Chapter 3. Species densities, assembly order, and competence jointly determine the diversity-disease relationship

Lisa M. Rosenthal[1,2], Wesley R. Brooks[3], David M. Rizzo[2]

[1] Graduate Group in Ecology, University of California, Davis, CA 95616, USA

[2] Department of Plant Pathology, University of California, Davis, CA 95616, USA

[3] DataLab, University of California, Davis, CA 95616, USA

Abstract

Since individual species vary in abundance and host competence, species composition strongly influences disease dynamics. In the midst of biodiversity loss, forecasting its effects on disease depends on community (dis)assembly, the processes determining how species are added (or lost) from communities. We simulated community assembly by planting mesocosms, nested along a richness gradient, and tested how relationships between richness and either species loss order or overall density affect disease risk. Mesocosms containing up to 6 crop species of varying competence were inoculated with a soilborne fungal pathogen, *Rhizoctonia solani*. Disease was measured as species-level prevalence, community-level prevalence, and total number of diseased plants. Regardless of metric, richness limited disease when species loss order negatively correlated with competence and overall species density was intransient with richness. When total density increased with richness or species were lost randomly, richness primarily correlated positively or weakly with disease. Our results are consistent with theoretical expectations and represent the first empirical study simultaneously testing the influence of species densities, disassembly order, and competence on diversity-disease relationships.

Introduction

How does community assembly affect disease risk? Since host species vary in their ability to become infected and transmit a given pathogen (i.e. 'host competence'), patterns in community (dis)assembly, defined as processes that determine which species and how many are added (or lost) from communities, can differentially impact infectious disease in communities undergoing biodiversity loss (Joseph *et al.* 2013; Halliday *et al.* 2019). Understanding how and when species loss influences disease dynamics is paramount for predicting and managing future outbreaks; addressing the role of community assembly may be key for identifying conditions underlying diversity-disease relationship in natural ecosystems.

When highly competent host species are least likely to be extirpated during species loss, there is an increased likelihood that diversity and overall transmission risk negatively covary (Ostfeld & LoGiudice 2003), a phenomenon termed the 'dilution effect' (Keesing *et al.* 2006). Negative relationships between competence and extirpation risk may be attributed to life history tradeoffs. For example, 'weedy' species tend to dedicate fewer resources to disease resistance (Cronin *et al.* 2010; Heckman *et al.* 2019). However, deterministic and stochastic forces shape community disassembly (Fukami *et al.* 2005; Halliday *et al.* 2019). When species loss order is unrelated to host competence, changes in disease risk are less predictable and the diversity-disease relationship is expected to be idiosyncratic (Ostfeld & LoGiudice 2003; Joseph *et al.* 2013).

Even when competence negatively correlates with extirpation risk, changes in disease also depend on how overall density covaries with diversity loss (Searle *et al.* 2016). At one extreme, total community abundance may remain invariant with richness. Under this 'substitutive assembly', a loss of diversity may release competent host species from strong resource limitations, leading to a higher absolute density of competent hosts, and therefore, higher disease risk in species-poor communities (Rudolf & Antonovics 2005; Mihaljevic *et al.* 2014). Conversely, community abundance may positively correlate with diversity ('ad-

ditive assembly'), such that the densities of individual species do not covary with richness. Host densities would be greatest in species-rich communities and for generalist pathogens with density-dependent transmission, disease risk should positively correlate with diversity (Rudolf & Antonovics 2005; Mihaljevic *et al.* 2014). Altogether, it is expected that a negative diversity-disease relationship is most likely when community assembly is substitutive and extirpation risk negatively correlates with competence (Joseph *et al.* 2013; Johnson *et al.* 2015).

Despite a strong theoretical foundation explaining how competence, species order loss, and abundance may influence the diversity-disease relationship, empirical tests are limited. Correlational studies of natural communities are powerful for identifying how species composition varies across richness levels (Johnson *et al.* 2013; Lacroix *et al.* 2014), which is essential for predicting how communities would likely disassemble. Nevertheless, the effects of different assembly patterns on disease risk may be best addressed under an experimental framework where species composition can be manipulated independently of diversity. To date, experimental studies focused on the dilution effect have compared random versus non-random species loss (Liu *et al.* 2018; Johnson *et al.* 2019) and additive versus substitutive assembly (Johnson *et al.* 2013; Wojdak *et al.* 2014). However, interactions between these two influential axes of community assembly have yet to be empirically investigated.

