Received: 18 February 2021 | Revised: 18 March 2021 | Accepted: 10 August 2021

DOI: 10.1111/ele.13871

ECOLOGY LETTERS cnrs WILEY

LETTER

Community-level prevalence of a forest pathogen, not individual-level disease risk, declines with tree diversity

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Funding information
USDA Forest Service Pacific Southwest Research Station & Forest Health Protection; Gordon and Betty Moore Foundation; NSF-NIH Ecology and Evolution of Infectious Diseases, Grant/Award Number: DEB-1115664; NSF Graduate Research Fellowship; NSF Ecosystem Science, Grant/Award Number: ES-1753965

Editor: Kevin Lafferty

Abstract

Understanding why diversity sometimes limits disease is essential for managing outbreaks; however, mechanisms underlying this 'dilution effect' remain poorly understood. Negative diversity-disease relationships have previously been detected in plant communities impacted by an emerging forest disease, sudden oak death. We used this focal system to empirically evaluate whether these relationships were driven by dilution mechanisms that reduce transmission risk for individuals or from the fact that disease was averaged across the host community. We integrated laboratory competence measurements with plant community and symptom data from a large forest monitoring network. Richness increased disease risk for bay laurel trees, dismissing possible dilution mechanisms. Nonetheless, richness was negatively associated with community-level disease prevalence because the disease was aggregated among hosts that vary in disease susceptibility. Aggregating observations (which is surprisingly common in other dilution effect studies) can lead to misinterpretations of dilution mechanisms and bias towards a negative diversity-disease relationship.

KEYWORDS
community assembly, competence, dilution effect, diversity-disease, infectious disease, *Phytophthora ramorum*, Simpson's paradox, species composition, sudden oak death

INTRODUCTION

Human-caused biodiversity loss (Cardinale et al., 2012) alters interactions among hosts and pathogens with cascading effects on infectious diseases of humans, plants and wildlife. Susceptible hosts are often hypothesised to be more vulnerable to infections in depauperate communities than in nearby richer communities, a phenomenon coined the 'dilution effect' (Civitello et al., 2015; Magnusson et al., 2020; Ostfeld & Keesing, 2012). However, the relationship between infection risk and diversity may also be positive (Guilherme Becker & Zamudio, 2011), idiosyncratic (Salkeld et al., 2013), or context-dependent (Halliday & Rohr, 2019; Liu et al., 2020). If diversity predictably covaries with factors that limit disease, conservation of biodiversity could be a viable win-win strategy; if not, targeted management of specific species would be needed (Rohr et al., 2020). Thus, it is essential to understand why diversity affects disease dynamics to forecast and manage disease outbreaks under global change (Johnson et al., 2015; Rohr et al., 2020).

Higher diversity communities may be associated with less disease risk for individuals if they contain species that contribute little to inoculum pressure and reduce transmission risk (Keesing et al., 2006). 'Diluter' species might regulate the densities of high-competence hosts, or those that efficiently acquire and transmit pathogens, via competition for finite resources (Figure 1a; Strauss et al., 2015). Decreases in diversity have been associated with increases in infections for plant, animal, and zoonotic

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diseases (Johnson et al., 2012; Mitchell et al., 2002; Ostfeld & Keesing, 2000). Covariance between competent host densities and diversity likely depends on additional relationships among host competence, nestedness and total density. However, few studies have investigated these linkages thus far (e.g. Johnson et al., 2013; Lacroix et al., 2014).

The dilution effect may also be driven by richness per se (Figure 1b). For example, communities of greater diversity might be associated with less disease if diluter species reduce encounters between infectious and susceptible hosts (e.g. by ingesting propogules; Schmeller et al., 2014) or if they lower the likelihood of transmission given an encounter (e.g. by altering microclimates; Zhu et al., 2000). Since multiple dilution mechanisms can operate simultaneously, diversity-associated mechanisms driven by encounter/transmission reduction can be deduced after accounting for competent host densities (Strauss et al., 2016, 2018).

Furthermore, diversity–disease relationships may change whether disease is measured for particular host individuals or species, or the overall host community (Figure 1c). For instance, the individual risk of hantavirus infection in the most susceptible rodent species did not vary across habitats, but seroprevalence of the entire rodent community was greater in rural settings compared to forests (Piudo et al., 2011). Differences arise because disease in a focal host controls for species-specific susceptibility, whereas community-level prevalence aggregates across species and is sensitive to the average susceptibility of individuals from all species. Unlike individual-level disease risk, community-level prevalence does not measure risk of acquiring infections (it measures disease burden on the entire community) and is predisposed to decline with diversity due to the mathematical inevitability of adding low-susceptibility or non-susceptible species to the denominator of prevalence. While the majority

