

LETTER

Forest species diversity reduces disease risk in a generalist plant pathogen invasion

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Abstract

Empirical evidence suggests that biodiversity loss can increase disease transmission, yet our understanding of the 'diversity-disease hypothesis' for generalist pathogens in natural ecosystems is limited. We used a landscape epidemiological approach to examine two scenarios regarding diversity effects on the emerging plant pathogen *Phytophthora ramorum* across a broad, heterogeneous ecoregion: (1) an *amplification effect* exists where disease risk is greater in areas with higher plant diversity due to the pathogen's wide host range, or (2) a *dilution effect* where risk is reduced with increasing diversity due to lower competency of alternative hosts. We found evidence for pathogen dilution, whereby disease risk was lower in sites with higher species diversity, after accounting for potentially confounding effects of host density and landscape heterogeneity. Our results suggest that although nearly all plants in the ecosystem are hosts, alternative hosts may dilute disease transmission by competent hosts, thereby buffering forest health from infectious disease.

Keywords

Bayesian hierarchical model, emerging infectious disease, forest ecosystem, landscape epidemiology, *Phytophthora ramorum*, spatial autocorrelation, species diversity, sudden oak death, zero-inflation.

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INTRODUCTION

Emerging and re-emerging infectious diseases of plants and animals continue to pose threats to ecosystem services and public health worldwide (Daszak *et al.* 2000; Kilpatrick *et al.* 2010). The economic and ecological impacts they cause, coupled with accelerating anthropogenic environmental change, have stimulated increased interest in understanding the function of biodiversity in disease ecology (Mack *et al.* 2000; Pongsiri *et al.* 2009). Recent studies across a range of host-pathogen systems suggest that the species diversity of ecological communities can moderate the prevalence of infectious disease through several mechanisms, collectively referred to as *dilution* and *amplification effects* (Keesing *et al.* 2006). To date, the majority of studies analysing diversity-disease risk relationships have presented evidence for dilution effects, whereby increased species diversity decreases pathogen transmission and disease incidence (Keesing *et al.* 2010). Amplification effects, in contrast, are predicted to occur when increased diversity leads to an intensification of disease risk in a community. However, the possibility of pathogen amplification in multi-host systems remains a subject of debate due to scant empirical evidence (Dobson 2004; Begon 2008). In multi-host systems, the role of diversity may be complex when asymmetric competency (i.e. effectiveness of passing on infection) exists among host species (LoGiudice *et al.* 2008). For instance, an increase in competent hosts within a community might be expected to amplify risk, but if considerable differences in host competency exist among community members, then the addition of less competent, alternative hosts may

reduce inoculum load and overall infection risk (Ostfeld & Keesing 2000a).

Our current understanding of the role that species diversity plays in disease ecology hinges primarily on vector-borne zoonoses, agroecosystems and experimental plant communities (Mitchell *et al.* 2002; Pautasso *et al.* 2005; LoGiudice *et al.* 2008), with surprisingly little attention given to how diversity-disease relationships vary in natural ecosystems (i.e. those with minimal human impact) and across broad geographical regions (but see Seabloom *et al.* 2009). Host-pathogen interactions in ecologically realistic settings are embedded within communities, ecosystems and landscapes, and as a result, processes on these larger spatial scales likely play a key role in disease dynamics at the local scale (Ostfeld *et al.* 2005). Over the past few decades, a growing number of plant pathogens have invaded a variety of forest communities over landscape- to regional-scales (Aukema *et al.* 2010). Since the 'diversity-disease hypothesis' was first proposed by Elton (1958) – which recognised that plant diseases could be ameliorated in 'complex' ecosystems if this complexity reduced host density – studies from a range of experimental plant systems, including agroecosystems, have found that high species diversity is often associated with lowered disease risk (Knops *et al.* 1999; Mitchell *et al.* 2002; Pautasso *et al.* 2005; but see Borer *et al.* 2010). However, it remains uncertain whether diversity-disease risk relationships exist with generalist forest pathogens in multi-host communities with high levels of natural variation in plant species diversity and across large heterogeneous landscapes.

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Experimental studies can provide a causal understanding of factors driving infection risk, yet it is increasingly recognised that observational approaches are needed to investigate ecological processes operating at large spatial scales that extend beyond those feasible for experimentation (Sagarin & Pauchard 2010). The burgeoning field of landscape epidemiology integrates concepts and approaches from disease and landscape ecology to study disease dynamics in ecosystems across time and space (Holdenrieder *et al.* 2004; Ostfeld *et al.* 2005) and may provide a novel framework for studying dilution and amplification effects in natural ecosystems across broad geographical regions. Studies that utilise geographic information systems (GIS) and other geospatial technologies are especially needed to assimilate the types of large spatially explicit datasets that can enable analysis of diversity–disease risk relationships in the context of local-, landscape- and regional-scale heterogeneity in the environment.

