Received: 1 September 2021 | Revised: 12 November 2021 | Accepted: 9 December 2021

DOI: 10.1002/ecy.3622

REPORTS

ECOLOGY
ECOLOGICAL SOCIETY OF AMERICA

Species densities, assembly order, and competence jointly determine the diversity–disease relationship

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

[1]Department of Plant Pathology, University of California, Davis, California, USA

[2]Graduate Group in Ecology, University of California, Davis, California, USA

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

Correspondence
Lisa M. Rosenthal
Email: lrosenthal@usgs.gov

Funding information
Jastro Shields; NSF Graduate Research Fellowship, Grant/Award Number: 1650042; UC Agricultural Experiment Station award

Handling Editor: David J. Civitello

Abstract

Since species vary in abundance and host competence (i.e., ability to get infected and transmit a pathogen), changes in species composition caused by biodiversity loss impacts disease dynamics. Forecasting effects of species composition on disease depends on community (dis)assembly, processes determining how species are added to (or lost from) communities. We simulated community assembly by planting mesocosms, nested along a richness gradient, and tested how relationships between richness, species assembly order, and overall density affect disease risk. Mesocosms with up to six 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 assembly order negatively correlated with competence and total density remained unchanged with richness. When density increased with richness or species assembled randomly, richness primarily correlated positively or weakly with disease. Our results align with theoretical expectations and represent the first empirical study to test the influence of species densities, assembly order, and competence on diversity–disease relationships.

KEYWORDS

community assembly, dilution effect, mesocosm, plant pathogen, *Rhizoctonia solani*

INTRODUCTION

Communities undergoing or recovering from biodiversity loss following disturbance experience shifts in composition that can differentially impact infectious diseases (Halliday et al., 2020; Johnson et al., 2013). Since species vary in their ability to become infected and transmit a pathogen (i.e., "host competence"), processes determining which species and how many are added to (or lost from) communities, hereafter defined as community (dis)assembly, can influence associations between disease risk and biodiversity loss (Halliday et al., 2019, 2020; Joseph et al., 2013). Understanding how species loss influences disease dynamics is paramount for predicting and managing future outbreaks (Rohr et al., 2020); addressing the role of community assembly may help identify conditions underlying diversity–disease relationships in natural ecosystems.

There is an increased likelihood that diversity and overall transmission risk negatively covary, a phenomenon known as the "dilution effect" (Keesing et al., 2006), when highly competent host species are least likely to be extirpated (Ostfeld & LoGiudice, 2003). Negative relationships between competence and extirpation risk may be attributed to life history trade-offs. 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 assembly (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 (Joseph et al., 2013; Ostfeld & LoGiudice, 2003).

Even when competence negatively correlates with extirpation risk, changes in disease also depend on how overall density covaries with species loss (Searle et al., 2016). At one extreme, total community abundance may remain invariant with richness. Under this "substitutive assembly," diversity loss may release competent host species from strong resource limitations, leading to more competent hosts, and therefore, higher disease risk in species-poor communities (Mihaljevic et al., 2014; Rudolf & Antonovics, 2005). Conversely, densities of individual species might not covary with richness, such that overall community abundance positively correlates with diversity ("additive assembly"). Total density would be greatest in diverse communities and for generalist pathogens with density-dependent transmission, disease risk should increase with diversity (Mihaljevic et al., 2014; Rudolf & Antonovics, 2005). Altogether, it is expected that a dilution effect is most likely when community assembly is substitutive and extirpation risk negatively correlates with competence (Johnson et al., 2015; Joseph et al., 2013).

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 identify how species composition varies across richness (Johnson et al., 2013; Lacroix et al., 2014; Rosenthal et al., 2021), which helps predict how communities might disassemble. Nevertheless, the effects of different assembly patterns on disease risk may be best addressed in experiments where species composition is manipulated independently of diversity. To date, experimental studies on the dilution effect have compared random versus nonrandom species loss (Johnson et al., 2019; Liu et al., 2018) 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 is 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 (Young et al., 2014) or average community disease severity or prevalence (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., 2021).