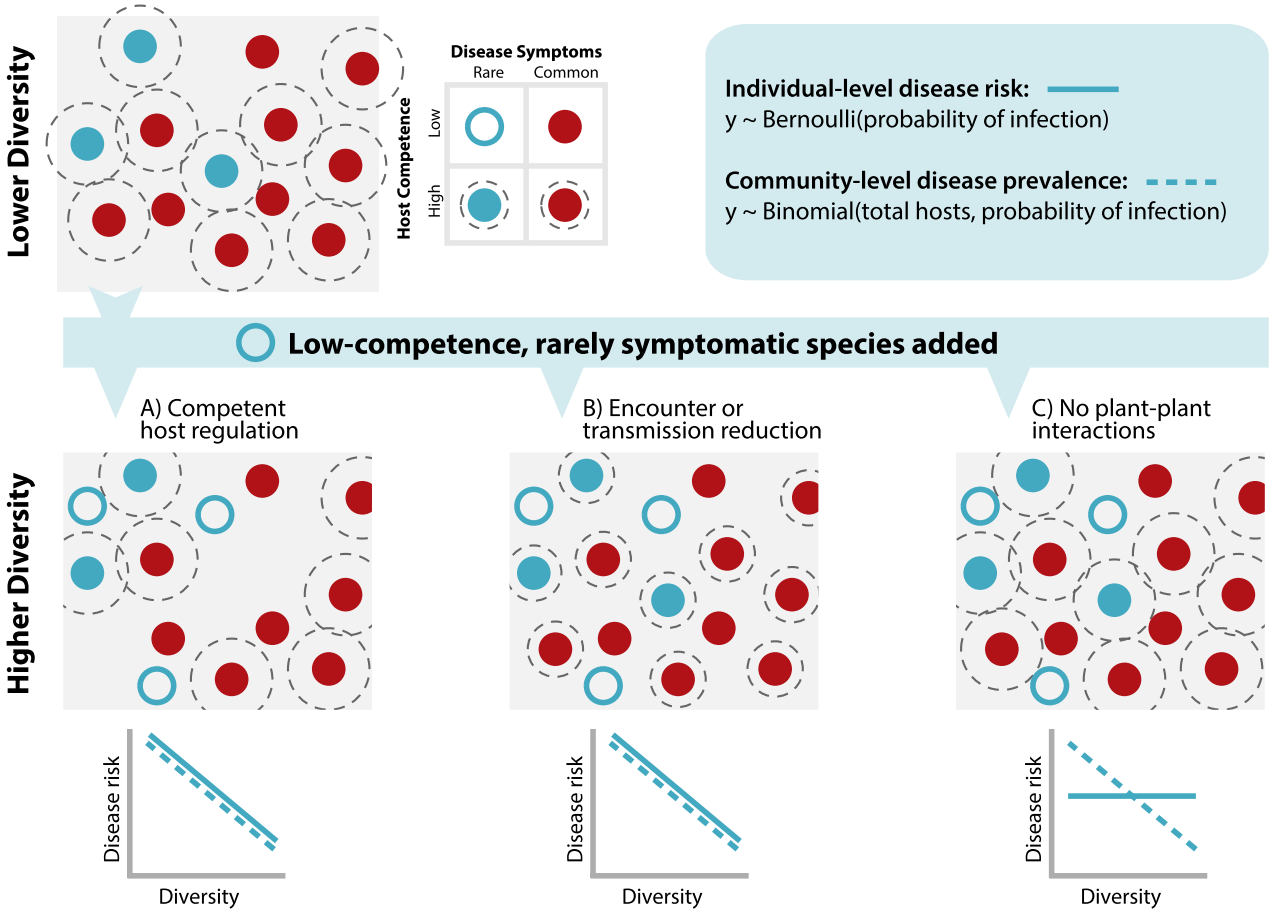

FIGURE 1 Negative diversity–disease relationships assessed at the community level may be affected by multiple dilution mechanisms and/or measurements of disease risk. The addition of low-competence, rarely symptomatic species (i.e. 'diluter' species) to higher diversity communities may potentially limit transmission risk, as measured by average individual-level disease risk, (a) by reducing the density of competent hosts ('competent host regulation', modified *sensu* Keesing et al., 2006), or (b) by reducing encounter rates or probability of transmission between infectious and susceptible individuals ('encounter reduction' or 'transmission reduction' *sensu* Keesing et al., 2006). The addition of these species may also (c) have no effect on plant-plant interactions, resulting in no corresponding change in individual-level disease risk. Across all three scenarios, the overall proportion of commonly symptomatic species is lower in the higher diversity community, causing a negative relationship between diversity and community-level disease prevalence. The area of the dashed halos represent total potential inoculum pressure exerted by competent hosts

of studies discussed within the dilution effect context measure disease risk in a particular host, many focus on community-wide disease. Community-level prevalence comprised *ca*. 11%, 27% and 15% of studies from dilution effect meta-analyses by Civitello et al. (2015), Magnusson et al. (2020), and Salkeld et al. (2013), respectively (Table S1). Variation in disease metrics alters diversity–disease relationships (Luis et al., 2018; Roberts & Heesterbeek, 2018; Young et al., 2014). This overlooked distinction between individual- and community-level observations might inflate evidence for dilution effects.

To empirically evaluate dilution mechanisms underpinning the disease–diversity relationship and the influence of aggregation, we studied plant communities impacted by sudden oak death, an emerging forest disease that has killed at least 48 million stems of tanoak (*Notholithocarpus densiflorus*) and oak species (*Quercus* spp.) in coastal California and southwestern Oregon since 1995 (Cobb et al., 2020; Rizzo & Garbelotto, 2003). The causal agent, *Phytophthora ramorum*, is an invasive oomycete pathogen with a wide host range, though some hosts exhibit symptoms more often than others. Field studies in California suggest that transmission is driven primarily by two species: bay laurel (*Umbellularia californica*) and, to a lesser extent, tanoak (Davidson et al., 2005, 2008). Whether other forest plant species also contribute to inoculum pressure via asymptomatic sporulation, reduce transmission success, or have no effect on transmission is unknown.

We combined laboratory competence measurements with high-resolution plant community and disease symptom data from a large network of plots in the Big Sur region of California. In a previous analysis of this field-collected dataset, community-level disease prevalence declined with both plant species richness and Shannon–Wiener diversity index, even after accounting for the densities of known competent hosts, bay laurel and tanoak (Haas et al., 2011). Although other species might underly dilution mechanisms, such as 'competent host regulation' (via asymptomatic sporulation) or 'encounter/transmission reduction' (modified *sensu* Keesing et al., 2006), it is difficult to assess without investigating individual-level disease risk. In order to test whether this negative diversity–disease relationship arose from dilution mechanisms, or from the fact that disease was averaged across the community, we tested three hypotheses:

1. The dilution effect is driven by competent host regulation, indicated by decreases in individual- and community-level disease risk with diversity, with associated decreases in competent host density.
2. The dilution effect is driven by encounter/transmission reduction, indicated by decreases in individual- and community-level disease risk with diversity, which persist after accounting for changes in competent host density.
3. The negative diversity–disease relationship is a product of how disease is measured, indicated by decreases in community-level, but not individual-level, disease risk with diversity.

Our study explores the empirical foundation linking community composition, competence and different disease metrics. Understanding these links is essential to predicting where diseases may emerge or decline as a function of global threats to biodiversity.

MATERIALS AND METHODS

Study system

Our study was conducted in redwood and mixed evergreen forest types in the Big Sur region of California. Redwood forests are typified by redwood (*Sequoia sempervirens*) canopies, with bay laurel, tanoak, pacific madrone (*Arbutus menziesii*) and various oak species in the subcanopies. Mixed evergreen forests occupy drier sites and consist of similar species excluding redwood.

In this system, woody plants fell into three categories in regard to *P. ramorum*: `commonly symptomatic', 'rarely symptomatic' and nonhosts. We considered bay laurel, tanoak, coast live oak (*Quercus agrifolia*) and Shreve oak (*Q. parvula*) to be commonly symptomatic hosts because they accounted for the majority of detected infections. Infected true oaks and tanoaks may develop lethal stem cankers, while bay laurels do not experience disease-induced mortality (Rizzo et al., 2005). Infectious propagules (sporangia) formed on foliar and branch lesions are most prolifically produced on bay laurel, followed by tanoak (Davidson et al., 2005, 2008), and are very rarely observed on true oaks (Vettraino et al., 2008). Infections on other, more rarely symptomatic hosts typically lead to nonlethal foliar and branch lesions.

Plot network design and data collection

In 2006 and 2007, plant community and disease data were collected in 500 m^2 plots established to monitor long-term sudden oak death dynamics (see Metz et al., 2011). All woody stems at least 1 cm diameter at breast height were recorded for species identity, live/dead status, and visually assessed for *P. ramorum* symptoms. Plant individuals with any symptomatic live stems were considered diseased. Note that we assessed disease—not infections, opening the possibility that some plants were asymptomatically infected (Denman et al., 2009).