Moreover, the relationship between diversity and disease can be sensitive to whether disease is measured at the community or species level. The dilution effect typically addresses how diversity impacts the risk of individuals acquiring disease, and hence disease is evaluated for particular host species. The dilution effect has also been assessed using community-level disease metrics, including total propagule load (e.g. Young *et al.* 2014) or average community disease severity or prevalence (e.g. Haas *et al.* 2011; Mitchell *et al.* 2002), which capture overall transmission potential or disease burden, respectively. Despite their fundamental epidemiological differences, various metrics of disease for focal species and overall host communities frequently appear together in the dilution effect literature and can lead to

divergent outcomes on diversity-disease patterns (Rosenthal et al. in review).

Here we used artificial plant mesocosms to examine the effects of species identities and densities on disease caused by a fungal plant pathogen, *Rhizoctonia solani*. This generalist soil-borne pathogen causes visible aboveground damping-off symptoms when it infects belowground host tissue (Otten *et al.* 2003). Transmission occurs between plants through infective hyphae and slows or arrests when resources are unavailable (Bailey *et al.* 2000). Mesocosms inoculated with *R. solani* are ideal for testing community disease ecology theory because symptoms are obvious, epidemics are fast, high replication is feasible, and environmental conditions, host heterogeneity, and inoculum amounts are relatively easy to control (e.g. Otten *et al.* 2001, 2003, 2005).

We planted mesocosms spanning a plant species richness gradient to test how communities with random and non-random species loss order, as well as communities with substitutive and additive assembly, affect disease risk. We measured disease at the community and species level to explore how disease metrics might affect conclusions. To explain variation in disease risk under different assembly patterns, we assessed the direct effects of richness, species identities, and densities on species-level disease prevalence. Our study empirically investigates how community assembly affects the diversity-disease relationship, which is important for understanding of how biodiversity loss may impact emerging diseases.

Methods

Study system

We used a model system to test how species composition affects disease risk in a greenhouse experiment. Mesocosm communities were planted and select individuals were inoculated with *R. solani* (AG2-1, Genbank accession #MZ496522). Generalist pathogens are frequently transmitted via a small subset of hosts, despite being able to infect a larger consortium of hosts. We mimicked this pattern by choosing 6 commercially available crop

plants that vary in competence. To estimate competence, we summarized intra-specific plant-plant transmission (rather than inoculum-plant transmission) by measuring disease prevalence of all uninoculated individuals (more details below). Host plants included radish (high competence), arugula (moderate), and basil, red romaine, green lettuce, and butter lettuce (low). These plants were also selected because they germinate at high rates and emerge at similar times.

Experimental design

We simulated community disassembly by creating series of mesocosm communities with 1, 2, 4, and 6 crop species (Appendix S2: Fig. S2, Fig. S3). To be consistent with typical ecological communities, depauperate assemblages contained a nested subset of their richer counterparts (Stephens *et al.* 2016) and relative species abundances followed a log-normal distribution (Roche *et al.* 2012). Within each series, the density (total number of individuals) was either positively correlated with richness ('additive assembly') or remained constant ('substitutive'). Communities with substitutive assembly contained 238 individuals and those with additive assembly consisted of 85, 154, 238, and 304 individuals. Additionally, the relationship between host competence and the order in which species were lost was either strongly positive ('non-random assembly order') or idiosyncratic ('random'). For example, the non-random disassembly treatment consisted of the same species order for each replicate—that is, high-competence radish was always present and most abundant, while low-competence butter lettuce was rarest. Meanwhile, the random order treatment had a different disassembly order for each replicated series. Using a 2x2 factorial design, we manipulated two axes of community disassembly across a richness gradient to test how patterns in species loss affect disease risk (4 richness levels x 10 replicated series = 40 mesocosms for each treatment).

Additional mesocosms were created for various reasons. To estimate host competence,

we ensured that for each species, there were at least 4—6 single-species communities consisting of 238 individuals. To estimate background damping off symptoms and germination rates, we planted one single-species mesocosm for each species at the highest density of 304 individuals. These controls were not inoculated. Our analysis of disease risk also parses effects of richness from densities of species and with the design as described above, the relative abundance of radish negatively correlates with richness. To better decouple the two variables, we augmented the design with nine trays that had higher relative densities of radish (Appendix S2: Fig. S4).

