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Title: Foliar disease incidence in a tropical seedling community is density dependent and varies along a regional precipitation gradient.

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Data Accessibility: The data and code associated with this study are available in the following Zenodo repositories: Data – 10.5821/zenodo.10456097, Code - 10.5281/zenodo.10456335

Abstract:

1. Many studies identify fungal and oomycete phytopathogens as natural enemies capable of influencing plant species composition and promoting diversity in plant communities. However, little is known about how plant-pathogen interactions vary along regional abiotic gradients or with tree species characteristics, which limits our understanding of the causes of variation in tree species richness.
2. We surveyed 10,756 seedlings from 272 tree species for disease symptoms along a mean annual precipitation gradient in the tropical wet forests of Central Panama for three months in the early wet season (June – August) and two months in the following dry season (March - April). Over 99% of observed disease symptoms were caused by necrotrophic foliar pathogens, while less than 1% of symptoms were attributed to soilborne pathogens. Foliar disease incidence was inversely related to mean annual precipitation, a pattern which may be due to greater disease susceptibility among dry forest species.
3. Foliar disease incidence increased with conspecific seedling density but did not respond to the proximity of conspecific adults. Although foliar disease incidence decreased as mean annual precipitation increased, the strength of conspecific density- or distance-dependence was independent of the precipitation gradient.
4. Seedlings of common tree species and species dispersed by non-flying mammals had a higher risk of foliar pathogen incidence. Increased disease in common species may help reduce their dominance.
5. **Synthesis:** The increases in foliar pathogen incidence with conspecific seedling density, species abundance, and dispersal mechanism indicate that foliar disease incidence is non-

random and may contribute to the regulation of tropical plant communities and species coexistence. Furthermore, the relationships between foliar disease incidence, dispersal mechanism, and precipitation suggest plant-pathogen interactions could shift as a response to climate change and disruption of the disperser community.

Resumen:

1. Muchos estudios han identificaban a los fitopatógenos fúngicos y oomicetos como enemigos naturales con capaz de influir la composición de especies de plantas y promover la diversidad en las comunidades vegetales. Sin embargo, se sabe poco sobre cómo varían las interacciones planta-patógeno a lo largo de gradientes abióticos regionales o con las características de las especies de árboles, lo que limita nuestra comprensión de las causas de la variación en la riqueza de especias de árboles.
2. Encuestemos 10,756 plántulas pertenecientes a 272 especies de árboles, observando síntomas de enfermedades a lo largo de un gradiente de precipitación media anual en los bosques húmedos tropicales de Panamá Central. Cumplimos el estudio durante tres meses en la temporada de lluvias (junio-agosto) y dos meses en la siguiente temporada seca (marzo-abril). Más del 99% de los síntomas observados fueron causados por fitopatógenos necrótrofos foliares, mientras que menos de 1% de los síntomas se atribuyeron a fitopatógenos del suelo. La incidencia de enfermedades foliares mostro una relación inversa con la precipitación media anual, un patrón que podría deberse a una mayor susceptibilidad a enfermedades entre las especies de bosques secos.
3. La incidencia de enfermedades foliares aumentó con la densidad de plántulas conspecíficos, pero no respondió a la proximidad de adultos conspecíficos. Aunque la incidencia de enfermedad foliar disminuyó a medida que aumentaba la precipitación

media anual, el efecto de la dependencia de la densidad o la proximidad conspecífico fue independiente de la gradiente de precipitación.

4. Las plántulas de especies comunes y especies dispersadas por mamíferos no voladores tenían un riesgo más alto de incidencia de patógenos foliares. Un aumento de enfermedades en especies comunes puede ayudar a reducir su dominancia.
5. **Síntesis:** Los aumentos en la incidencia de patógenos foliares con la densidad de plántulas conspecíficos, la abundancia de especies, y el mecanismo de dispersión indican que la incidencia de enfermedades foliares no es aleatoria y puede contribuir a la regulación de comunidades vegetales tropicales y a la coexistencia de especies. Además, las relaciones entre la incidencia de enfermedades foliares, el mecanismo de dispersión, y precipitación sugieran que las interacciones planta-patógenos podrían cambiar como respuesta al cambio climático y a la alteración de la comunidad dispersores.