We studied 151 plots where the pathogen was confirmed present using culture-based methods (Figure 2; see Appendix S1 for details, including how our selected

plots differed from Haas et al., 2011). We adopted host/nonhost categorisations from Haas et al. (2011), defined by whether or not natural infections had been identified on that species (Davidson et al., 2003). We measured density of species using total basal area, which better captures variation in tree sizes than counts of individual plants, and the number of individuals, which directly influences community-wide disease prevalence ($\frac{\text{Infected host individuals}}{\text{Total host individuals}}$). Plot diversity was characterised using species richness of woody plants.

To account for other sources of heterogeneity that may correlate with species richness, the same climatic, topographic and landscape characteristics used by (Haas et al., 2011) were estimated for each plot. We used the 30-year mean wet-season precipitation (December–May) calculated from Parameter Elevation Regression on Independent Slopes Model (PRISM; Daly et al., 1994); potential solar insolation (PSI; Dubayah & Rich, 1995); and the area of host vegetative coverage within 200 m of plot center (Meentemeyer et al., 2008b).

Host and community competence

To evaluate how the entire plant community might contribute to overall inoculum pressure, we estimated host competence from the 10 most commonly occurring species in the two forest types (13 species in total; Rosenthal et al. in press). In Spring 2019, leaves from 32 individuals per species were collected in the Big Sur region and inoculated with *P. ramorum* in the laboratory. Sporulation was quantified after 5 days of incubation by scraping the leaves, collecting the solution and counting sporangia under the microscope.

We estimated community competence (K) as the cumulative density of each species weighted by their competence (modified from Johnson et al., 2013): $K = \sum_i^S c_i n_i$, where c_i is the mean competence and n_i is the total basal area of species i for S total species per plot. Each species' component contribution to K was calculated as $k_i = c_i n_i$. For species not examined in the competence assay, we assumed missing values were the median of the quantified host competencies. Since these species comprised

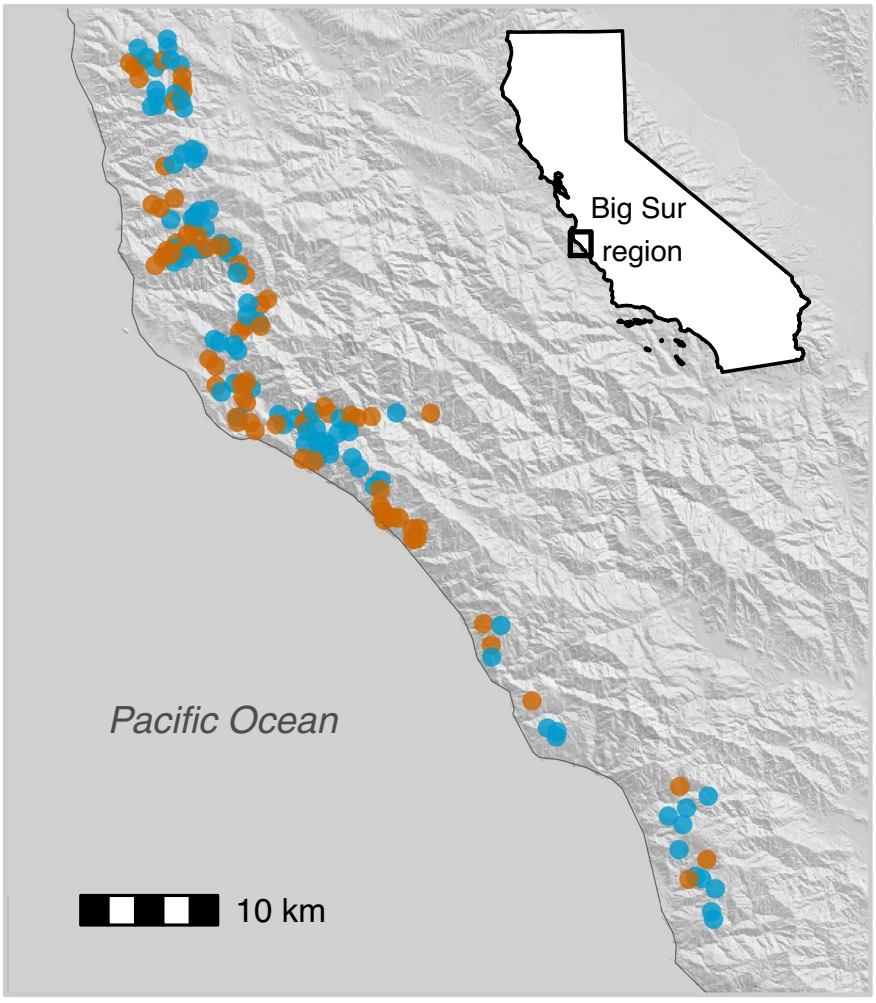

FIGURE 2 Map of 151 study plots located in the Big Sur coastal region of California, USA. Bounding box in the inset state map designates the closeup area. Plots were split among mixed evergreen (blue) and redwood (orange) forest types

only 0.7% of the basal area in the dataset, assumptions about their values had a negligible effect.

Statistical analyses

How density varies with richness

To understand linkages between community composition and disease, we evaluated several measurements of density in relation to plot richness and forest type. Densities of known competent hosts, bay laurel and tanoak, were investigated in separate hurdle models. We predicted the probabilities of their occurrences with a Bernoulli generalised linear model (GLM) and when a species was present in a plot, its basal area was estimated with a gamma GLM. We used a gamma GLM to explore if densities of these hosts could be explained by the relationship between total plant basal area and richness. Additionally, we analysed the total number of either commonly or rarely symptomatic host plants per plot, using separate negative binomial GLMs.

How community competence varies with richness

To test whether a negative covariance between community competence and richness might explain the past negative diversity–disease relationship, community competence was modeled with a log-normal likelihood and included predictors for plot richness and forest type. A predictable relationship between community competence and diversity is predicated on nested communities. We calculated a nestedness metric based on overlap and decreasing fill (NODF; Almeida-Neto et al., 2008) and compared it against 999 null permutations (proportional row and column totals; Strona & Fattorini, 2014) using an online software (Strona et al., 2014).

How disease risk varies with richness, known competent hosts and community competence

To address if competent host regulation, encounter/transmission reduction or aggregation of observations drove the previous negative diversity–disease relationship, we estimated disease risk at the community and individual level. For both hierarchical levels, we contrasted three explanatory models, which included covariates for M1) richness, M2) richness and basal area of tanoak and bay laurel, and M3) richness and community competence. If competent host regulation was a driving mechanism, we expected individual-level disease risk to be negatively associated with richness in M1 and positively associated with either host densities in M2 or community competence in M3. Additionally, if plant species besides tanoak or bay laurel enhanced transmission risk, M3 would have a greater predictive performance than M1 and M2. If encounter/transmission reduction was a contributing factor, we expected to still see a negative effect of richness on individual-level disease risk after incorporating host densities in M2 and/or community competence in M3. Lastly, if the negative diversity–disease relationship was a product of aggregation of observations, we expected to see a negative effect of richness on disease risk at the community, but not individual level.

