Europe (Hansen *et al.* 2008; Brasier & Webber 2010). Identifying the epidemiological role of community members is central to optimizing control strategies for this disease because incomplete understanding of each species' epidemiological characteristics can undermine management actions (Ndeffo Mabah & Gilligan 2010).

Post-mortality resprouting is likely to play a role in maintaining both forest pathogens and host populations. Susceptible population size is critical to maintaining pathogen populations when infection results in host mortality (*sensu* Swinton & Gilligan 1996); hence, we expect that host-density thresholds for *P. ramorum* persistence exist in California (Fig. 4c; Park, Gubbins & Gilligan 2001; Holt *et al.* 2003; Gilligan & van den Bosch 2008). Beech bark disease and chestnut blight are classic examples of plant diseases where basal sprouting plays a role in host persistence. On a regional scale, American beech (*Fagus grandifolia*) varies in its propensity to reproduce via basal sprouting (Kitamura & Kawano 2001), which may explain why the species is in decline in some regions and not in others (Garnas *et al.* 2011). Variation in resprouting rates is probably even more important for the persistence of American chestnut because successful recruitment from seed is rare (Paillet 2002; Loo 2009).

Many of the long-term changes to redwood stand composition and feedback on *P. ramorum* populations will depend on pre-disease stand composition. For example, Ramage, O'Hara & Forrestel (2011) found extensive tanoak and redwood basal sprouting following emergence of sudden oak death and suggested seedling recruitment from species outside of the immediate stand may be limited in these forests. In parts of California with intensive conifer silviculture, very high rates of tanoak resprouting could lead to a short-term increase in herbicide use given that tanoak competes with more economically valuable trees such as redwood (Bowcutt 2011). In the absence of management, pathogen populations will continue to limit tanoak from reaching the overstorey in redwood–tanoak forests (Fig. 4b), these stands will become more redwood dominated, and pathogen populations may eventually decline (Power & Mitchell 2004; Cobb, Meentemeyer & Rizzo 2010). However, when bay laurel and tanoak co-occur, growth rates of overstorey bay laurel are likely to increase and lead to positive feedback on pathogen populations (Cobb, Meentemeyer & Rizzo 2010).

We have taken steps towards the development of a theoretical framework of forest disease impacts to overstorey host species. We found that community-structured transmission, rates of resprouting, infection and mortality determine the nature and extent of host population decline. We have demonstrated the utility of this framework in qualitatively predicting population and biomass changes for an aerially dispersed generalist plant pathogen. *Phytophthora ramorum* also illustrates the importance of vegetative reproduction in rescuing plant hosts from extinction by generalist pathogens. In contrast, generalist pathogens can cause substantial extinction risk to mammals (Frick *et al.* 2010) and other taxa (de Castro & Bolker 2005) where no process analogous to basal sprouting can maintain these hosts. Similarly, soil-borne root pathogens such as *P. cinnamomi* may pose even greater extinction threats than plant pathogens which attack above-ground tissues because the below-ground structures critical to host persistence from basal sprouts are killed by these pathogens (see de Castro & Bolker 2005; Smith, Sax & Lafferty 2006). Community-level studies of different wildland disease outbreaks will aid development of a rigorous theory describing the interactions of host, pathogen and community characteristics on the outcome of forest disease. This study suggests that pathogenicity, virulence and community-driven epidemiology along with rates of resprouting are parameters crucial to characterizing the impacts of forest disease to stand and community structure. Synthesizing management efforts to address conservation and natural resource goals in the face of emerging forest pathogens will benefit from quantifying these variables and integrating them into epidemiological or ecological analyses of disease dynamics.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. Study site locations, assessment of survival model assumptions and a detailed description of the development, formulation and parameterization of the mathematical model.

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