Here we used artificial plant mesocosms to examine effects of species identities and densities on disease caused by a generalist fungal pathogen, *Rhizoctonia solani*. This soil-borne pathogen is primarily of agricultural concern of seedlings. *R. solani* infects near the soil line, leading to a seedling's collapse and typically death (Otten et al., 2003). In close proximity (<3 cm), transmission occurs between neighboring plants within a few days through infective hyphae and slows or arrests when resources are unavailable (Bailey et al., 2000). Mesocosms inoculated with *R. solani* are ideal for testing disease ecology theory because symptoms are conspicuous, epidemics are fast, high replication is feasible, and environmental conditions, host heterogeneity, and inoculum amounts are relatively easy to control (Otten et al., 2001, 2003, 2005).

We planted mesocosms spanning a plant species richness gradient to test how communities with random and nonrandom 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

Mesocosm communities grew in a greenhouse and were inoculated with *R. solani* AG2-1 (Genbank accession #MZ496522), which is often associated with cruciferous plants. Since *R. solani* can lose virulence in storage, we ensured pathogenicity by reisolating it from an inoculated arugula plant (*Eruca vesicaria* spp. *Sativa*). We used six crop plants that varied in competence. To estimate competence, we summarized intraspecific plant–plant transmission by measuring disease prevalence of uninoculated individuals (Appendix S1). Host plants included a high-competence host, radish (*Raphanus sativus*); a moderate-competence host, arugula; and low-competence hosts, basil

(*Ocimum basilicum*) and three varieties of lettuce (*Lactuca sativa*; red, green, and butter).

Experimental design

We simulated community assembly by creating series of mesocosms consisting of four richness levels (1, 2, 4, 6) and used a factorial design to explore treatments of species assembly order (random/nonrandom) and density (substitutive/additive), replicated 10 times (40 trays per treatment; see Appendix S1 and Appendix S2: Figures S1 and S2). Species-poor communities were nested subsets of their richer counterparts (Graham et al., 2009) and relative abundances followed a log-normal distribution (Roche et al., 2012). For each series, density positively correlated with richness (additive; 85, 154, 238, and 304 total plants) or remained at a constant moderate density (substitutive; 238 plants). Also, the relationship between host competence and species order was strongly negative (nonrandom) or idiosyncratic (random). For example, the nonrandom treatment consisted of the same order for each replicate: high-competence radish was always present and most abundant, while low-competence butter lettuce was rarest. The random treatment had a different species order for each replicate. We added mesocosms to estimate host competence, background disease symptoms, and germination rates (see Appendix S1).

Pathogen inoculations and disease assessment

Mesocosms consisted of trays (25.4 × 25.4 × 6.2 cm) filled with autoclaved sand fertilized with 10% by weight Unified Formula (UC Davis College of Agricultural and Environmental Sciences). Over three days, seeds were sown randomly in a hexagonal grid (Appendix S2: Figure S2), dusted with vermiculite, and covered with a clear dome. Trays were randomized in the greenhouse. Trays were watered every 2–3 days and weighed for 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 3, once most seedlings emerged, 12 individuals per tray were inoculated with *R. solani* (see Appendix S1). Challenged plants were randomly located and stratified by species according to relative abundances. On day 18, the final disease statuses of individuals were recorded. Plants were considered to be diseased when they exhibited damping-off symptoms, which include basal stem lesions and/or collapse. In preliminary trials (Appendix S1), we determined that disease symptoms were good proxies for *R. solani* infections, but latent or pre-symptomatic infections were possible in this system. Non-emerged plants or those killed by herbivores (1.5% of plants), and one tray that was accidently not watered were omitted from analysis.

Statistical analyses

Diversity–disease relationships as a function of community assembly

To understand how the dilution effect may change under different assembly patterns, we analyzed species-level disease risk in communities undergoing four treatments: (1) additive, nonrandom; (2) substitutive, nonrandom; (3) additive, random; and (4) substitutive, random. We also analyzed disease at the community level to determine how the choice of disease metric may alter conclusions. Separate generalized linear (mixed) models (GL[M]M) were used for the four treatments.

At the species level, disease prevalence of non-challenged plants was estimated as the number of infected plants given the total number of plants using a beta-binomial likelihood. Models controlled for day planted and temperature and included a tray-varying intercept. Also, intercepts and coefficients of richness varied by species. Although distance to challenged plants increases individual-level risk to infection, we did not incorporate a spatial term in our species-level models because no spatial patterns were associated with species (Appendix S1).