To isolate how inclusion of rarely symptomatic host species might alter the calculation of community-level disease prevalence, community-wide disease was analysed both for all hosts (commonly and rarely symptomatic species) and for the four commonly symptomatic host species. Community-level disease prevalence was estimated by modeling I_j, the number of diseased plants in plot j, given n_j, the total number of host plants (j = 151 plots). To capture overdispersion in the response variable, we used a beta-binomial likelihood with μ_j, the expected value of probability of disease p_j, and a dispersion parameter θ:

$$\begin{aligned} I_j &\sim \text{Binomial}(n_j,\ p_j) \\ p_j &\sim \text{Beta}(alpha_j,\ beta_j) \\ alpha_j &= \mu_j\,\theta \\ beta_j &= (1-\mu_j)\,\theta \\ logit(\mu_j) &= \alpha_0 + BX_j \end{aligned} \tag{1}$$

where α_0 is the global intercept and B is a vector of coefficients for the covariates contained in the data matrix X_j. In addition to the covariates mentioned above (richness, host basal areas and community competence), we incorporated variables for forest type, sample year, precipitation, PSI and host vegetation in the surrounding landscape in order to control for confounding effects from the sampling design and landscape heterogeneity.

Individual-level disease risk was assessed for the four commonly symptomatic hosts. We modeled I_i, the disease status of individual i of species s located in plot j, using a Bernoulli likelihood with a mean probability p_i (i = 4206 individuals from 151 plots and 4 species):

$$\begin{aligned} I_i &\sim \text{Bernoulli}(p_i) \\ \text{logit}(p_i) &= \alpha_{j[i]} + \alpha_{s[i]} + \beta_{s[i]} richness_{j[i]} + \gamma BA_i \\ \alpha_j &\sim \text{Normal}(BX_j,\ \sigma_{plot}) \\ \begin{bmatrix} \alpha_s \\ \beta_s \end{bmatrix} &\sim \text{MVNormal}\left(\begin{bmatrix} \alpha_0 \\ \overline{\beta} \end{bmatrix},\ \boldsymbol{\Sigma} \right) \end{aligned} \tag{2}$$

where intercept α_j varied by plot, intercept α_s and the effect of richness β_s varied by species, and γ characterised the basal area of the plant (summed among live stems) in order to account for size-dependent variation in susceptibility. Plot-level intercepts were normally distributed with a mean BX_j, defined by the same predictor variables as described previously in the community-level models. α_s and β_s were drawn from a multivariate normal distribution, defined by means α_0 and $\overline{\beta}$ and covariance matrix $\boldsymbol{\Sigma}$.

Host densities and community competence were square root transformed to spread the right-skewed distributions. All variables were centered and scaled by dividing by 2 SD (Gelman, 2008). Collinearity was assessed by confirming that correlations between continuous variables were <0.5 (Figure S1). We contrasted model predictive performance by computing approximate leave-one-out cross-validation, comparing models based on the difference in expected log pointwise predictive density (ELPD; Vehtari, 2017). We tested for spatial autocorrelation using a Moran's I correlogram on the mean residuals from the best performing community-level disease prevalence including all hosts. No significant spatial clustering emerged (Figure S2). Additional information about our treatment of spatial autocorrelation is in Appendix S1.

Model fitting

Models were written in the Bayesian programming language Stan (Stan Development Team, 2019) and analysed in the R environment (R Core Team, 2019; Stan Development Team, 2020). Packages used for our analysis are listed in Appendix S1. We used weakly informative priors and 4 chains with 2000 iterations each. Model fits were visually evaluated by comparing observed values against posterior predictive draws (Figure S3–S6). Parameter estimates with 90% highest posterior density intervals (HPDI) that did not contain zero (or one, when expressed as odds ratios) were considered to have important, non-zero effects on the response variable. A common default in Bayesian analyses is to use 90% HPDIs because they are more stable than 95% intervals (Goodrich et al. 2020).

RESULTS

Across 151 plots, 5798 trees and shrubs were included in our study and 18 species were considered hosts and 9 as nonhosts (Table S2). Four commonly symptomatic species accounted for 99.6% of detected infections. Symptoms were primarily found on the two most ubiquitous and abundant species, bay laurel (923 symptomatic/1104 total plants, 83.6%) and tanoak (1153 symptomatic/2189 plants, 52.6%), while there were fewer on coast live oak (36 symptomatic/296 plants, 12.2%) and Shreve oak (28 symptomatic/617 plants, 4.5%). The other 14 host species were rarely symptomatic. Of these species, only eight redwoods and one California buckeye (*Aesculus californica*) were symptomatic.

How density varies with richness

Total basal area of all species remained constant across richness in both forest types (Figure 3a). Bay laurel occurred more frequently in richer plots, while tanoak occurrence did not vary strongly with richness (Figure 3b, c). When present, the basal area of bay laurel had a weakly negative relationship with richness (median, 90% HPDI = −0.15 [−0.31, 0.03]), whereas that of tanoak did not vary considerably (−0.08 [−0.29, 0.14]; Figure 3b, c). Additionally, the number of rarely symptomatic host plants increased with richness, while the number of commonly symptomatic host plants did not change substantially (Figure 3d, e).

How community competence varies with richness

Mixed evergreen and redwood forest communities were both significantly nested (mixed evergreen: $NODF_{obs} = 50.2$, $p < 0.001$; redwood: $NODF_{obs} = 58.5$, $p < 0.001$), indicating that depauperate communities were nested subsets of their richer counterparts (Figure 4a). Species-poor communities were more likely to contain ubiquitous species, while richer communities also consisted of rarer species, which tended to be less competent. Bay laurel and tanoak were more competent than the other measured species (Figure 4b). Within mixed evergreen forests, the species that contributed most to community competence were bay laurel followed by tanoak, and in redwood forests they were tanoak, followed by bay laurel and redwood (Figure 4c). Although redwood is a low-competence host, it is the largest tree species in the forest and very common. Total community competence was higher in redwood forests than mixed evergreen forests and it declined in plots with higher richness (Figure 4d).

How disease risk varies with richness, known competent hosts and community competence

Across all models, surrounding host vegetation had consistently positive effects, redwood forests and historical precipitation levels had negative or no effects, and sampling year and PSI had negligible effects on community- and individual-level disease risk (Table S3–S5). After accounting for variation related to these factors, the importance of richness on disease risk and its association with known competent hosts and community competence varied depending on how disease was measured.