Pathogen inoculations and disease assessment

Mesocosms were planted in seedling propagation trays (25.4 x 25.4 x 6.2 cm) filled with autoclaved sand with 10% by weight fertilizer mix (in-house formula for plant growth). Over the course of three days (trays split randomly), seeds were directly sown in a hexagonal grid, dusted with 150 ml of vermiculite, and covered with a clear humidity dome. Locations of seeds were randomly assigned (Appendix S2: Fig. S3) and trays were randomized within the greenhouse. Trays were watered every 2–3 days and weighed to ensure consistent application. Plants grew under 16 h artificial light and air temperature was 23°C on average. Soil temperature nonetheless varied and on the last day, was measured in all trays with a thermometer probe.

On day three, once the majority of seedlings emerged, 12 individuals per tray were inoculated with *R. solani*. Species of challenged plants were selected proportionally to their relative abundances. Inoculum was prepared by scattering double-autoclaved poppy seeds onto 4-day-old fungal colonies grown on potato dextrose agar, and incubating at 24°C in the dark for three additional days (Otten et al. 2001). Colonized poppy seeds were individually placed under soil line 2 mm away from challenged plants. Plants were considered diseased when they exhibited aboveground symptoms, which include basal stem lesions and/or

seedling collapse. On day 18, the final disease statuses of individuals were recorded. Non-emerged plants, plants killed by herbivores, and one tray that was accidently not watered were omitted from analysis.

At the end of a preliminary trial, seedlings with and without disease symptoms (10 each for all species) were plated onto Rhizoctonia solani-selective media (water agar supplemented with chloramphenicol and benomyl; Paulitz & Schroeder 2005). Seedling crowns were surface sterilized (5% bleach 30 s, rinsed with water), plated, and visually inspected for hyphae at 24 and 48 h. All symptomatic plants produced hyphae after 24 h, confirming that disease symptoms were good proxies for *R. solani* infections. Some asymptomatic plants of radish (40%), basil (30%), arugula (10%), and green romaine (10%) produced hyphae after 48 h. Although potentially less virulent, latent or pre-symptomatic infections are possible in this study system.

Statistical analyses

Diversity-disease relationships as a function of community disassembly—To understand how the diversity-disease relationship may change under different disassembly patterns, we analyzed communities undergoing four assembly treatments: i) additive, non-random; ii) substitutive, non-random; iii) additive, random; and iv) substitutive, random. Using separate generalized linear (mixed) models (GL(M)M) for the four treatments, we analyzed disease at the community and species level to determine how disease metrics may alter conclusions.

At the community level, disease of non-challenged plants was estimated as total disease prevalence using a beta-binomial likelihood and number of diseased plants using a negative binomial likelihood. Models included an effect for richness and controlled for day planted and soil temperature. At the species level, disease prevalence of non-challenged plants was estimated for each host species using a beta-binomial likelihood. Models controlled for day

planted and temperature and included species-varying intercepts and coefficients of richness and a tray-varying intercept. Green lettuce and butter lettuce were omitted from analysis of the deterministic treatments since they were only present in the highest richness level. Extra single species trays were included in the analysis of the substitutive, random treatment since they would only improve model certainty and not bias the results.

Drivers of species-level disease prevalence—To explore drivers of variation in disease risk, we assessed effects of richness and species densities on species-level disease prevalence. Here, we evaluated disease from all mesocosm trays together (rather than the four treatments separately). With a beta-binomial likelihood, disease prevalence was estimated by modeling the number of diseased plants of each species in each tray, given the total number of non-challenged plants. Models included species-varying intercepts and coefficients of richness, terms to control for day planted and temperature, and a tray-varying intercept. We contrasted models with additional tray-level covariates, which included combinations of individual species densities and total density of all other species. Densities of individual species were square root transformed to spread the right-skewed distribution and all variables were centered and scaled by dividing by 2 SD (Gelman 2008). Predictive performance of models were compared based on the difference in expected log pointwise predictive density (ELPD) using 10-fold cross-validation (Vehtari *et al.* 2017).

Model fitting—GL(M)Ms were coded in R (R Core Team 2019) and estimated using Bayesian methods from the package `brms` (Bürkner 2017). 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 and for convergence, we ensured Rhat values were ≤ 1.01 (Vehtari *et al.* 2020). Parameter estimates with 90% highest posterior density intervals (HPDI) that did not contain zero were considered to have important, non-zero effects.