At the community level, disease of non-challenged plants was estimated as total prevalence using a beta-binomial likelihood and number of diseased using a negative binomial likelihood. Models included a coefficient for richness and controlled for day planted and soil temperature. Extra details on the models are in Appendix S1.

Drivers of species-level disease prevalence

To explore drivers of variation in disease risk, we estimated coefficients of richness and species densities on species-level disease prevalence. Here, we evaluated disease from all inoculated mesocosm trays together (see Appendix S1). Disease prevalence of non-challenged plants was modeled with a beta-binomial likelihood. 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. 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 (v4.1.1, R Core Team, 2021) and estimated using Bayesian methods from the package brms (Bürkner, 2017). We used weakly informative priors and four 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., 2021). Parameter estimates with 90% highest posterior density intervals (HPDI) that did not contain zero were considered to have important, non-zero effects. All R packages used in this analysis are detailed in Appendix S1.

RESULTS

A total of 32,926 non-challenged seedlings in 162 inoculated mesocosms were analyzed for symptoms. Also 1824 seedlings were grown in six 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 assembly

With additive assembly, community disease prevalence positively correlated with richness, regardless of whether species loss order was nonrandom or random (Figure 1a,c). With substitutive assembly and random species loss, richness likely had an unimportant association (Figure 1d). Only with substitutive and nonrandom assembly did richness have a strongly negative effect on community disease prevalence (Figure 1b). Results were qualitatively identical for density of diseased plants (Appendix S2: Figure S3).

When disease prevalence was evaluated for particular host species, effects of richness were strongly positive with additive assembly (Figure 2a,c). For communities with substitutive, nonrandom assembly, the 90% HPDI of

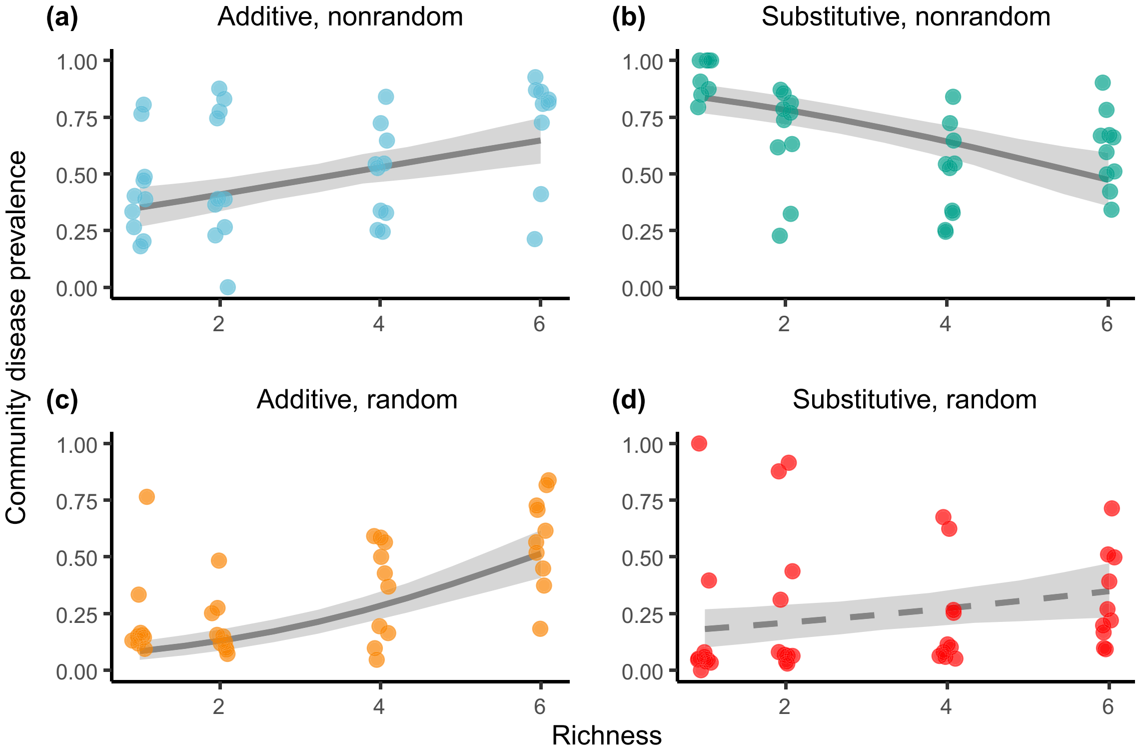

FIGURE 1 Relationships between richness and community-level disease prevalence for different community assembly patterns. Points are horizontally jittered. Lines and shaded regions represent the median and 90% highest posterior density intervals (HPDI) of the posterior estimate of the mean. Solid lines indicate that the 90% HPDI of the coefficient of richness did not contain zero