Disease prevalence aggregated among all hosts in the community decreased with richness (median odds ratio, 90% HPDI = 0.68 [0.49, 0.90]; Figure 5a). We included bay laurel basal area, tanoak basal area and community competence into subsequent models, all of which had positive effects (Figure 5a). After accounting for variation in bay laurel and tanoak density, the negative richness-disease covariance weakened only slightly (0.70 [0.52, 0.96]), and it further weakened when community competence was instead incorporated (0.79 [0.58, 1.08]). Disease prevalence was best predicted by the model

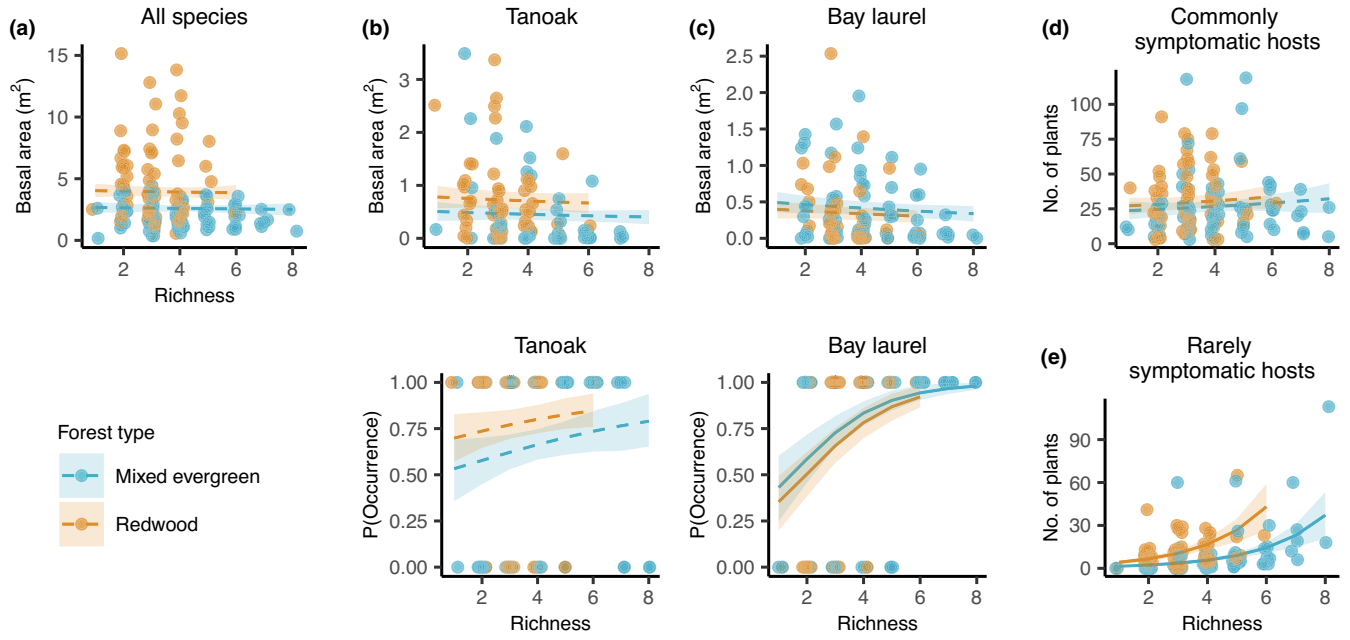

FIGURE 3 Relationships between richness and various measurements of plant density by forest type. (a) Density measured as total plant basal area. (b, c) Hurdle models measuring tanoak or bay laurel density, which assessed probability of occurrence (bottom) and, conditional upon presence, assessed basal area (top). (d) Density measured as number of commonly symptomatic host plants. (e) Density measured as number of rarely symptomatic host plants. Points are horizontally jittered. Lines and shaded regions represent the median and 90% HPDI of the posterior estimate of the mean. Solid lines indicate the 90% HPDI of the effect of richness did not cross zero

featuring richness and host basal area (M2), outperforming the models including community competence (M3: $\Delta ELPD = -13.9$, $SE_\Delta = 3.6$) and richness only (M1: $\Delta ELPD = -20.1$, $SE_\Delta = 5.8$).

When detected infections were examined exclusively among the four commonly symptomatic species, richness no longer had a nonzero effect on disease prevalence (odds ratio: 0.84 [0.61, 1.17]; Figure 5b). Bay laurel and community competence had positive effects, while the effect of tanoak diminished. The negligible effect of richness did not change when models included host basal area or community competence. The model with richness and host basal area (M2) performed better than the models with community competence (M3: $\Delta ELPD = -16.8$, $SE_\Delta = 4.7$) and richness alone (M1: $\Delta ELPD = -23.7$, $SE_\Delta = 6.7$).

Individual-level disease risk models accounted for species-specific disease rates, which were highest for bay laurel, followed by tanoak, coast live oak and Shreve oak (Figure 6a). The models also controlled for size-dependent variation in susceptibility, which was greater for larger individuals (Table S5). Richness on average was not strongly correlated with disease risk in the model including richness only (odds ratio: 1.31 [0.62, 2.75]), and its effect did not substantially change after including predictors for host basal area or community competence (Figure 5c). Across the three explanatory submodels, species-specific effects of richness for coast live oak, Shreve oak, and tanoak were unlikely important (90% HPDI contained one); meanwhile, richness had a positive effect on disease risk for bay laurel, with credible intervals slightly smaller or larger depending on the covariates included in the model (Figure 6b). Disease risk was not strongly correlated with tanoak basal area, positively correlated with community competence, and strongly, positively correlated with bay laurel basal area (Figure 5c). The model including richness and host basal area (M2) marginally outperformed models including community competence (M3: $\Delta ELPD = -3.7$, $SE_\Delta = 2.3$) or richness alone (M1: $\Delta ELPD = -4.9$, $SE_\Delta = 2.5$).

DISCUSSION

Despite frequent tests of negative diversity–disease relationships in natural ecosystems, the mechanisms remain poorly resolved. We tested how relationships among species richness, densities of keys hosts, community competence, and disease risk metrics vary in a forest system previously shown to exhibit negative diversity-disease patterns (Haas et al., 2011). Richness had no limiting effect on individual-level disease risk, and therefore neither competent host regulation nor encounter/transmission reduction were possible dilution mechanisms. Rather than depending on the composition of the entire community, average risk of acquiring disease was largely driven by a single, common, highly competent host, bay laurel (Figure 5c). This species' density did not have a clear relationship with richness, which may explain the lack of a dilution effect evaluated at the individual level. In contrast, the negative effect of richness on community-level