Results

A total of 36,922 seedlings in 171 mesocosms were monitored and analyzed for symptoms. Also 1,824 seedlings were grown in 6 single-species uninoculated trays. Germination rates were 95–100% and background disease symptoms were nonexistent. Host competence was estimated as disease prevalence in monospecific trays for the following species: radish (mean, SD = 0.94 [0.08]), arugula (0.33 [0.27]), basil (0.03 [0.02]), red romaine (0.02 [0.02]), green lettuce (0.006 [0.005]), and butter lettuce (0.02 [0.006]).

Diversity-disease relationships as a function of community disassembly

With additive assembly, community disease prevalence positively correlated with richness, regardless of whether species loss order was non-random or random (Fig. 3.1a, 3.1c). With substitutive assembly and random species loss, richness likely had an unimportant association (Fig. 3.1d). Only with substitutive and non-random disassembly did richness have a strongly negative effect on community disease prevalence (Fig. 3.1b). Results were qualitatively identical for density of diseased plants (Appendix S2: Fig. S5).

When disease prevalence was evaluated for particular host species, effects of richness were strongly positive with additive assembly (Fig. 3.2a, 3.2c). For communities with substitutive, non-random disassembly, the 90% HPDI of the coefficient for richness was entirely negative for all species, except red romaine where the interval was [-1.87, 0.09] (Fig. 3.2b). For communities with substitutive, random disassembly, the effects of richness were negative for radish, positive for green lettuce, and negligible for all other species (Fig. 3.2d).

Drivers of species-level disease prevalence

After controlling for soil temperature, which had a negative effect, and planting day, the importance of richness on species-level disease prevalence varied depending on which

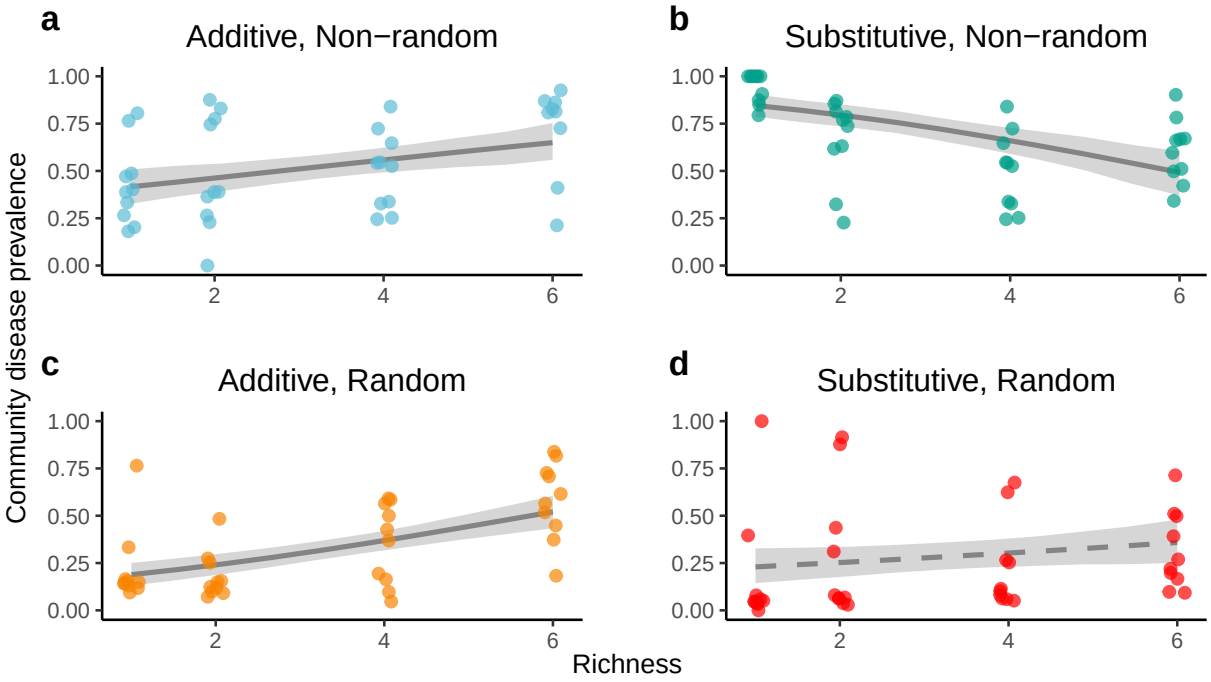

Figure 3.1: Relationships between richness and community-level disease prevalence for different community assembly patterns. Lines and shaded regions represent the median and 90% HPDI of the posterior estimate of the mean. Solid lines indicate the 90% HPDI of the coefficient of richness did not contain zero.