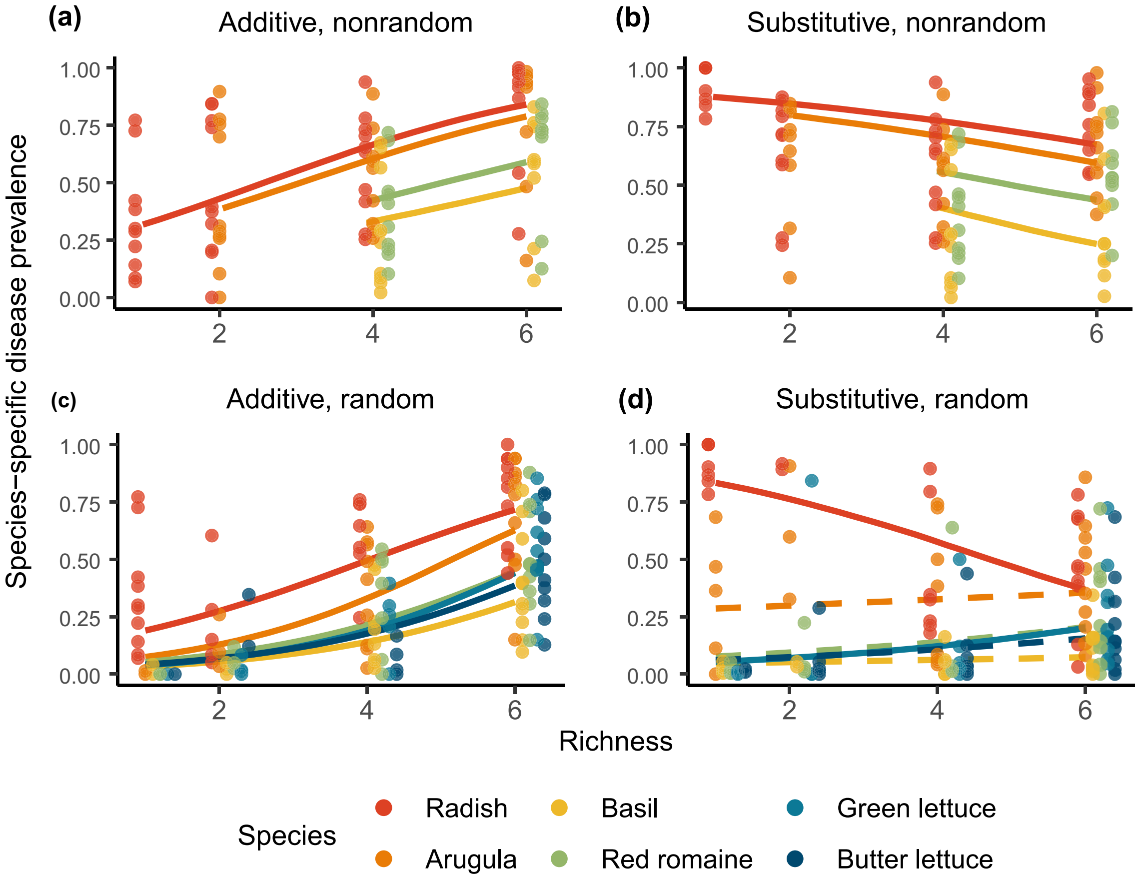

FIGURE 2 Relationships between richness and species-level disease prevalence for different community assembly patterns. Points are horizontally jittered. Solid lines indicate that the 90% highest posterior density intervals (HPDI) of the species-specific coefficient of richness did not contain zero