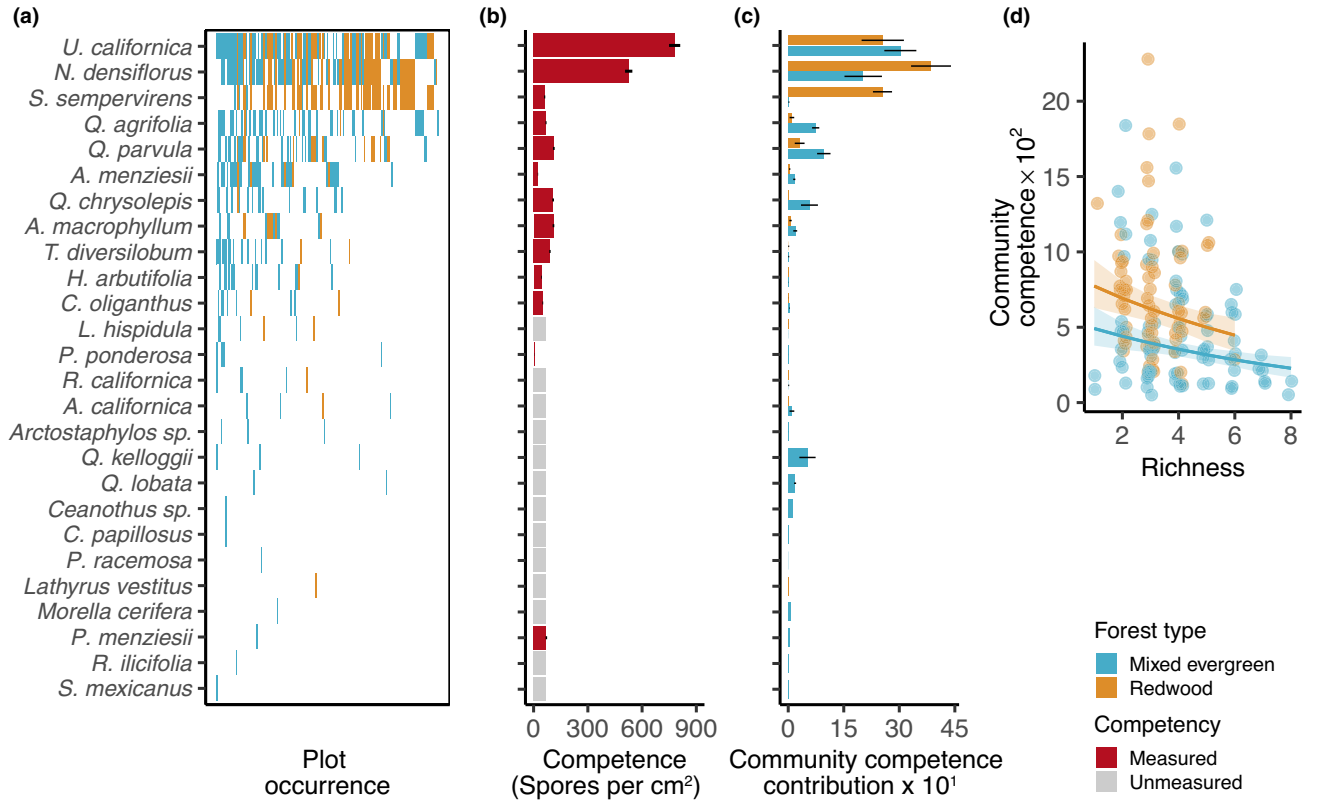

FIGURE 4 Nestedness and the linkages between host competence and diversity in both forest types. (a) Matrix of species that are present among the 151 study plots. The top rows represent the most ubiquitous species and the leftmost columns represent the richest plots. In a perfectly nested set of communities, the depauperate communities would consist of a subset of the species present their richer counterparts, causing this matrix to be filled entirely in the upper left-hand side. (b) Sporulation potential (mean Â± SE) as assessed in laboratory inoculation assays. Species are in order of rank ubiquity. (c) Each species' contribution towards community competence (mean Â± SE). (d) The relationship between richness and community competence with points horizontally jittered. Line and shaded region represents the median and 90% HPDI of the posterior estimate of the mean. Solid lines indicate the 90% HPDI of the effect of richness did not cross zero. Species not included in the laboratory sporulation assays (grey) are estimated as the median of those that were measured. Analyses are shown separately for each forest type, mixed evergreen (blue) and redwood (orange).

disease prevalence was solely attributable to its positive covariance with the number of rarely symptomatic host species. Rarely symptomatic host species reduced the relative density of commonly symptomatic hosts without significantly altering their individual risks of disease (Figure 1c). Aggregating disease prevalence at the community level may misattribute dilution mechanisms and bias towards negative diversity–disease relationships, which has consequential implications for the effects of conserving biodiversity in disease-impacted ecosystems.

Diversity-associated mechanisms of individual-level disease risk

While multiple host species of varying competence may contribute to transmission risk (Hamer et al., 2011; Searle et al., 2016), sometimes generalist pathogens are influenced by the presence of a single host species (Wilber et al., 2020). The risk of acquiring disease symptoms primarily depended upon the basal area of bay laurel, which we uncovered using models that estimated individual-level disease risk. Less competent hosts were not essential in predicting disease risk. Consistent with other field studies in California, basal area of the next most competent host, tanoak, was not influential (Meentemeyer et al., 2008a; Simler-Williamson et al., 2021; Swiecki & Bernhardt, 2002) and community competence, a weighted mean of all species' transmission potentials, had a weaker effect than bay laurel and did not improve model predictive performance relative to the model including bay laurel density. Accordingly, the effect of richness mostly hinged upon its correlation with bay laurel occurrence and abundance.

Theory predicts that when the most competent species has a low extirpation risk, communities are nested, and total density remains invariant with diversity ('substitutive assembly'), there should be a higher density of competent hosts in species-poor communities (Joseph et al., 2013; Mihaljevic et al., 2014; Rudolf & Antonovics, 2005). Each of these conditions was met and indeed, we found that the basal area of bay laurel was slightly higher in depauperate communities (Figure 3a). However, bay laurel was also less likely to persist in species-poor