Here, we present results from a landscape-scale, observational study of diversity–disease risk relationships with the emerging plant pathogen, *Phytophthora ramorum* (*P. ramorum*), across forests of the Big Sur, California ecoregion (Fig. 1). *Phytophthora ramorum* is the causal agent of the infectious diseases sudden oak death and ramorum blight in North America and Europe (Rizzo *et al.* 2005; Brasier & Webber 2010). Since discovered in North America in the mid-1990s near San Francisco Bay, this environmentally transmitted pathogen has killed millions of oak (*Quercus* spp.) and tanoak (*Notholithocarpus densiflorus*) trees in coastal forests of California and Oregon (Rizzo *et al.* 2005). *Phytophthora ramorum* is a generalist, with over 100 species of plants (including ferns, gymnosperms and angiosperms) classified as hosts in native and horticultural settings (Rizzo *et al.* 2005). Over 40 of the known host species are native to California and Oregon coastal forests. The diseases the pathogen causes on this range of hosts are

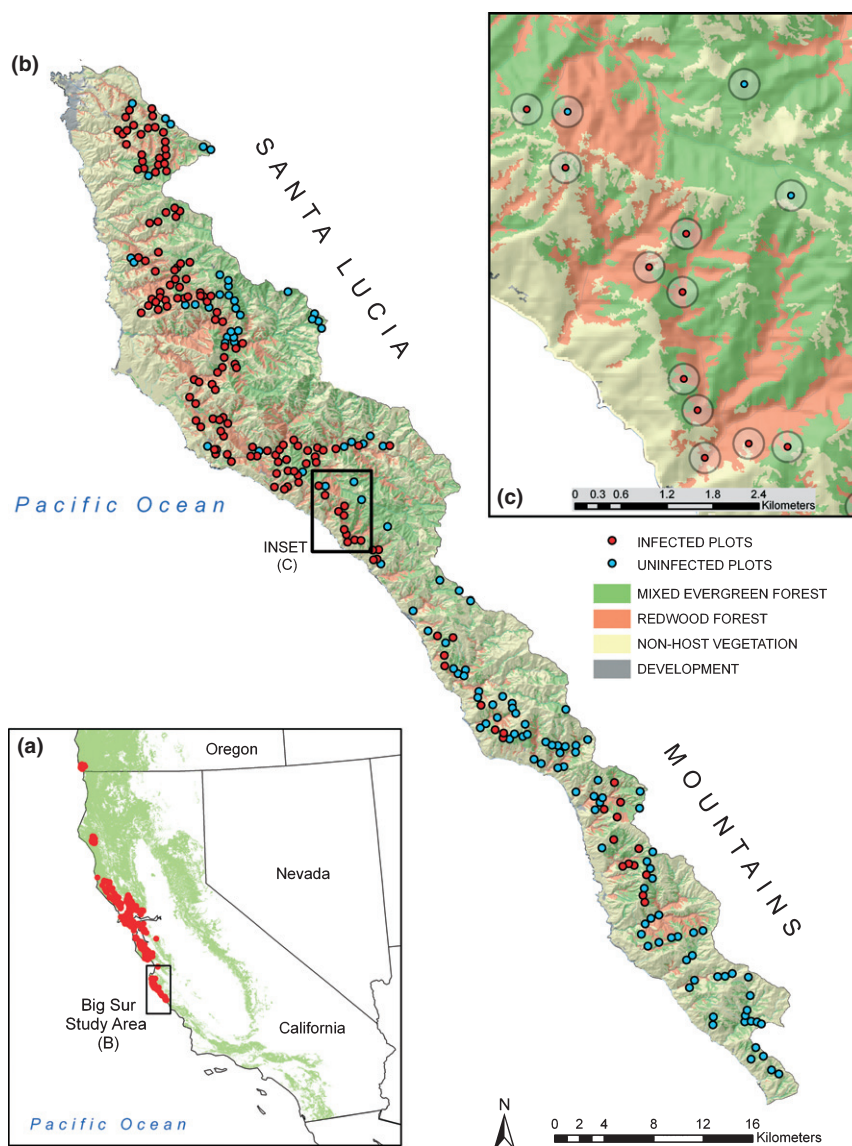


Figure 1 (a) Current geographic distribution of *P. ramorum* in North America (shown in red), extending from Monterey County in central California to Curry County, Oregon in the north. Host vegetation is shown in green. (b) The Big Sur study area (79 356 ha) extends 100 km along the Pacific slope of the Santa Lucia range. Host forest community types (mixed-evergreen and redwood forests) are mapped throughout the region. *P. ramorum* infected plots are shown in red ($n = 151$) with uninfected plots shown in blue ($n = 127$). (c) Inset map shows the high degree of spatial heterogeneity in forest community types. Circles represent the 200-m radius buffers from plot centre used to obtain the amount of host vegetation cover surrounding each plot.

expressed in two ways: (1) main stem canker infections that often cause mortality in oaks, tanoak and a few other species and (2) sublethal foliar and twig infections in tanoak and other non-oak hosts.

The *P. ramorum* pathosystem provides an interesting model for studying diversity–disease risk relationships because the pathogen infects almost all woody plant individuals throughout the region, yet hosts exhibit asymmetric transmission and susceptibility (Davidson *et al.* 2005, 2011; Cobb *et al.* 2010). Main stem canker infections (as on oaks and tanoak) have not been shown to produce inoculum in the field, whereas on some hosts foliar and twig infections can produce large amounts of inoculum that is primarily transported via wind-driven rainsplash (Davidson *et al.* 2005, 2008). The two most competent, inoculum-producing foliar hosts in California forests, bay laurel (*Umbellularia californica*) and tanoak, appear critical for natural spread of the pathogen between individual plants and across landscapes (Davidson *et al.* 2005, 2008). Tanoak is unique in our study system because it is the only known host to exhibit lethal canker infections while simultaneously producing inoculum on infected leaves and twigs (Davidson *et al.* 2008). The amount of inoculum produced by *P. ramorum* on a majority of its other host species (i.e. alternative hosts) in California and Oregon forests is unknown (Rizzo *et al.* 2005).