the coefficient for richness was entirely negative for all species, except red romaine where the interval was [−1.87, 0.09] (Figure 2b). For communities with substitutive, random assembly, the effects of richness were negative for radish, positive for green lettuce, and negligible for all other species (Figure 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 other tray-level variables were accounted for (Appendix S2: Table S1). 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] = −0.15 [−1.36, 0.99]), densities of radish (3.39 [2.71, 4.07]) and arugula (0.81 [0.31, 1.41]) had strongly positive effects, and densities of other species had weak effects. As long as explanatory variables explicitly included radish density and densities of other species (see Appendix S2: Table S1), predictive performance was equivalent and superior to the model incorporating total density only (ΔELPD, SE = −53.2, 8.2).

DISCUSSION

Theory predicts that variation in disease risk is affected by how community composition changes as species are lost (Joseph et al., 2013; Ostfeld & LoGiudice, 2003). 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 order negatively correlated with competence (nonrandom) and overall species density remained invariant with richness (substitutive). In communities with additive assembly or those with substitutive and random assembly, 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, nonrandom assembly, species-level disease risk was lower in richer communities because there were fewer competent hosts, such as radish and arugula. “Competent host regulation” (modified sensu Keesing et al., 2006) suggests that fewer competent hosts lead to lower transmission risk, and is often evoked to explain 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 S1). Densities and identities of species primarily accounted for strong richness effects and are key for explaining variation in disease risk.

For communities with substitutive, random assembly, correlations between richness and species-level disease risk were weak for most species, which is consistent with models measuring 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 (∼13 times stronger than arugula) and to its density negatively covarying with richness. Even though densities of host species had an overall random association with richness, for trays containing a given species, that species’ density would negatively covary with richness (Johnson et al., 2019; Rottstock et al., 2014). This is an outcome of substitutive assembly. We speculate that regardless of how competence relates to extirpation risk, substitutive assembly may increase the odds of a dilution effect for high competence hosts.

Regardless of species order, additive assembly produced positive correlations between richness and species-level disease. Since densities of 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 kind of result likely represents 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 (Mihaljevic et al., 2014; Rudolf & Antonovics, 2005).

Diversity–disease relationships measured with species-level disease risk largely agreed with those using community-level metrics, recorded as absolute or relative density of diseased plants. Our findings from substitutive communities, that nonrandom species loss increased community-wide disease and random species loss had little effect, were consistent with 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, suggest that species 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 assembly, 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). Few studies have examined the relationship between overall host density and diversity in natural ecosystems (but see Guo et al., 2006; Rosenthal et al., 2021). Likewise, the strength of the competence–extirpation-risk relationship can transform a negative diversity–disease relationship to an idiosyncratic relationship (Joseph et al., 2013). 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 competence-extirpation risk associations, add noise to community nestedness, and introduce more uncertainty in diversity–disease patterns. Finally, although various disease metrics did not qualitatively alter 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.

ACKNOWLEDGMENTS

We thank Orchard Park Greenhouse and staff for research access; S. Rosenthal and L. Ng for 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, G. Gilbert, two reviewers, and the editor for feedback. Supplies were funded by a Jastro-Shields grant and UC Agricultural Experiment Station. Support to L. M. Rosenthal was provided by an NSF Graduate Research Fellowship.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data and code (Rosenthal, 2021) are available in Zenodo: https://doi.org/10.5281/zenodo.5774258.

ORCID

Lisa M. Rosenthal https://orcid.org/0000-0003-4030-7587

REFERENCES

Bailey, D.J., W. Otten, and C.A. Gilligan. 2000. “Saprotrophic Invasion by the Soil-Borne Fungal Plant Pathogen *Rhizoctonia Solani* and Percolation Thresholds.” *New Phytologist* 146: 535–44.

Bürkner, P.-C. 2017. “brms: An *R* Package for Bayesian Multilevel Models Using *Stan*.” *Journal of Statistical Software* 80: 1–28.

Cronin, J.P., M.E. Welsh, M.G. Dekkers, S.T. Abercrombie, and C. E. Mitchell. 2010. “Host Physiological Phenotype Explains Pathogen Reservoir Potential: Epidemiology and Host Physiology.” *Ecology Letters* 13: 1221–32.