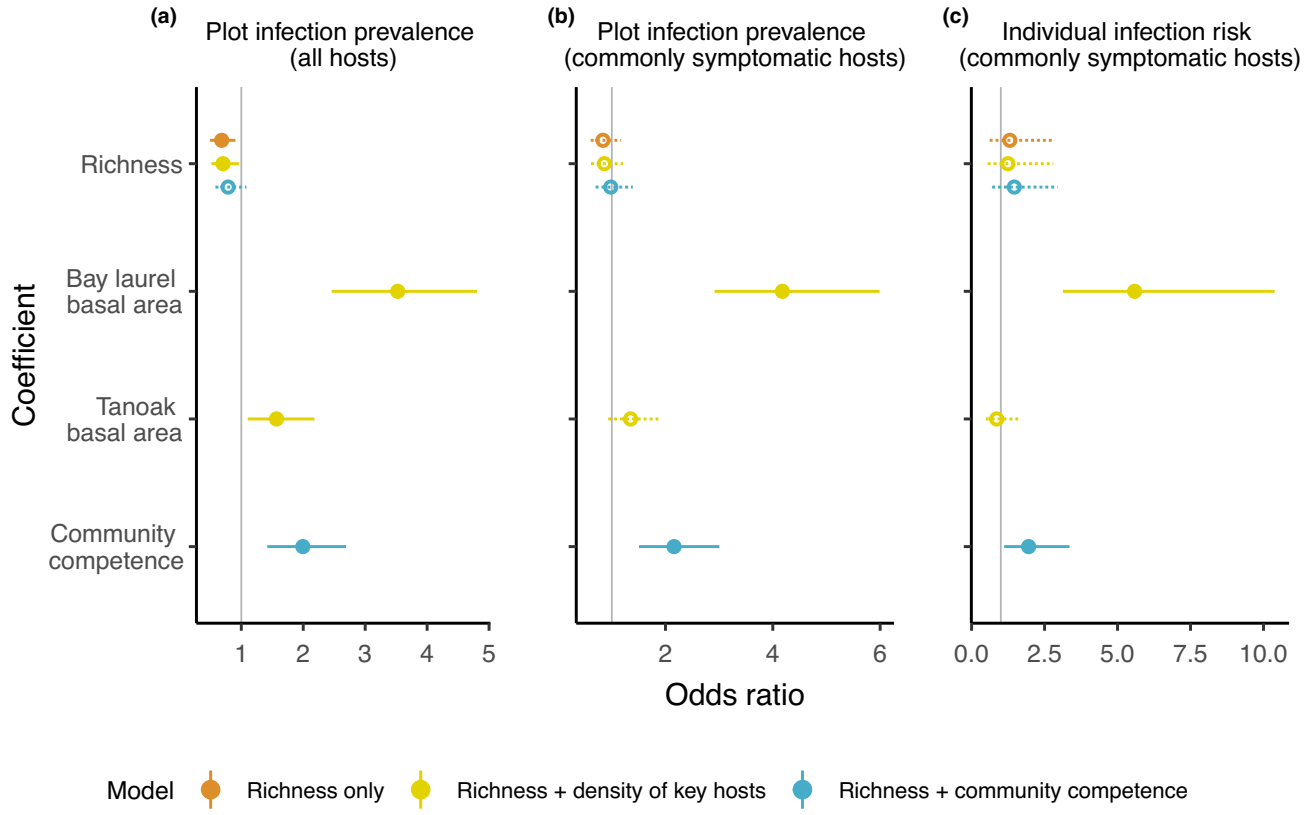

FIGURE 5 Effects of community-related covariates of disease risk models evaluated as (a) community-level disease prevalence for all hosts, (b) community-level disease prevalence for commonly symptomatic hosts, and (c) individual-level disease risk for commonly symptomatic hosts. Note that panel C shows the mean effect of richness (β, Equation 2), while species-specific parameters of the individual-level disease risk models are displayed in Figure 6. The three colors represent the three explanatory models (M1. Richness only, M2. Richness + density of key hosts, M3. Richness + community competence) being contrasted within each disease risk metric. Posterior estimates are displayed with the median and 90% HDPI, with intervals not crossing one shown with a solid line and closed points

communities (Figure 3a). The combined effect of these two opposing variables (basal area and occurrence) likely led to a weak overall association between bay laurel density and richness, and no corresponding shift in individual-level disease risk averaged among the commonly symptomatic species.

By contrast, community competence, based on laboratory sporulation assays, did decline with richness, and yet this did not lower individual-level disease risk in more diverse plots. Measurements from artificial inoculations do not integrate variation due to host phenology, forest structure, and climate (Dodd et al., 2008; Davidson et al., 2011; Simler-Williamson et al., 2021; Rosenthal et al. in press), nor variation within species or individuals (Stewart Merrill & Johnson, 2020). These challenges are logistically difficult to overcome for a broad set of large, long-lived tree species. Community competence currently weights the contribution from bay laurel and less competent hosts. If community competence were calibrated to more accurately reflect natural inoculum pressure, it might primarily reflect bay laurel density.

When effects of richness were parsed for each species, richness had undetectable effects on disease risk for tanoak or oaks, but it had a positive effect for bay laurel. This result could be highly impactful given how central bay laurel is to pathogen spread. The positive effect of richness may reflect a correlation with unaddressed, disease-inducing factors, such as microclimates or pathogen invasion history. Plots with greater richness may have been invaded earlier by this nonnative pathogen, and thus *P. ramorum* would have more time to spread within those stands (Cobb et al., 2020).

Diversity-associated mechanisms of community-level disease risk

Richness was negatively associated with disease prevalence for all hosts in a plot, which is best explained by the relative abundance of commonly symptomatic species. The number of rarely symptomatic host plants increased with richness, while the number of commonly symptomatic plants (accounting for 99.6% of detected infections) did not change. The proportion of commonly symptomatic hosts negatively covaried with richness, limiting the fraction of community-wide disease. Without rarely symptomatic species, models of community-level disease prevalence led to similar conclusions as the

individual-level analysis—bay laurel density drove detected infections and richness did not have a strong effect. By aggregating disease among all hosts in a community, low-competence, rarely symptomatic hosts numerically diluted the proportion of symptomatic plants without affecting transmission risk to susceptible populations.

Differences in the diversity–disease relationship across hierarchical levels

Individual- and community-level disease risk varied independently with respect to the density of competent hosts and proportion of symptomatic hosts, respectively. Thus, the direction and drivers of the diversity–disease relationship are distinct across hierarchical levels. However, this distinction is easily conflated. For instance, the negative effect of richness on community-level disease prevalence remained after accounting for tanoak and bay laurel densities. Haas et al. (2011) acquired similar results and hypothesised richer communities contained either more noncompetent plants that interfered with inoculum dispersal pathways (`encounter reduction'; Figure 1b), or fewer asymptomatic, competent hosts that illusively caused infections ('competent host regulation'; Figure 1a). Noncompetent species inhibit encounter rates when they physically block local transmission. Pathogens with root-to-root transmission are good candidates to observe this mechanism, unlike *P. ramorum* where sporangia travel distances of up to 4 km (Hansen et al., 2008; Mascheretti et al., 2008). Yet, richness became unimportant after adding community competence to the model predicting community-level prevalence (Figure 5a), suggesting that asymptomatic transmission from many forest species may explain the negative diversity–disease relationship. However, we instead interpret this finding as a spurious correlation since our individual-level models indicate that bay laurel was the primary host driving disease.