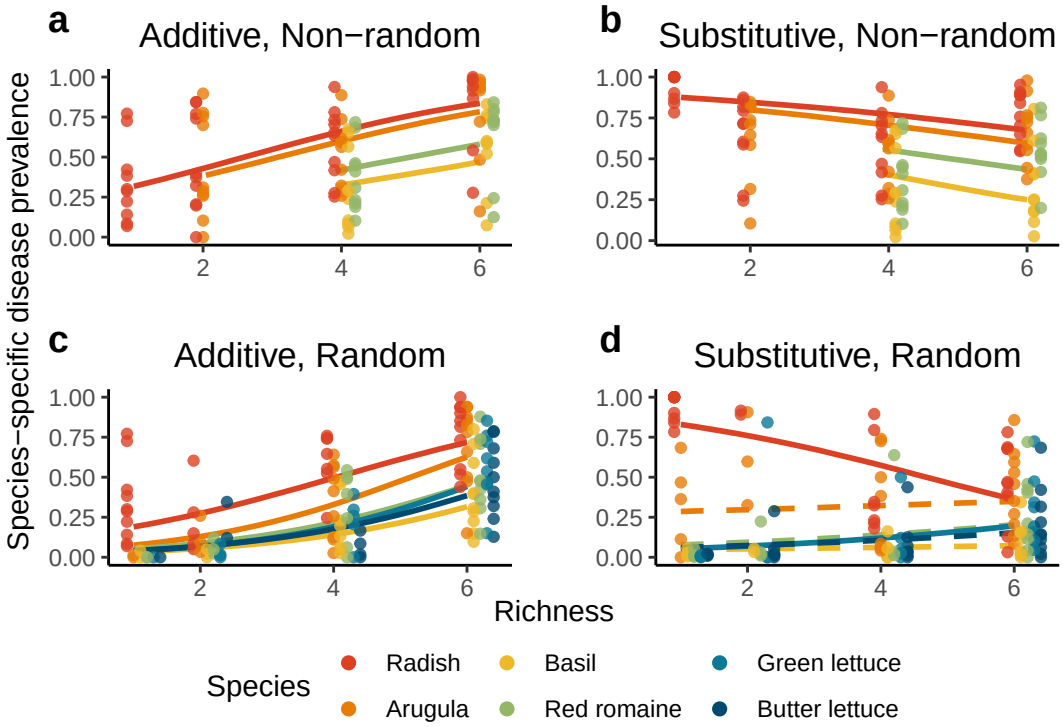

Figure 3.2: Relationships between richness and species-level disease prevalence for different community assembly patterns. Solid lines indicate the 90% HPDI of the coefficient of richness did not contain zero.

other tray-level variables were accounted for (Appendix S2: Table S2). Richness positively covaried with disease risk in the model incorporating total density, which also had a strong positive effect. Yet, the importance of richness weakened when information on the densities of specific species was included. In the model including densities of each species, the mean coefficient of richness was likely unimportant (median log-odds, 90% HPDI =0.46 [-0.19, 1.17]), densities of radish (3.63 [2.94, 4.45]) and arugula (0.85 [0.29, 1.4]) had strongly positive effects, while densities of other species had weak effects. As long as radish density was explicitly modeled and densities of other species were included (see Appendix S2 for details), predictive performance was equivalent and superior to the model incorporating total density only (ΔELPD, SE = -52.2, 10.6).

Discussion

Theory predicts that whether species loss enhances or suppresses disease risk depends on community assembly (Joseph *et al.* 2013). We empirically contrasted the relationship between richness and various disease metrics in communities with different assembly patterns. Whether disease was measured for focal species or the overall host community, our results closely aligned with expectations. Richness limited disease when species loss order negatively correlated with competence ('non-random disassembly') and overall species density remained invariant with richness ('substitutive'). In communities with additive assembly or those with substitutive and random disassembly, richness was positively or weakly associated with disease, apart from a few deviations. The effect of richness on species-level disease prevalence was best explained by variation in densities and identities of species, highlighting the consequential impact of compositional shifts on future outbreaks.