Key Words: plant-pathogen interactions, Janzen-Connell hypothesis, tropical tree diversity, precipitation gradient, seedling disease, Panama, foliar pathogens

Introduction:

Fungal and oomycete phytopathogens (hereafter pathogens) have a well-established role in tropical seedling dynamics (Augspurger, 1984; Bagchi et al., 2014; Eck et al., 2019; Mangan et al., 2010). Pathogens can increase the diversity of seedling assemblages (Bagchi et al., 2014; Krishnadas et al., 2018; Mangan et al., 2010). Many studies and experiments find that pathogens disproportionately attack seedlings occurring at high conspecific densities (Bell et al., 2006; Gilbert et al., 2001) or close to conspecific adults (Negative Conspecific Density/Distance Dependence, NCDD; Augspurger, 1984; Augspurger & Kelly, 1984; Gilbert et al., 2001; Howe et al., 1985), as predicted by the Janzen-Connell hypothesis (Connell, 1971; Janzen, 1970). In

young seedlings, pathogen-driven NCDD can be caused by soilborne pathogens, which infect roots (e.g., Augspurger, 1984), or foliar pathogens, which infect leaves (e.g., X. Liu et al., 2022), but it is unclear what their relative contributions to NCDD are. Additionally, species typically restricted to drier forests and shade-intolerant species are particularly susceptible to pathogen attack, which may limit their distributions and reinforce abiotic niches (McCarthy-Neumann & Kobe, 2008; Spear et al., 2015; Spear & Broders, 2021). Taken together, the literature suggests that pathogens play an integral role in structuring tropical tree communities (Comita & Stump, 2020; Milici et al., 2020; Terborgh, 2020).

Manipulative experiments that connect pathogens to patterns of NCDD in seedlings (e.g., by inhibiting fungi via fungicides) are frequently restricted to small spatial scales of a few square meters (e.g., Bagchi et al., 2014; Hazelwood et al., 2021; Mangan et al., 2010; McCarthy-Neumann & Kobe, 2008). Characterizing patterns of pathogen attack on seedlings across larger, regional, scales is nigh impossible with manipulative experiments. Although large-scale examinations of disease patterns are generally more feasible with observational studies, studies of regional variation in pathogen impacts are uncommon in tropical forests. Direct observations of pathogen incidence in seedling assemblages across multiple forest sites, especially through the crucial first year of a seedling's life, enable a wider-scale view of their response to the distribution and characteristics of their hosts. Such work also allows us to observe if pathogen attack is density-dependent at large spatial scales, and whether any density dependence varies seasonally, across regional abiotic gradients, and among species.

Pathogen pressure on seedlings likely varies throughout the year in seasonal tropical forests. The moist conditions during the rainy season favour pathogen transmission between seedlings, while the lack of rainfall during the dry season creates poor conditions for the