We examine two hypotheses regarding the effect of species diversity in this pathosystem: (1) an *amplification effect* exists in which disease risk is greater in areas with higher plant species diversity due to the broad host range of *P. ramorum*, or in contrast, (2) a *dilution effect* exists whereby risk is reduced with increasing diversity due to lower competency of alternative hosts when compared with bay laurel and tanoak. We use a landscape epidemiological approach – combining field data on pathogen abundance and host community structure with geospatial data on landscape context – to examine effects of plant species diversity on disease risk across a large, spatially heterogeneous region. When studying emerging diseases across landscape to regional scales, the large-scale process of extinction and recolonisation and the fine-scale contagious process of pathogen dispersal may lead to complexities in observational datasets, including zero-inflation (i.e. data with an excess of zeros) and spatial autocorrelation, both of which can bias parameter estimation and associated measures of uncertainty (Gschlöl & Czado 2008). We account for landscape-scale processes of invasion by comparing inference among three Bayesian hierarchical models with varying complexity: (1) a binomial generalised linear model (GLM), (2) a zero-inflated binomial GLM and (3) a zero-inflated binomial generalised linear mixed model (GLMM) with a spatial random effect. Our results provide insights into dilution and amplification effects of biodiversity on a generalist plant pathogen invasion across a broad, environmentally heterogeneous region with high natural variation in plant diversity and a wide range of hosts.

MATERIALS AND METHODS

Study area

We established a long-term *P. ramorum* monitoring network consisting of 280 plots (500 m²) throughout a 79 356 ha study area within the Big Sur ecoregion, extending 100 km along coastal, central California (Fig. 1). Plots were randomly located across a range of ecological conditions stratified by elevation, latitude, fire history and forest community type (mixed-evergreen and redwood forests), and were

located in areas with and without the pathogen. Each plot was established at least 300 m from its nearest neighbour. It is unknown exactly when *P. ramorum* initially invaded the region, though impacts were first noted in the mid-1990s (Rizzo & Garbelotto 2003). The pathogen is patchily distributed at broad and fine spatial scales across the region, with stands of uninfected hosts spatially juxtaposed with severely impacted stands, despite few apparent environmental differences between sites (Meentemeyer *et al.* 2008). The region's topography is characterised by deeply dissected slopes and drainages, with elevation ranging from sea level to 1571 m within 5 km of the coast (Hensen & Usner 1996). A diversity of plant communities occurs throughout the area, though plots were only established in two dominant forest types containing host vegetation: mixed-evergreen forests occurring along moister slopes and redwood forests at lower elevations (Plate 1).

Field data

We collected data over a 2 year period from June to October of 2006 and 2007. Plot coordinates were recorded using survey-grade global positioning system receivers. In each plot, we identified the

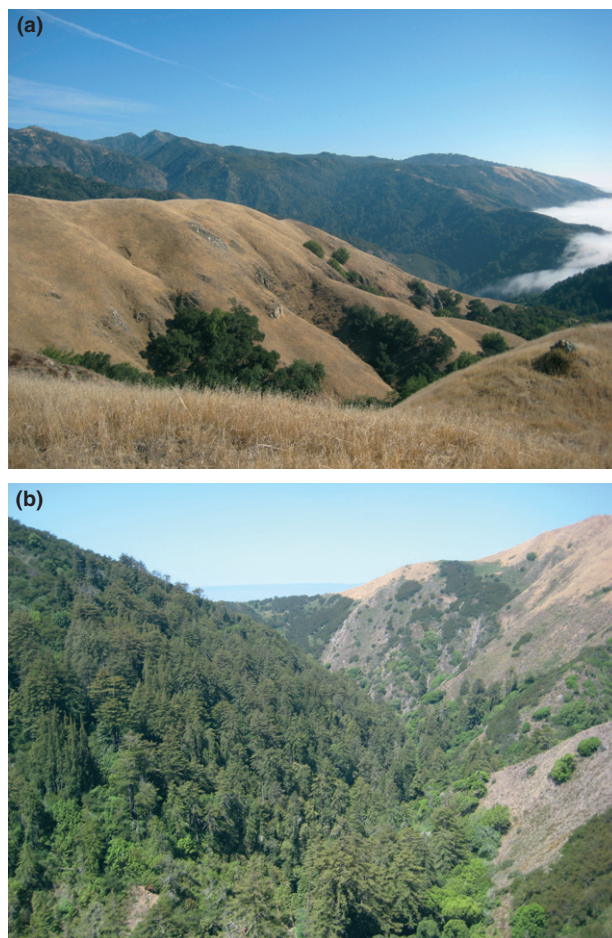


Plate 1 Examples of landscape heterogeneity in the Big Sur, California ecoregion showing (a) mixed-evergreen forest and (b) redwood forest. These forest community types are optimal habitat for *P. ramorum* throughout the study area. There were 162 plots located in mixed-evergreen forest and 116 plots in redwood forest. Photo credit: S. E. Haas.