In communities with substitutive, non-random assembly, species-level disease risk was likely lower in richer communities because there were fewer competent host plants, such as radish and arugula. 'Competent host regulation' (modified *sensu* Keesing *et al.* 2006), which

suggests that a lower density of competent hosts leads to lower transmission risk, is often evoked as a mechanism underlying dilution effects (Johnson *et al.* 2012; Strauss *et al.* 2015). Species-level disease prevalence across all trays was positively associated with richness when density of all species was incorporated into the model; however, the marginal effect of richness was negligible after also conditioning upon the densities of individual species (Appendix S2: Table S2). Densities and identities of species are key for explaining variation in disease risk, and at least in our case, can primarily account for strong richness effects.

For communities with substitutive, random assembly, correlations between richness and species-level disease risk were weak for most species, which is consistent with simulation models measuring overall transmission risk (Joseph *et al.* 2013). Somewhat unexpectedly, radish disease prevalence increased with species loss. The dilution effect for radish is attributed to its strong positive effect on disease (*c.a.* 16 times stronger than arugula) and to its density negatively covarying with richness. Although densities of host species had an overall random association with richness, for trays containing a given species, density of that species underwent compensatory declines with increasing richness. For example, when radish was present in species-poor assemblages, its density was high and in species-rich assemblages, its density was low. This is an outcome of community nestedness and substitutive assembly. We speculate that irrespective of how competence relates to extirpation risk, strong community nestedness and substitutive assembly can increase the odds of a dilution effect for high competence hosts.

Regardless of species loss order, additive assembly produced positive correlations between richness and species-level disease. Since densities of highly important hosts, radish and arugula, did not increase during disassembly and species-poor communities always contained fewer individuals, depauperate communities fostered less disease. While disease severity on individuals can still be lowest in high-diversity communities despite additive assembly (Johnson *et al.* 2013), this likely represent a special case in which transmission is heavily reduced by direct interactions with nonhosts. We did not detect disease-limiting effects from richness

per se and thus, our findings are consistent with simulation models that assume transmission is unaffected by species interactions (Rudolf & Antonovics 2005; Mihaljevic *et al.* 2014).

Diversity-disease relationships were largely insensitive to whether disease was measured at the species or community level (recorded as absolute or relative density of diseased plants). Our findings from substitutive communities—that non-random species loss increased community-wide disease risk and random species loss had little effect—were consistent with experimental studies measuring total infectious propagules (Johnson *et al.* 2019) or community-averaged disease severity (Liu *et al.* 2018). Results from communities with additive assembly, which led to positive associations between disease and richness, further suggest that species loss order has minimal effects on community-level disease when densities of individual species are maintained.

What do results from our experiment, which tested hypothetical endpoints of community disassembly, portend for natural communities? Substitutive and additive assembly might arise when species niches either perfectly overlap or partition, respectively, but a saturating relationship between richness and density may be more realistic. Saturating host abundance should lead to disease risk rising, then falling across a richness gradient (Mihaljevic et al. 2014). Despite its strong influence on disease dynamics, few studies have examined the relationship between overall host density and diversity in natural ecosystems (but see Guo *et al.* 2006, Rosenthal et al. in review) and remains an important topic. Likewise, the strength of the competence-extirpation risk relationship can transform a negative diversity-disease relationship to one that is idiosyncratic. Although disease-prone species tend to be more resistant to perturbations (Gibb *et al.* 2020), extirpations are subject to environmental and demographic stochasticity. Additional stochasticity may weaken any negative extirpation risk-competence associations, add noise to community nestedness, and introduce more uncertainty in diversity-disease patterns. Finally, although various disease metrics did not lead to qualitatively different associations with richness, it is critical to consider what epidemiological information can be gleaned from each metric and its applications to disease

management goals.

Overall, our experimental study confirmed many theoretical predictions outlining effects of community assembly on disease risk. It is necessary to continue gathering information on how natural communities will likely (dis)assemble, which involves examining relationships among diversity, species densities, host competence and susceptibility, and likelihoods of extirpation. Through a combination of models, manipulative experimental systems, and surveys of natural communities, we can advance our understanding of how community assembly will impact disease, identify which species loss patterns are most likely to occur under various disturbances pressures, and devise actionable plans to mitigate emerging outbreaks.