93 transmission of foliar and soilborne pathogens. Furthermore, in the seasonal tropical forests of
94 Panama, the onset of the rainy season coincides with the peak in germination (Garwood, 1983),
95 and seedlings are most susceptible to pathogens in the weeks following germination (Garrett,
96 1970). Thus, pathogen pressure on seedlings may be highest at the start of the rainy season and
97 diminish towards its end. This pattern has been observed in soilborne pathogen attack on the
98 tropical tree species *Platypodium elegans* (Augspurger, 1983), and may be generalizable to other
99 species. Conversely, it may take time for pathogens to find and infect a suitable host, and
100 pathogen pressure may be initially low and increase throughout the rainy season; a pattern that
101 has been observed for foliar fungi on trees and shrubs in Panama (Piepenbring et al., 2015). We
102 know of no study that has systematically surveyed tropical forest seedlings at a community scale
103 to establish temporal patterns in the incidence of either soilborne or foliar disease.
104 Pathogen-seedling interactions are also likely to vary along regional abiotic gradients,
105 potentially leading to pathogens influencing regional variation in plant community composition.
106 Moisture is thought to be an important abiotic modifier of plant-pathogen interactions at the
107 community level (Gillett, 1962; Lebrija-Trejos et al., 2023; Y. Liu & He, 2019) and a
108 strengthening of pathogen-caused NCDD in moist areas has been hypothesized as an explanation
109 for the positive relationship between precipitation and tree species richness that is commonly
110 observed in the American tropics (Bever et al., 2010; Givnish, 1999; Milici et al., 2020). The
111 interaction between moisture, pathogens, and their plant hosts has been extensively researched in
112 agroecosystems, showing that pathogen outbreaks are generally more severe under wetter
113 conditions (Velásquez et al., 2018), which has also been observed in tropical tree seedlings
114 (Swinfield et al., 2012). However, wet forest tree seedlings appear less susceptible to pathogen
115 damage and death than dry forest tree seedlings (Spear et al., 2015). This may be caused by a

116 growth-defensive trade-off where wet forest species are more slow-growing but better defended
117 against natural enemies than dry forest species (Coley et al., 1985). Thus, although pathogens
118 may benefit from wetter conditions, variation in plant traits associated with defence may counter
119 this effect.

120 Several plant species traits and demographic characteristics may be related to disease
121 susceptibility: for example, seed mass, rarity, and seed dispersal mechanism. Smaller-seeded
122 species may be more susceptible to disease because they are less well-provisioned than larger-
123 seeded species (Dalling et al., 2011). Adult abundance may also affect disease patterns in the
124 seedling community. When adults of a given species are locally abundant this may result in the
125 accumulation of host-specific pathogens and cause their seedlings to experience increased
126 disease incidence and mortality (Bachelot et al., 2017; Liang et al., 2016; Y. Liu et al., 2015).
127 Conversely, the small population sizes of rare species can result in lower defence-gene diversity,
128 and increase their susceptibility to pathogens (Marden et al., 2017). Dispersal mechanism may
129 also be related to disease incidence patterns because it is during dispersal that seeds may move
130 into enemy-free space (Howe & Smallwood, 1982). Species that are highly sensitive to
131 pathogens may be under high selective pressure to disperse far away from their parent (Eck et al.,
132 2019). Bat dispersal may successfully disperse seeds away from natural enemies (Sugiyama et
133 al., 2018) and endozoochorous bird dispersal may cleanse seeds of pathogens to reduce disease
134 incidence (Fricke et al., 2013), but it is uncertain if all dispersal modes are equally helpful for
135 evading pathogen infection.

136 While there is considerable evidence suggesting that pathogens influence tree
137 composition (e.g., Augspurger & Kelly, 1984; Bagchi et al., 2014; Gilbert et al., 2001; Mangan
138 et al., 2010; Spear et al., 2015), we lack a full understanding of how disease incidence varies

among species and in response to temporal or regional abiotic variation, particularly in highly diverse tropical tree communities. To identify patterns in pathogen infection and sources of variation, we surveyed tree seedling recruits for symptoms of pathogen infection within five forest plots along a regional gradient of mean annual precipitation in central Panama. Surveys were conducted in both the wet and dry season and predominantly encountered foliar pathogens. These data were used to test the following predictions:

(i) Pathogen incidence will increase as time since germination progresses and will intensify with mean annual precipitation across a regional precipitation gradient.

(ii) Pathogen incidence on seedlings will be greatest for locally abundant species and small-seeded species, and will vary depending on dispersal agents.