Community-level observations cannot directly explain processes occurring between (susceptible and infectious) individuals, and our study represents a case of Simpson's paradox, in which correlations are not preserved during data aggregation (Simpson, 1951). Salkeld and Antolin (2020) illustrated that disease aggregated across large spatial scales can lead to spurious correlations with diversity and explanatory factors, and these relationships might reverse if reexamined using individual- or species-level data. Our results, and others' (Piudo et al., 2011), confirm that aggregating disease at the community level can generate this pattern. Although not examined in our study, community-wide disease caused by multiple pathogens (e.g. ``community pathogen load'' *sensu* Mitchell et al., 2002, which also averages across species) can produce similar mismatches (e.g. Hantsch et al., 2013). To be clear, we believe individual- and community-level disease metrics are equally valid and important to study; however, mechanisms used to explain diversity–disease relationships need to reflect the levels at which disease was measured.

We also suspect that community-level prevalence may negatively correlate with diversity more frequently

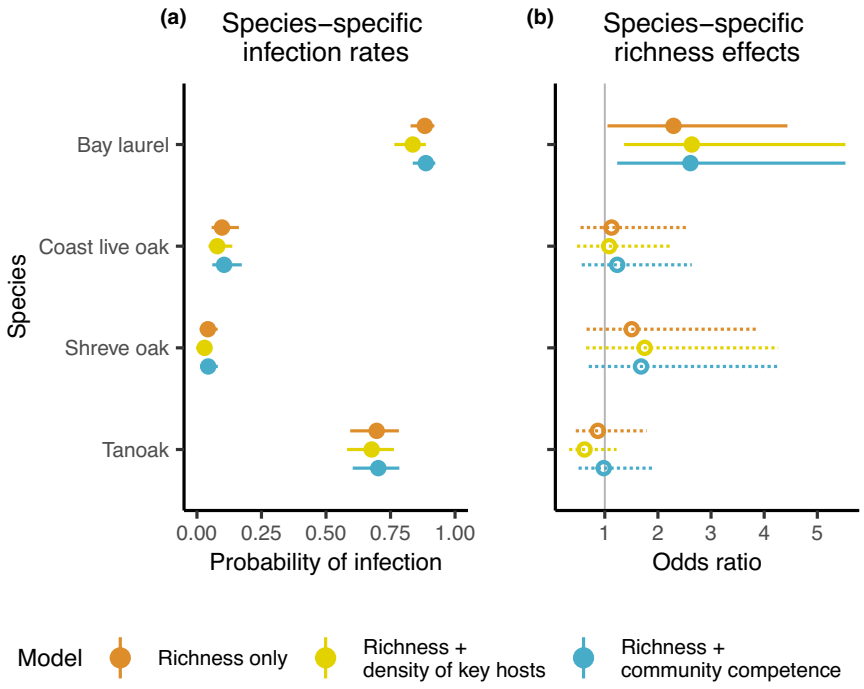

FIGURE 6 Additional posterior estimates of the individual-level disease risk models, including species-specific (a) intercepts and (b) effects of richness, representing α_s and β_s for species s respectively (see Equation 2). Posterior estimates are displayed with the median and 90% HDPI, with intervals not crossing one shown with a solid line and closed points

than individual-level disease risk under specific assembly patterns. When depauperate communities are dominated by disease-prone species—which is more often the case than not (Gibb et al., 2020; Joseph et al., 2013), even in the absence of dilution mechanisms, less susceptible species added to higher diversity communities would increase the likelihood of observing a decline in overall prevalence. Diversity often negatively covaries with community-level prevalence (Bradley et al., 2008; Liu et al., 2018; Moore & Borer, 2012), but not always (Hydeman et al., 2017; Milholland et al., 2017; Vaz et al., 2007). Community-wide disease risk is not uncommon under the dilution effect purview (e.g. Table S1; Mitchell et al., 2002), and whether it biases toward negative diversity–disease relationships deserves closer attention.

Given that diversity–disease relationships may change across hierarchical levels, what was the most appropriate measure of disease? Response variables need to match questions meaningful to management (Johnson et al., 2015). Managing ecosystem health is an important goal. In our system, the overall percentage of diseased host plants is critical for predicting how disease-induced mortality affects fuels, carbon sequestration, or resilience to large-scale disturbance (Cobb et al., 2020; Metz et al., 2011; Simler et al., 2018). Conserving biodiversity may still improve ecosystem health when richness is correlated with a lower proportion of susceptible species. Other times, the goal is to manage the health of specific hosts, which aligns with the majoritarian notion of the dilution effect. We examined disease risk in four species and accounted for differences in species-specific susceptibility. Here, maintaining diverse forest stands would not reduce the risk of individuals acquiring disease and targeted management of bay laurel is needed.

CONCLUSION

Two unresolved topics in disease ecology involve exploring how diversity correlates with species composition and the consequences on disease risk, and how disease measured at the individual or community level affects conclusions (Johnson et al., 2015). We found that the overall density of the most competent species likely did not have a strong relationship with richness and, consequently, richness did not limit individual-level disease risk. Empirical tests of this pattern must continue in other naturally assembled communities, especially in forests and other understudied systems. We also found that richness can have a positive or negligible effect on disease at the individual level while concomitantly having a negative effect at the community level. Understanding these multilevel differences is key for managing the health of the ecosystem versus specific forest species. Looking forward, one solution is to explicitly define the currently vague description of `disease risk', which will require discussion among research, management, and policy priorities (see Keesing et al., 2006). A more expansive prospect is for researchers to contrast various metrics of disease to uncover how, why, and for which species biodiversity affects disease.

ACKNOWLEDGEMENTS

The authors thank Felicia Keesing, Tara Stewart-Merrill, and Emily Brodie for feedback on earlier drafts. The authors thank the reviewers and editor, whose comments undoubtedly improved the manuscript. We thank the Landels-Hill Big Creek Reserve, CA State Parks, US Forest Service, and many private landowners for research access. Field data collection was funded by the NSF-NIH Ecology and Evolution of Infectious Diseases (DEB-1115664) and NSF Ecosystem Science (ES-1753965) programs, USDA Forest Service Pacific Southwest Research Station & Forest Health Protection, State and Private Forestry, the Gordon and Betty Moore Foundation, and an NSF Graduate Research Fellowship awarded to LMR.

AUTHORSHIP

DMR conceived the plot network study design. LMR designed the conceptual framework and led the analysis in consultation with ABS-W. LMR wrote the first manuscript draft and all authors contributed to revisions.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/ele.13871.

OPEN RESEARCH BADGE

This article has earned an Open Data badge for making publicly available the digitally-shareable data necessary to reproduce the reported results. The data is available at: https://doi.org/10.17605/OSF.IO/GZFJR, URL: https://osf.io/gzfjr/.

DATA AVAILABILITY STATEMENT

We confirm that, should the manuscript be accepted, data will be publicly available. Data and model code are located in a public Open Science Framework project: https://osf.io/gzfjr/.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the Supporting Information section.

How to cite this article: Rosenthal, L.M., Simler-Williamson, A.B. & Rizzo, D.M. (2021) Community-level prevalence of a forest pathogen, not individual-level disease risk, declines with tree diversity. *Ecology Letters*, 24, 2477–2489. https://doi.org/10.1111/ele.13871