Fukami, T., T. Martijn Bezemer, S.R. Mortimer, and W.H. Putten. 2005. “Species Divergence and Trait Convergence in Experimental Plant Community Assembly.” *Ecology Letters* 8: 1283–90.

Gibb, R., D.W. Redding, K.Q. Chin, C.A. Donnelly, T.M. Blackburn, T. Newbold, and K.E. Jones. 2020. “Zoonotic Host Diversity Increases in Human-Dominated Ecosystems.” *Nature* 584: 398–402.

Graham, S.P., H.K. Hassan, N.D. Burkett-Cadena, C. Guyer, and T. R. Unnasch. 2009. “Nestedness of Ectoparasite-Vertebrate Host Networks.” *PLoS One* 4: e7873.

Guo, Q., T. Shaffer, and T. Buhl. 2006. “Community Maturity, Species Saturation and the Variant Diversity-Productivity Relationships in Grasslands.” *Ecology Letters* 9: 1284–92.

Haas, S.E., M.B. Hooten, D.M. Rizzo, and R.K. Meentemeyer. 2011. “Forest Species Diversity Reduces Disease Risk in a Generalist Plant Pathogen Invasion.” *Ecology Letters* 14: 1108–16.

Halliday, F.W., R.W. Heckman, P.A. Wilfahrt, and C.E. Mitchell. 2019. “Past Is Prologue: Host Community Assembly and the Risk of Infectious Disease over Time.” *Ecology Letters* 22: 138–48.

Halliday, F.W., J.R. Rohr, and A.-L. Laine. 2020. “Biodiversity Loss Underlies the Dilution Effect of Biodiversity.” *Ecology Letters* 23: 1611–22.

Heckman, R.W., F.W. Halliday, and C.E. Mitchell. 2019. “A Growth–Defense Trade-off Is General across Native and Exotic Grasses.” *Oecologia* 191: 609–20.

Johnson, P.T.J., D.M. Calhoun, T. Riepe, T. McDevitt-Galles, and J. Koprivnikar. 2019. “Community Disassembly and Disease: Realistic-but Not Randomized-Biodiversity Losses Enhance Parasite Transmission.” *Proceedings of the Royal Society B* 286: 20190260.

Johnson, P.T.J., R.S. Ostfeld, and F. Keesing. 2015. “Frontiers in Research on Biodiversity and Disease.” *Ecology Letters* 18: 1119–33.

Johnson, P.T.J., D.L. Preston, J.T. Hoverman, J.S. Henderson, S.H. Paull, K.L.D. Richgels, and M.D. Redmond. 2012. “Species Diversity Reduces Parasite Infection through Cross-Generational Effects on Host Abundance.” *Ecology* 93: 56–64.

Johnson, P.T.J., D.L. Preston, J.T. Hoverman, and K.L.D. Richgels. 2013. “Biodiversity Decreases Disease through Predictable Changes in Host Community Competence.” *Nature* 494: 230–3.

Joseph, M.B., J.R. Mihaljevic, S.A. Orlofske, and S.H. Paull. 2013. “Does Life History Mediate Changing Disease Risk when Communities Disassemble?” *Ecology Letters* 16: 1405–12.

Keesing, F., R.D. Holt, and R.S. Ostfeld. 2006. “Effects of Species Diversity on Disease Risk.” *Ecology Letters* 9: 485–98.

Lacroix, C., A. Jolles, E.W. Seabloom, A.G. Power, C.E. Mitchell, and E.T. Borer. 2014. “Non-random Biodiversity Loss Underlies Predictable Increases in Viral Disease Prevalence.” *Journal of the Royal Society, Interface / the Royal Society* 11: 20130947.

Liu, X., F. Chen, S. Lyu, D. Sun, and S. Zhou. 2018. “Random Species Loss Underestimates Dilution Effects of Host Diversity on Foliar Fungal Diseases under Fertilization.” *Ecology and Evolution* 8: 1705–13.

Mihaljevic, J.R., M.B. Joseph, S.A. Orlofske, and S.H. Paull. 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., D. Tilman, and J.V. Groth. 2002. “Effects of Grassland Plant Species Diversity, Abundance, and Composition on Foliar Fungal Disease.” *Ecology* 83: 713–1726.