(iii) Pathogen incidence will increase with proximity to conspecific adults and/or the local density of conspecific seedlings and this relationship will strengthen with increasing mean annual precipitation.

Methods:

Study Sites

Seedling surveys were conducted within the forests of central Panama, along a regional precipitation gradient (Fig. 1; Table 1). Mean annual precipitation (MAP) across these sites ranges from near the lower limit for tropical wet forests (1700 mm y^{-1}; Richards et al., 1996) to almost double that amount. We did not include truly arid forests in our study. We conducted the surveys in five 1-ha (100 m x 100 m) forest plots, all of which are part of the network of 65 1-ha ForestGEO plots found across Panama (Condit et al., 2013, 2022). The plots were chosen because they span almost the entirety of the regional precipitation gradient across the canal zone of Panama. Mean annual precipitation at these sites ranged from 1874 to 2848 mm, with a 4-5

month dry season occurring between December and May (Condit et al., 2013). The trees within each plot are censused every five years, and all stems ≥ 1 cm dbh are tagged, identified to species, and mapped (Condit, 1998b). We obtained permission to work at these sites through the Panamanian Ministerio de Ambiente (permit number: SE/P-6-19).

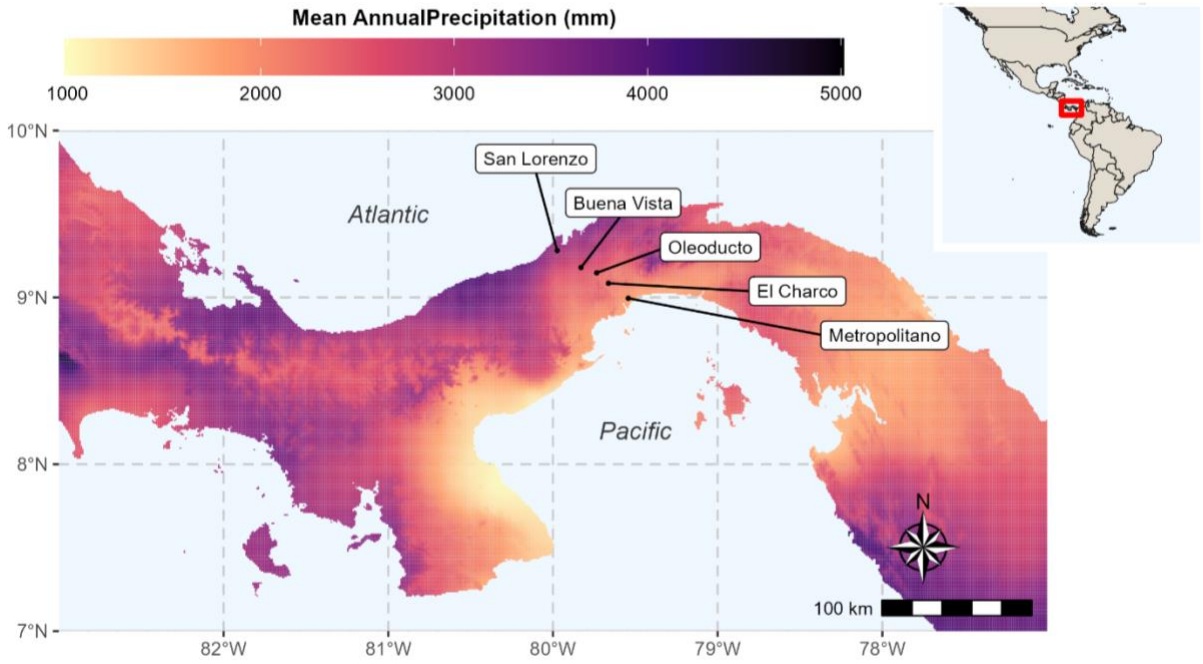


Figure 1: A map of the locations of the five forest plots used in this study. The sites were selected because they span nearly the entirety of the annual precipitation gradient in Panama. Summary information for each site is provided in Table 1.