species of all woody plants (hosts and non-hosts) that satisfied the following size requirements: trees and vines with a diameter at breast height (dbh) ≥ 1 cm and shrubs that reached an area ≥ 1 m² (see Table S1 in Supporting Information for a list of surveyed species). Symptoms of *P. ramorum* infection were recorded for hosts satisfying size requirements and symptomatic tissue from a subset of individuals was brought to the laboratory for pathogen isolation using a *Phytophthora*-selective media (PARP; Davidson *et al.* 2005). For analysis purposes, we considered symptomatic plants as 'infected' only if cultured samples confirmed the presence of *P. ramorum* in the respective plot. The following variables were derived for each plot: species richness (number of host and non-host species), Shannon–Wiener diversity index H' (accounts for species richness and evenness), competent host density (calculated for bay laurel and tanoak separately) and forest community type (mixed-evergreen or redwood forest).

Geospatial data

We accounted for effects of landscape heterogeneity on disease risk by including three GIS-derived variables for each plot: average precipitation over the wet season, potential solar insolation (PSI) and area of host vegetation cover surrounding each plot. We used the parameter elevation regression on independent slopes model (PRISM; Daly *et al.* 1994) to map the 30-year (1971–2000) monthly average precipitation (800-m spatial grain) between December and May, which corresponds to the rainy season when conditions are favourable to *P. ramorum* infection (Davidson *et al.* 2005). To account for topographic variation in solar energy, we derived average PSI over the wet season from a U.S. Geological Survey 30-m digital elevation model. Potential solar insolation uses the cosine of illumination on slope equation to measure the potential solar energy incident at a location on Earth's surface (Dubayah 1994). The amount of host vegetation cover around each plot was obtained by summing the area of mixed-evergreen and redwood forest within a 200-m radius (12.5 ha) from plot centre, using vegetation maps described in Meentemeyer *et al.* (2008; Fig. 1c). This distance was previously found as the scale that *P. ramorum* responds most strongly to forest heterogeneity (Condeso & Meentemeyer 2007).

Data analysis

Statistical analyses were conducted using R version 2.12.1 (R Development Core Team 2010). We used a new method for fitting Bayesian Markov random field models called INLA (integrated nested Laplace approximations) (Rue *et al.* 2009). The class of models capable of being fit using this approach need to have certain characteristics. As INLA is valid for generalised Gaussian Markov random field models, it can be used in situations where a latent Gaussian model (i.e. a linear regression type model) represents the underlying process of interest and is observed via one of several possible sampling or measurement distributions (e.g. Gaussian, binomial, Poisson). In these specifications, the hierarchical model can be reparameterised such that both the latent Gaussian process and regression coefficients form a Gaussian Markov random field and the remaining model parameters (termed 'hyperparameters') are few. As these models have a large number of approximately Gaussian unknowns and only a few remaining parameters, the INLA procedure can be used to find approximate marginal posterior distributions with considerably less

computational effort than stochastic methods such as Markov Chain Monte Carlo. We used the INLA package (Rue & Martino 2009) to fit models with varying complexity: (1) a binomial GLM, (2) a zero-inflated binomial GLM and (3) a zero-inflated binomial GLMM with a spatial random effect. The proportional response variable was disease incidence, calculated by grouping the number of infected hosts with the number of uninfected hosts per plot.

We describe the most sophisticated model only (i.e. the GLMM), as the remainder of models are simplifications of it (see Appendix S1 for information on each model). For the total number of plants (n_i) at each plot i , for m total plots, we record each individual plant as a binary variable (i.e. infected/uninfected) and let the sum of these be binomial distributed with probability θ_i if the pathogen has reached plot i and zero if the pathogen has not yet reached the plot. As pathogen absence is difficult to observe perfectly, a zero-inflated binomial model for the data could help account for this source of uncertainty (as well as the transient nature of this ongoing epidemic).

In constructing the process portion of the models, we used the traditional 'logit' function to link the probabilities θ_i to a set of covariates:

$$\text{logit}(\theta_i) = \beta_0 + \beta_1 x_{1,i} + \dots + \beta_q x_{q,i} + \varepsilon_i,$$

where the x variables represent plot-level characteristics for all q regression coefficients. The errors, ε_i , were allowed to be spatially correlated so they could absorb any latent autocorrelation beyond that described by the covariates. We specified an intrinsic conditional autoregressive model (ICAR) for the errors, where to create the necessary spatial proximity matrix, we obtained 'pseudo-residuals' by fitting the zero-inflated GLM with i.i.d. errors and then computed Moran's I spatial statistic to assess potential autocorrelation in these pseudo-residuals. The spatial proximity matrix was based on the interplot neighbourhood distance (in km) that had the lowest P -value for Moran's I and we assumed that this distance was the scale in which dominant latent spatial structure should be accounted for by our model (Figure S1 and S2). To specify the parameter portion of the model, the regression coefficients were given a Gaussian prior, $\beta \sim N(0, 1000 \cdot I)$, the precision parameter for the latent errors was given a gamma prior, $\tau \sim \text{Gamma}(1, 5e-05)$ and the logit of the zero-inflation probability for the likelihood was given a standard normal prior distribution, $N(0,1)$.