Ostfeld, R.S., and K. LoGiudice. 2003. “Community Disassembly, Biodiversity Loss, and the Erosion of an Ecosystem Service.” *Ecology* 84: 1421–7.

Otten, W., J.A.N. Filipe, D.J. Bailey, and C.A. Gilligan. 2003. “Quantification and Analysis of Transmission Rates for Soil-borne Epidemics.” *Ecology* 84: 3232–9.

Otten, W., J.A.N. Filipe, and C.A. Gilligan. 2005. “Damping-off Epidemics, Contact Structure, and Disease Transmission in Mixed-Species Populations.” *Ecology* 86: 1948–57.

Otten, W., D. Hall, K. Harris, K. Ritz, I.M. Young, and C.A. Gilligan. 2001. “Soil Physics, Fungal Epidemiology and the Spread of *Rhizoctonia solani*.” *New Phytologist* 151: 459–68.

R Core Team. 2021. *R: A Language and Environment for Statistical Computing*. Vienna: R Foundation for Statistical Computing.

Roche, B., A.P. Dobson, J.-F. Guégan, and P. Rohani. 2012. “Linking Community and Disease Ecology: The Impact of

Biodiversity on Pathogen Transmission." *Philosophical Transactions of the Royal Society B* 367: 2807–13.

Rohr, J.R., D.J. Civitello, F.W. Halliday, P.J. Hudson, K.D. Lafferty, C.L. Wood, and E.A. Mordecai. 2020. "Towards Common Ground in the Biodiversity–Disease Debate." *Nature Ecology & Evolution* 4: 24–33.

Rosenthal, L. 2021. "lisamr/mesocosm2021_public: accepted_Ecology (v3.0)." *Zenodo*. https://doi.org/10.5281/zenodo.5774258

Rosenthal, L.M., A.B. Simler-Williamson, and D.M. Rizzo. 2021. "Community-Level Prevalence of a Forest Pathogen, Not Individual-Level Disease Risk, Declines with Tree Diversity." *Ecology Letters* 24: 2477–89.

Rottstock, T., J. Joshi, V. Kummer, and M. Fischer. 2014. "Higher Plant Diversity Promotes Higher Diversity of Fungal Pathogens, while it Decreases Pathogen Infection per Plant." *Ecology* 95: 1907–17.

Rudolf, V.H.W., and J. Antonovics. 2005. "Species Coexistence and Pathogens with Frequency-Dependent Transmission." *American Naturalist* 166: 112–8.

Searle, C.L., M.H. Cortez, K.K. Hunsberger, D.C. Grippi, I.A. Oleksy, C.L. Shaw, S.B. de la Serna, C.L. Lash, K.L. Dhir, and M.A. Duffy. 2016. "Population Density, Not Host Competence, Drives Patterns of Disease in an Invaded Community." *American Naturalist* 188: 554–66.

Strauss, A.T., D.J. Civitello, C.E. Cáceres, and S.R. Hall. 2015. "Success, Failure and Ambiguity of the Dilution Effect among Competitors." *Ecology Letters* 18: 916–26.

Vehtari, A., A. Gelman, and J. Gabry. 2017. "Practical Bayesian Model Evaluation Using Leave-One-out Cross-Validation and WAIC." *Statistics and Computing* 27: 1413–32.

Vehtari A., Gelman A., Simpson D., Carpenter B., Bürkner P.-C. 2021. "Rank-Normalization, Folding, and Localization: An Improved $\hat{R}$ for Assessing Convergence of MCMC (with Discussion)." *Bayesian Analysis* 16(2): https://doi.org/10.1214/20-ba1221

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

Young, H.S., R. Dirzo, K.M. Helgen, D.J. McCauley, S.A. Billeter, M. Y. Kosoy, L.M. Osikowicz, D.J. Salkeld, T.P. Young, and K. Dittmar. 2014. "Declines in Large Wildlife Increase Landscape-Level Prevalence of Rodent-Borne Disease in Africa." *Proceedings of the National Academy of Sciences USA* 111: 7036–41.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

How to cite this article: Rosenthal, Lisa M., Wesley R. Brooks, and David M. Rizzo. 2022. "Species Densities, Assembly Order, and Competence Jointly Determine the Diversity–Disease Relationship." *Ecology* 103(5): e3622. https://doi.org/10.1002/ecy.3622