Seedling Surveys

We conducted monthly surveys of seedling disease at each of our five sites for three months during the early rainy season (June – August 2018) and two months during the late dry season (March & April 2019) for a total of five surveys per site. Wet season surveys coincided with the germination of many tree species (Garwood, 1983) to observe pathogen infection in the weeks and months immediately following germination, when seedlings are most-vulnerable (Garrett, 1970). Dry season surveys were conducted towards the end of the dry season when seedlings are

most likely to experience drought stress and have been exposed to pathogens for many months (Piepenbring et al., 2015).

A survey consisted of ten 10 x 1 m belt transects, whose locations were randomly assigned and adjusted to avoid overlap. For each survey, ten new transects were selected at a site, so that each transect represented a unique sample of seedlings. We completed novel sets of transects during each survey to maximize the number of seedling and tree species included in our study because tropical seedling densities are generally quite low and unevenly distributed across the forest floor (Comita et al., 2010). Our approach allowed us to quickly assess a large number of seedlings for symptoms. However, a trade-off of this study design is that we were unable to track individual seedling performance and therefore the consequences of observed seedling infections were unknown. Along each transect, leaf litter was carefully removed to expose small seedlings, and all tree seedlings were identified to species and categorized as symptomatic or asymptomatic of infection. A seedling was identified as symptomatic of foliar disease if at least 20% of one leaf affected by necrotic lesions, or if they exhibited the orange-yellow spots or dry white coating associated with rust and downy or powdery mildew disease. A seedling was identified as symptomatic of soilborne disease if they exhibited a wrinkling and wilting of the stem at the soil interface consistent with vascular wilt caused by soilborne pathogens. Locations of seedlings were recorded to the nearest 1 m to allow calculation of local conspecific and heterospecific seedling densities and possible effects of nearby adult trees. Since many trees can persist for years at low stature in the forest understory (Hubbell, 1998; Rozendaal et al., 2010), we defined seedlings as individuals that had recruited within the 2018 germination cycle. New recruits were identified by the presence of cotyledons, few true leaves, short stature, and/or green stem. Seedlings that could not be identified to species in the field were collected and identified

either through reference to a dichotomous key (Garwood, 2009) or after consultation with a field botanist with expertise in the Panamanian flora (A. Hernandez; Smithsonian Tropical Research Institute). Seedlings that remained unidentifiable were dried, photographed, and assigned a morpho-species code.

STATISTICAL ANALYSIS

Data Preparation:

For analyses of species traits on tree seedling disease probability, all observations for which we had accompanying trait data (seed mass, or dispersal mechanism) were included in the models. Dispersal mechanism data were obtained from Wright et al. (2016) and seed mass data were obtained from Wright et al. (2010). To quantify adult abundance we used the most-recent tree census data (Condit et al., 2022) associated with our five study sites, which includes information on the diameter at breast height (DBH measured at 1.3 m above the ground) of stems ≥ 10 mm DBH in the 1-ha plot. A species' abundance was evaluated as the total basal area of that species at each site surveyed, $\sum \pi(\frac{DBH}{2})^2$, corresponding to a seedling observation. Because trait and abundance data were not available for all species, models evaluating relationships between traits or abundance and disease prevalence included only a subset of the seedling data. For dispersal mechanism, our dataset included sufficient observations to model the following dispersal types: small bird, large bird, bat, non-volant mammal, and wind. Due to limited observations of water (n = 1) and explosive dispersal (n = 36), these dispersal types were not evaluated. Models evaluating adult basal area included 8,264 observations from 96 species, models of seed mass included 3,320 observations from 99 species, and models of dispersal mechanism included 8,434

observations from 116 species. Models that evaluated the relationships between symptom incidence and both mean annual precipitation and time used the entire dataset of 10,756 seedlings from 272 species.