We fit separate models for species richness and the Shannon–Wiener diversity index H' due to collinearity between these diversity measurements. Because we collected field data over 2 years, we added sampling year as an indicator covariate with the year 2006 being zero. All variables were tested for multicollinearity and continuous variables were scaled prior to model implementation. Marginal posterior distributions were summarised by 95% Bayesian credible intervals (i.e. BCI; the 0.025 and 0.975 quantiles of the posterior distribution) and deviance information criterion (DIC) statistics were used to compare models (Spiegelhalter *et al.* 2002).

RESULTS

A total of 10 472 known host plants (from 11 501 plants representing 21 host and 25 non-host species; Table S1) were assessed for *P. ramorum* symptoms across all 278 plots. Of these, 23% of hosts from 151 plots were classified as infected following laboratory confirmation of *P. ramorum* in the respective plot. The mean

proportion of infected host plants within diseased plots was 0.37 and exhibited positive skew (range = 0.01–0.95, skew = 0.52). There were no infected hosts in 127 plots (46% of plots), with the majority of uninfected plots clustering towards the southern portion of the study area (Fig. 1). Approximately 80% of all plots contained one or more bay laurel trees, 61% had at least one tanoak and 49% contained both competent hosts. Tanoak was the most abundant host and had higher disease incidence than all other host species, followed by bay laurel (Fig. 2; Table S1). The abundance of these two competent hosts varied by forest type with mixed-evergreen forests containing 20% bay laurel (1172/5833 host plants) and 15% tanoak (889 plants), whereas redwood forest contained 11% bay laurel (488/4639 host plants) and 49% tanoak (2274 plants). Bay laurel density exhibited weak correlations with species richness (SR) and the Shannon–Wiener diversity index (H') ($r_{\text{SR, BAY}} = 0.05$; $r_{H', \text{BAY}} = -0.02$), as did tanoak density ($r_{\text{SR, TOAK}} = -0.17$; $r_{H', \text{TOAK}} = -0.32$). Species richness ranged from 1 to 12 species per plot (mean = 5), while species richness of hosts only ranged from 1 to 9 (mean = 4). The Shannon–Wiener diversity index (H') ranged from 0.00 to 2.10 (mean = 1.01). The plot network spanned a wide range of landscape heterogeneity: average precipitation (71–192 mm), PSI (0.2–1.00 watts m^{-2}) and amount of host vegetation cover within 200 m (0.27–12.56 ha). See Table S2 for descriptive statistics of all covariates.

All three Bayesian hierarchical models indicated a negative relationship between disease incidence and species diversity (measured as both SR and H'), after accounting for competent host density and landscape heterogeneity. The zero-inflated binomial GLMMs with the

ICAR spatial effect had better model fit based on DIC criterion ($\text{DIC}_{\text{SR}} = 1050$, $\text{DIC}_{H'} = 1061$) compared with the simpler zero-inflated binomial GLMs ($\text{DIC}_{\text{SR}} = 2194$, $\text{DIC}_{H'} = 2312$) and the standard binomial GLMs ($\text{DIC}_{\text{SR}} = 3598$, $\text{DIC}_{H'} = 3727$). As such, we present further results for the spatial models only (see Table S3 for all results). Based on the graph of Moran's I P -values as a function of interplot distance, we chose a neighbourhood distance of 5150 m to calculate our spatial proximity matrix for the species richness (SR) model and 6201 m for the Shannon–Wiener diversity (H') model (Figure S2). Table 1 compares parameter estimates for models when competent host density was included and excluded. The negative diversity effects remained and model fit substantially improved after incorporating density effects ($\Delta\text{DIC}_{\text{SR}} = 2055$; $\Delta\text{DIC}_{H'} = 1947$). Covariate relationships were considered statistically significant if the 95% Bayesian credible intervals did not overlap zero. For models that included competent host density, the probability of *P. ramorum* infection was negatively associated with species richness ($\hat{\beta}_{\text{SR}} = -0.83$, BCI = -1.14 to -0.55) and the Shannon–Wiener diversity index ($\hat{\beta}_{H'} = -0.48$, BCI = -0.79 to -0.19) (the beta hat corresponds to the posterior mean), and positively associated with bay laurel and tanoak density: species richness model ($\hat{\beta}_{\text{BAY}} = 1.22$, BCI = 0.90–1.57; $\hat{\beta}_{\text{TOAK}} = 0.96$, BCI = 0.61–1.32) and the Shannon–Wiener diversity model ($\hat{\beta}_{\text{BAY}} = 1.16$, BCI = 0.82–1.53; $\hat{\beta}_{\text{TOAK}} = 0.91$, BCI = 0.53–1.30). In both diversity models, we found negative effects for average precipitation, forest type (parameter estimated for redwood forest) and sampling year (parameter estimated for 2007). We found a positive effect of host vegetation cover within the 200-m buffer around each plot. Neither model showed a significant effect for PSI.