Acknowledgements

We thank Orchard Park Greenhouse staff and facility for assistance and research access; S. Rosenthal and L. Ng for planting materials; C. Todd, I. Todd, C. Engert, S. Fajardo, and K. Frangioso for planting assistance; M. Cubeta for advice on R. solani; F. Keesing, A. Latimer, and G. Gilbert for feedback. Research supplies were funded by a Jastro-Shields and Shields grant to LMR, UC Agricultural Experiment Station funding to DMR. Additional support to LMR was provided by an NSF Graduate Research Fellowship.

Bailey, D.J., Otten, W. & Gilligan, C.A. (2000). Saprotrophic invasion by the soilborne fungal plant pathogen Rhizoctonia solani and percolation thresholds. *New Phytol.*, 146, 535–544.

Bürkner, P.-C. (2017). **brms**: An *R* Package for Bayesian Multilevel Models Using *Stan*. *J. Stat. Softw.*, 80.

Cronin, J.P., Welsh, M.E., Dekkers, M.G., Abercrombie, S.T. & Mitchell, C.E. (2010). Host physiological phenotype explains pathogen reservoir potential: epidemiology and host physiology. *Ecol. Lett.*, 13, 1221–1232.

Fukami, T., Martijn Bezemer, T., Mortimer, S.R. & Putten, W.H. (2005). Species divergence and trait convergence in experimental plant community assembly. *Ecol. Lett.*, 8, 1283–1290.

Gelman, A. (2008). Scaling regression inputs by dividing by two standard deviations. *Stat. Med.*, 27, 2865–2873.

Gibb, R., Redding, D.W., Chin, K.Q., Donnelly, C.A., Blackburn, T.M. & Newbold, T. *et al.* (2020). Zoonotic host diversity increases in human-dominated ecosystems. *Nature*, 584, 398–402.

Guo, Q., Shaffer, T. & Buhl, T. (2006). Community maturity, species saturation and the variant diversity?productivity relationships in grasslands. *Ecol. Lett.*, 9, 1284–1292.

Haas, S.E., Hooten, M.B., Rizzo, D.M. & Meentemeyer, R.K. (2011). Forest species diversity reduces disease risk in a generalist plant pathogen invasion. *Ecol. Lett.*, 14, 1108–1116.

Halliday, F.W., Heckman, R.W., Wilfahrt, P.A. & Mitchell, C.E. (2019). Past is prologue: host community assembly and the risk of infectious disease over time. *Ecol. Lett.*, 22, 138–148.

Heckman, R.W., Halliday, F.W. & Mitchell, C.E. (2019). A growth–defense trade-off is general across native and exotic grasses. *Oecologia*, 191, 609–620.

Johnson, P.T.J., Calhoun, D.M., Riepe, T., McDevitt-Galles, T. & Koprivnikar, J. (2019). Community disassembly and disease: realistic—but not randomized—biodiversity losses enhance parasite transmission. *Proc. R. Soc. B.*, 286, 20190260.

Johnson, P.T.J., Ostfeld, R.S. & Keesing, F. (2015). Frontiers in research on biodiversity and disease. *Ecol. Lett.*, 18, 1119–1133.

Johnson, P.T.J., Preston, D.L., Hoverman, J.T., Henderson, J.S., Paull, S.H. & Richgels, K.L.D. *et al.* (2012). Species diversity reduces parasite infection through

cross-generational effects on host abundance. *Ecology*, 93, 56–64.

Johnson, P.T.J., Preston, D.L., Hoverman, J.T. & Richgels, K.L.D. (2013). Biodiversity decreases disease through predictable changes in host community competence. *Nature*, 494, 230–233.

Joseph, M.B., Mihaljevic, J.R., Orlofske, S.A. & Paull, S.H. (2013). Does life history mediate changing disease risk when communities disassemble? *Ecol. Lett.*, 16, 1405–1412.

Keesing, F., Holt, R.D. & Ostfeld, R.S. (2006). Effects of species diversity on disease risk. *Ecol. Lett.*, 9, 485–498.

Lacroix, C., Jolles, A., Seabloom, E.W., Power, A.G., Mitchell, C.E. & Borer, E.T. (2014). Non-random biodiversity loss underlies predictable increases in viral disease prevalence. *J. R. Soc. Interface*, 11, 20130947.