We used the most-recent tree census data (Condit et al., 2022) for each of our survey locations to calculate the proximity (1/distance) of each seedling to its nearest conspecific and nearest heterospecific adult. The use of proximity facilitates comparison between analyses of proximity and density (i.e., positive estimates in both analyses indicate negative distance/density dependent disease incidence). For the density and proximity-dependence analyses we excluded data from species that were observed on fewer than three transects, species that did not vary in their proximity to conspecific adults (proximity models only), species for which adults were not observed in any plots (proximity models only), and species that did not vary in their conspecific seedling densities (density models only). In total, models evaluating density-dependent disease incidence included 3,492 observations from 52 species, and models of proximity-dependent disease incidence included 3,389 observations from 55 species.

Model Form:

All models assessed the probability that a seedling was symptomatic of disease using a binomial generalized linear mixed-effects model with a logit link, included month of census as a categorical fixed effect, and included random intercepts for species, site, and transect within site. Models of density or proximity included a random slope for species to estimate variation among species in effects of density or proximity on disease incidence. Effects of conspecific density or proximity could be nonlinear and bias our estimation (Detto et al., 2019; Hülsmann et al., 2021), so in our models of density or proximity we compared the model performance of linear and nonlinear functional forms (fitted using a GAMM with a spline for the effect of

density or proximity) using AIC. A linear functional form was best supported by our data (had a lower AIC) and as such is the form used for our analyses (ΔAIC 0.604 for conspecific density, ΔAIC 0.830 for proximity to nearest conspecific adult; Supp. Table 1 & 2).

We modelled the relationship between mean annual precipitation (MAP) or species characteristics on disease incidence by adding a fixed effect for MAP or species characteristic to the model form described above. We allowed MAP and month of census to interact to assess possible seasonal shifts in the effects of MAP on disease incidence.

We evaluated the effects of seedling density or distance to conspecific adult in separate models because models that evaluated them simultaneously failed to converge, likely due to data limitations (small number of observed symptomatic seedlings; n=364). In these models, we allowed conspecific and heterospecific density (or proximity) to interact with MAP. Although conspecific seedling density and proximity to conspecific adult are frequently correlated (Gilbert et al., 1994; Nathan & Muller-Landau, 2000), the correlation was weak in our dataset (Pearson's correlation: $r = 0.07$, df = 1695, $P = 0.003$).

All continuous explanatory variables were log-transformed and then standardized (by subtracting their mean and dividing by their standard deviation) to facilitate model convergence and allow direct comparison of parameter estimates. Both categorical explanatory variables, census month and dispersal mechanism, were evaluated as sum-to-zero contrasts so that the effect of each census or dispersal type can be interpreted relative to their mean incidence probabilities. The 95% confidence intervals were estimated via parametric bootstrapping with 1,000 iterations (Davison & Hinkley, 1997).

Observational studies may be affected by survivorship bias (e.g., Visser et al., 2018), because observing an individual is conditional on it surviving until the survey. In this study, we

may not have observed infections on the most vulnerable species because they succumb rapidly to their infections, biasing our observations towards tolerant species. As a result, this would bias parameter estimates and our conclusions. We checked for survivorship bias by estimating the proportion of the seedling community that could have been symptomatic for each census and at each site (P):

$$P = \frac{\sum_1^i (n_i w_i)}{\sum_1^i n_i} \quad \text{(Eq. 1)}$$

Where n_i is the number of seedlings observed in a census for species i, weighted by its species-specific probability of being observed as symptomatic (w_i, calculated as the proportion of species i observed as symptomatic across all sites and censuses). This value was summed for all seedling observations at each site and census combination and then divided by the total number of observations (n_i) for that site and census. A decrease in the observations of symptomatic species over time would indicate a loss of susceptible species consistent with expectations for survivorship bias. All statistical analyses were performed using R v.3.6.3 (R Core Team 2020) and using the package 'lme4' (Bates et al., 2015).