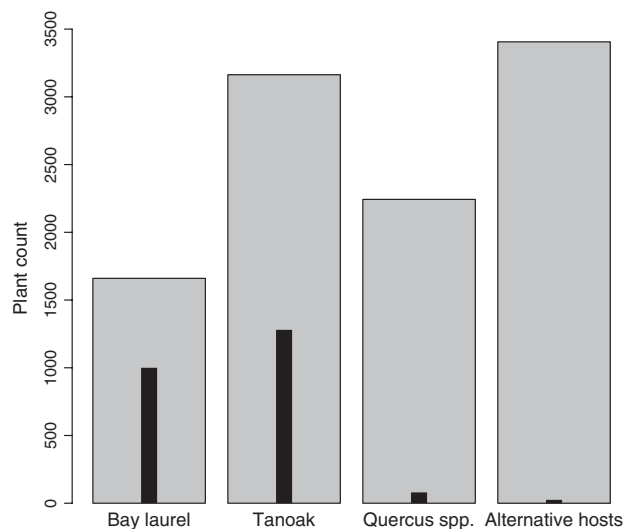


Figure 2 Total counts of host plants shown in grey bars and infected plants shown in black bars for four categories of host species ($n = 21$ host species in total) sampled across the 278 field plots in the Big Sur, California study area. The most competent foliar hosts – bay laurel (*Umbellularia californica*) and tanoak (*Notholithocarpus densiflorus*) – were the most abundant and also exhibited higher disease incidence than all other host species ($n_{\text{Bay Laurel}} = 1000$ infected plants/1660 total plants; $n_{\text{Tanoak}} = 1280$ infected plants/3163 total plants). ‘*Quercus* spp.’ includes the following four oak canker hosts, which have not been shown to transmit inoculum in the field: coast live oak (*Quercus agrifolia*), California black oak (*Q. kelloggii*), Canyon live oak (*Q. chrysolepis*) and Shreve’s oak (*Q. parvula* var. *shrevei*) ($n_{\text{Quercus spp.}} = 80$ infected plants/2243 total plants). ‘Alternative hosts’ includes 15 less competent host species ($n_{\text{Other spp.}} = 25$ infected plants/3406 total plants) (see Table S1 for a list of all host counts).

DISCUSSION

The alarming rate of species extinction worldwide gives urgency to understanding the relationship between the biodiversity of ecological communities and infectious disease risk. Despite a growing number of studies analysing the role of species diversity in disease ecology (Keesing *et al.* 2010), our understanding of diversity–disease relationships within the context of natural forest ecosystems and emerging plant pathogens is limited (Pautasso *et al.* 2005). The *P. ramorum* pathosystem is intriguing because it could potentially exhibit either an amplification effect due to the ability of the pathogen to infect multiple plant species, or in contrast, a dilution effect in which greater species diversity could lower disease risk by increasing the abundance of less competent hosts. Based on a large-scale survey across naturally growing plant communities, we found consistent negative relationships between disease incidence and plant species diversity, suggesting that a dilution effect is operating in the Big Sur *P. ramorum* pathosystem. Concerns have been raised regarding the difficulty of separating a dilution effect from an underlying host density effect (Begon 2008; Keesing *et al.* 2010; but see Johnson *et al.* 2009), yet the negative relationship we found between diversity and disease risk held after accounting for the potentially confounding effects of bay laurel and tanoak density and local- to landscape-scale environmental heterogeneity. In addition, our sparsely populated study system in a relatively remote wilderness area was minimally affected by human-impacted covariates (e.g. housing density, transportation and farming), which can further complicate interpretation of diversity–disease analyses in zoonotic systems (e.g. Swaddle & Calos 2008).

Table 1 Parameter estimates (marginal posterior means) from the zero-inflated binomial GLMM showing effects of species diversity and landscape context on *P. ramorum* disease risk, after excluding and including competent host density (bay laurel and tanoak). *Species richness* and *Shannon–Wiener diversity index* (H') models were fit separately due to collinearity between these diversity measurements. Covariates were obtained for all 278 field plots across the Big Sur study area. 95% BCI (i.e. the 0.025 and 0.975 quantiles of the posterior distribution) are shown in parentheses*.

	Species diversity	Bay laurel density	Tanoak density	Year†	Forest type‡	Precipitation	Host vegetation cover	DIC
<i>Species richness</i>								
Exclude	–0.49 (–0.56 to –0.42)	–	–	–0.32 (–0.45 to –0.19)	–0.40 (–0.53 to –0.28)	–0.30 (–0.37 to –0.23)	0.25 (0.18 to 0.32)	3105
Include	–0.83 (–1.14 to –0.55)	1.22 (0.90 to 1.57)	0.96 (0.61 to 1.32)	–0.75 (–1.33 to –0.21)	–0.83 (–1.37 to –0.28)	–0.73 (–1.19 to –0.29)	0.41 (0.12 to 0.71)	1050
<i>Shannon–Wiener diversity index</i> (H')								
Exclude	–0.57 (–0.65 to –0.51)	–	–	–0.39 (–0.52 to –0.26)	–0.45 (–0.58 to –0.32)	–0.43 (–0.51 to –0.36)	0.26 (0.19 to 0.33)	3008
Include	–0.48 (–0.79 to –0.19)	1.16 (0.82 to 1.53)	0.91 (0.53 to 1.30)	–0.73 (–1.33 to –0.15)	–0.72 (–1.31 to –0.14)	–0.74 (–1.18 to –0.32)	0.39 (0.07 to 0.73)	1061

GLMM, generalised linear mixed model; BCI, Bayesian credible intervals; DIC, deviance information criterion; PSI, potential solar insolation.