Liu, X., Chen, F., Lyu, S., Sun, D. & Zhou, S. (2018). Random species loss underestimates dilution effects of host diversity on foliar fungal diseases under fertilization. *Ecol. Evol.*, 8, 1705–1713.

Mihaljevic, J.R., Joseph, M.B., Orlofske, S.A. & Paull, S.H. (2014). The scaling of host density with richness affects the direction, shape, and detectability of diversity-disease relationships. *PLoS One*, 9, e97812.

Mitchell, C.E., Tilman, D. & Groth, J.V. (2002). Effects of grassland plant species diversity, abundance, and composition on foliar fungal disease. *Ecology*, 83, 713–1726.

Ostfeld, R.S. & LoGiudice, K. (2003). COMMUNITY DISASSEMBLY, BIODIVERSITY LOSS, AND THE EROSION OF AN ECOSYSTEM SERVICE. *Ecology*, 84, 1421–1427.

Otten, W., Filipe, J.A.N., Bailey, D.J. & Gilligan, C.A. (2003). QUANTIFICATION AND ANALYSIS OF TRANSMISSION RATES FOR SOILBORNE EPIDEMICS. *Ecology*, 84, 3232–3239.

Otten, W., Filipe, J.A.N. & Gilligan, C.A. (2005). DAMPING-OFF EPIDEMICS, CONTACT STRUCTURE, AND DISEASE TRANSMISSION IN MIXED-SPECIES POPULATIONS. *Ecology*, 86, 1948–1957.

Otten, W., Hall, D., Harris, K., Ritz, K., Young, I.M. & Gilligan, C.A. (2001). Soil BlackwellScienceLtd physics, fungal epidemiology and the spread of. *New Phytol.*, 10.

Paulitz, T.C. & Schroeder, K.L. (2005). A New Method for the Quantification of *Rhizoctonia solani* and *R. oryzae* from Soil. *Plant Dis.*, 89, 767–772.

R Core Team. (2019). R: A Language and Environment for Statistical Computing.

Roche, B., Dobson, A.P., Guégan, J.-F. & Rohani, P. (2012). Linking community and disease ecology: the impact of biodiversity on pathogen transmission. *Philos. Trans. R. Soc. Lond. B Biol. Sci.*, 367, 2807–2813.

Rudolf, V.H.W. & Antonovics, J. (2005). Species coexistence and pathogens with frequency-dependent transmission. *Am. Nat.*, 166, 112–118.

Searle, C.L., Cortez, M.H., Hunsberger, K.K., Grippi, D.C., Oleksy, I.A. & Shaw, C.L. *et al.* (2016). Population density, not host competence, drives patterns of disease in an invaded community. *Am. Nat.*, 188, 554–566.

Stephens, P.R., Altizer, S., Smith, K.F., Alonso Aguirre, A., Brown, J.H. & Budischak, S.A. *et al.* (2016). The macroecology of infectious diseases: a new perspective on global-scale drivers of pathogen distributions and impacts. *Ecol. Lett.*, 19, 1159–1171.

Strauss, A.T., Civitello, D.J., Cáceres, C.E. & Hall, S.R. (2015). Success, failure and ambiguity of the dilution effect among competitors. *Ecol. Lett.*, 18, 916–926.

Vehtari, A., Gelman, A. & Gabry, J. (2017). Practical Bayesian model evaluation using leave-one-out cross-validation and WAIC. *Stat. Comput.*, 27, 1413–1432.

Vehtari, A., Gelman, A., Simpson, D., Carpenter, B. & Bürkner, P.-C. (2020). Rank-Normalization, Folding, and Localization: An Improved $\widehat{\{R\}}$ for Assessing Convergence of

MCMC. *Bayesian Anal.*

Wojdak, J.M., Edman, R.M., Wyderko, J.A., Zemmer, S.A. & Belden, L.K. (2014). Host Density and Competency Determine the Effects of Host Diversity on Trematode Parasite Infection. *PLoS One*, 9, 10.

Young, H.S., Dirzo, R., Helgen, K.M., McCauley, D.J., Billeter, S.A. & Kosoy, M.Y. *et al.* (2014). Declines in large wildlife increase landscape-level prevalence of rodent-borne disease in Africa. *Proc. Natl. Acad. Sci. U.S.A.*, 111, 7036–7041.