*Only parameter estimates whose 95% BCI did not overlap zero are shown; PSI is the only covariate omitted based on this criterion.

†Parameter estimated for 2007.

‡Parameter estimated for redwood forest.

Our findings suggest that despite the presence of many alternative hosts in the *P. ramorum* pathosystem, there is little evidence that these hosts play a strong role in facilitating disease transmission in the forest and may actually protect oaks by diluting impacts of the highly competent hosts, bay laurel and tanoak. These results support the importance of quantifying reservoir competency of all community members, as only knowing whether a species is a host of a particular disease or not is not enough to predict its net effect on disease transmission (LoGiudice *et al.* 2008). For instance, if a focal species is a relatively inefficient reservoir, adding other hosts to the community might amplify disease risk if these added species exhibit greater transmission potential. In contrast, if the focal host is highly competent to begin with, then the addition of other (less competent) species may reduce inoculum load and overall infection risk in the community. This phenomenon could arise in pathogens that are environmentally transmitted if less competent species remove inoculum from the system (Keesing *et al.* 2006), which may occur in the *P. ramorum* pathosystem through the ‘dead-end’ *Quercus* hosts.

Although the effects of diversity on disease transmission have been described for multiple disease systems, with particular attention to vector-borne zoonoses, our understanding of the mechanisms governing these relationships and their applicability to a broader range of disease systems remains speculative (Keesing *et al.* 2010). For instance, the net effects of diversity on disease risk may differ between plant and animal systems due to differences in host movement and spatial structure, host immune response, the number of individuals monitored and levels of species richness (e.g. we assessed 11 000 + plants; 21 host and 25 non-host species). In plants, reduced host density is often the most important mechanism by which diversity reduces disease severity, particularly for host-specific fungal diseases in agroecosystems (Burdon & Chilvers 1982). In experimental grassland systems, Knops *et al.* (1999) and Mitchell *et al.* (2002) found that sites with high species richness had lower disease severity of specialist fungal diseases, but statistical analyses revealed host density as the main driver rather than species diversity *per se*. More complicated mechanisms may exist in pathosystems involving multiple host species. Borer *et al.* (2010) analysed infection prevalence of four generalist viral pathogens in grass species within an experimental

system, but did not find a relationship between host richness and infection levels. In the *P. ramorum* pathosystem, the roles of hosts are complex and it is unclear how transmission reduction by diluting species varies among communities differing in plant species composition. For example, bay laurel and tanoak can produce copious amounts of inoculum, but only tanoak experiences mortality from sudden oak death. *Quercus* canker hosts undergo disease-induced mortality, yet have not been shown to produce inoculum in the field and therefore likely function as pathogen sinks. Less is known about the transmission potential of other alternative hosts (e.g. redwood, madrone). There are also a number of key differences among hosts in how they are infected and how they respond to infection: evergreen vs. deciduous foliage, understory vs. overstory plants, impact of disease on host tissue, and the production of inoculum from infected tissue (Rizzo *et al.* 2005).

Density-dependent models of transmission are often used to describe pathogens spread through environmentally transmitted propagules or through random contact among individuals (Keesing *et al.* 2006). One density-dependent mechanism that may be occurring in our study system is ‘encounter reduction’ (Keesing *et al.* 2006), whereby the addition of less competent hosts may interfere with transmission pathways, making *P. ramorum* less abundant than in the presence of bay laurel or tanoak alone. For instance, the *Quercus* hosts may act as physical barriers to pathogen dispersal by intercepting inoculum transmitted through space, or by increasing the distance inoculum must traverse between competent hosts. Another possible mechanism is ‘susceptible host regulation’, in which interspecific competition for limiting resources may constrain the abundance of competent hosts (Keesing *et al.* 2006). 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 & Keesing 2000a). We found that bay laurel and tanoak were abundantly distributed across the study area and that both species were more frequently infected than other hosts. This pattern has been observed in other disease systems and may result from evolutionary bias by pathogens towards widespread, abundant hosts (Ostfeld & Keesing 2000b). Nonetheless, *P. ramorum* alternative hosts with relatively small lesions may still play an important role in the

epidemiological process because although such lesions do not kill leaves, they may support enough inoculum production in forests to function as reservoirs (Rizzo & Garbelotto 2003).

Part of the challenge in understanding diversity–disease relationships in natural ecosystems is knowing when the landscape context modifies or overrides the impact of local conditions (Ostfeld *et al.* 2005). We analysed diversity effects in a broader ecological context, accounting for environmental heterogeneity across the landscape in addition to species diversity and host density. We found that mixed-evergreen forests had higher infection probability compared with redwood forests. In a local-scale longitudinal study, Davidson *et al.* (2011) also found lower infection in redwood forests in 2001–02, but then no difference in 2002–03, followed by higher infection risk in redwood forests from 2003 to 2005. These findings suggest that *P. ramorum* transmission represents a complex ecological process dependent on a number of factors including, but not limited to, forest community type, invasion history and scale-dependent variation in habitat conditions (e.g. species composition, microclimate). In our study, plots in mixed-evergreen forest had proportionally twice as much bay laurel than redwood plots and it is likely that hosts in areas with a high abundance of bay laurel experience elevated transmission rates and infection risk. However, we also found that plots in redwood forests were often dominated by tanoak, which like bay laurel, can produce large amounts of inoculum and increase disease risk in forests. Additional studies are needed that more closely examine how differences in species composition between forest types influence disease dynamics, including effects of species turnover following mortality of tanoak and oaks.

Over the past few decades, forest pathogen outbreaks have occurred over landscape to regional scales (Jules *et al.* 2002; Meentemeyer *et al.* 2011). Despite the strong influence that landscape features can have on disease spread, acting as either barriers or conduits to pathogen dispersal, landscape epidemiology has received little attention from plant pathologists (Holdenrieder *et al.* 2004). When studying emerging diseases across geographically expansive areas, the distribution of the pathogen is expected to exhibit spatial and temporal dependency due to the invasion process of localised transmission and limited dispersal from the point of introduction (Madden *et al.* 2007). Models that accounted for the non-equilibrium structure of our dataset (i.e. zero-inflation and spatial autocorrelation) had better fit compared with non-spatial models and we therefore place more confidence in the observed negative effect of diversity on disease incidence knowing that we accounted for these complexities. Studying ecological processes at larger spatial scales will likely necessitate observation-driven approaches because such studies are conducted on spatial and temporal scales untenable to manipulative experiments (Sagarin & Pauchard 2010). However, it is often difficult to draw firm conclusions from ‘natural experiments’ (e.g. biological invasions) because of the complexity of natural environments and the impossibility of holding all habitat conditions constant with only diversity varying. Due to the correlative nature of our study, we cannot assign causal mechanisms to the patterns we found or disentangle the dilution mechanisms of encounter reduction from susceptible host regulation. Despite these challenges, our analyses accounted for multi-scale biotic and abiotic heterogeneity across the landscape and are an important first step towards illustrating how a dilution effect may manifest in a generalist plant pathogen emerging across a natural ecosystem.

In contrast to indigenous pathogens, exotic pathogens have not coevolved with the host or ecosystem they are invading and are

more likely than endemic pathogens to cause biodiversity loss through the extinction of hosts (Burdon *et al.* 2006; Desprez-Loustau *et al.* 2007). Some exotic forest diseases, such as chestnut blight and white pine blister rust, have severely impacted populations of a single plant species, leading to a cascade of changes in forest ecosystems (Rizzo 2005). When a pathogen is a generalist capable of infecting multiple species, including asymptomatic reservoirs, detection and management of disease impacts may be especially challenging. For example, the introduction of the generalist root pathogen *Phytophthora cinnamomi* had severe consequences for the Jarrah forests of Western Australia, in which most native eucalypts had little if any resistance (Shearer & Dillon 1995). Despite high plant diversity Jarrah forests were very susceptible to this generalist pathogen, illustrating a case where the insurance hypothesis fails for a non-specific pathogen (Weste *et al.* 2002). Similarly, *P. ramorum* has already begun altering forest stand structure along coastal California through mortality of oaks and tanoak (Rizzo & Garbelotto 2003). The low cost of infection in some hosts, such as bay laurel and redwood, may allow the pathogen to persist indefinitely in infested forests and initiate a shift to greater dominance of these species in the future through parasite-mediated competition (Cobb *et al.* 2010). Despite the broad host range of *P. ramorum*, our results suggest that retaining plant species diversity through management actions may mitigate risk of infection in coastal forests of California. More broadly, this research illustrates ecological conditions where high levels of host species diversity within a natural ecosystem can dilute disease risk in a generalist plant pathogen invasion across a large heterogeneous region.

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AUTHORSHIP

All persons entitled to authorship have been included. All authors have read the submitted version of the manuscript, approve of its submission and are aware they each hold responsibility for the accuracy, integrity and ethics of the manuscript and work described therein. The lead author (SH) collected data and implemented models. MH assisted with modelling methods and R-code, and DR and RK jointly designed the study. SH wrote the first draft of the manuscript and all authors contributed substantially to revisions.

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SUPPORTING INFORMATION

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

Appendix S1 Detailed description of the three Bayesian hierarchical models (i.e. binomial GLM, zero-inflated binomial GLM and zero-inflated binomial GLMM) implemented in this study.

Figure S1 'Pseudo-residuals' obtained from the *Species Richness* zero-inflated GLM.

Figure S2 Moran's I P -values for assessing global spatial autocorrelation in our pseudo-residuals from the *Species Richness* zero-inflated GLM plotted against the inter-plot neighbourhood distance (in km).

Table S1 A list of the 21 host and 25 non-host species surveyed across 278 plots in Big Sur, California that were included in our diversity–disease risk analyses of the *P. ramorum* pathosystem.

Table S2 Descriptive statistics of the eight covariates used in our diversity–disease risk analysis for the *P. ramorum* pathosystem throughout Big Sur, California.

Table S3 Parameter estimates (i.e. marginal posterior means) and deviance information criterion (DIC) for the three Bayesian hierarchical models analysing diversity–disease risk relationships in the *P. ramorum* pathosystem throughout Big Sur, California.

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