Results:

Overall, it was uncommon for us to encounter symptomatic seedlings; only 3% (364 seedlings) of the 10,756 seedlings (272 species) observed were symptomatic of disease. Of those symptomatic seedlings, over 99% (362 seedlings) were symptomatic of necrotrophic foliar pathogen damage (i.e., necrotic lesions on the leaves); therefore, the results of the analyses are specific to foliar pathogens as opposed to soilborne pathogens that damage roots and cause wilt-like symptoms (e.g., Augspurger, 1983). Densities of recently germinated seedlings were quite

low; the mean conspecific seedling density was 1 seedling m^{-2} (Interquartile range, IQR = 1-2 seedlings), and mean total seedling density was two seedlings m^{-2} (IQR = 1-5 seedlings m^{-2}). Seedling abundance was similar across sites, except for El Charco, which had few wet season seedling observations relative to the other sites, possibly due to a steep hillslope across half of the plot limiting areas suitable for germination (V. Milici, *pers. obs.*). Seedlings were much more abundant in the wet season than the dry season, especially in the site where the dry season is most pronounced (Table 1; Metropolitano). Our test for survivor bias showed no consistent pattern across the sites surveyed (Fig. S1).

Both mean annual precipitation (MAP) and month of census affected foliar disease incidence in the seedling community (Fig. 2). In general, foliar disease was more prevalent among the seedlings at the driest sites and decreased with increasing MAP (β_{MAP} = -0.65 ±SE = 0.27, P = 0.02), a relationship that was accentuated at the start of the rainy season in June ($\beta_{MAP \cdot June}$ = -1.56 ± 0.67, P = 0.02), reduced in the middle of the dry season in March ($\beta_{MAP \cdot March}$ = 0.59 ± 0.35, P = 0.09), and changed direction at the end of the dry season in April ($\beta_{MAP \cdot April}$ = 0.87 ± 0.38, P = 0.02). Overall, foliar disease incidence was lowest towards the beginning of the rainy season in June (β_{June} = -2.17 ± 0.69, P = 0.001), and highest towards the end of the dry season in March (β_{March} = 1.45 ± 0.33, P < 0.001) and April (β_{April} = 0.85 ± 0.34, P = 0.01).

Table 1: Summary of the seedling observations from each site, grouped by season. The wet season observations were made in June, July, and August (2018), and the dry season observations were made in March and April (2019). Longitude and latitude are recorded in decimal degrees and mean annual precipitation (MAP) in millimetres. The sites are organized by increasing MAP.

Site Name	Longitude	Latitude	MAP	Wet Season		Dry Season	
				No. Seedlings	No. Species	No. Seedlings	No. Species
Metropolitano	8.995	-79.543	1874	6612	36	67	24
El Charco	9.084	-79.663	2050	140	27	91	23
Oleoducto	9.146	-79.712	2311	774	48	119	23
Buena Vista	9.179	-79.829	2595	514	69	467	27
San Lorenzo	9.281	-79.975	2848	1595	89	377	58

Dispersal mechanism and adult abundance, but not seed mass helped explain the variation in foliar disease incidence among species. Species dispersed by non-volant mammals (e.g., monkeys, agouti, tapir) had a higher disease incidence than other dispersal types examined ($\beta_{Non\text{-}volant} = 4.89 \pm 1.53$, $P = 0.001$), with species with other dispersal mechanisms (small bird, large bird, bat, and wind dispersal) showing relatively similar disease incidence to each other (Table 2). Adult abundance (total basal area of conspecific adults within each 1-ha forest plot) was also associated with foliar disease incidence (Fig. 3). As site-wide adult abundance increased for a

given species, so too did disease incidence among its seedlings ($\beta_{Abundance}$ = 1.09 ± 0.37, P = 0.003). Seed mass, however, was not associated with variation in disease incidence among seedlings ($\beta_{SeedMass}$ = 0.24 ± 0.17, P = 0.16).

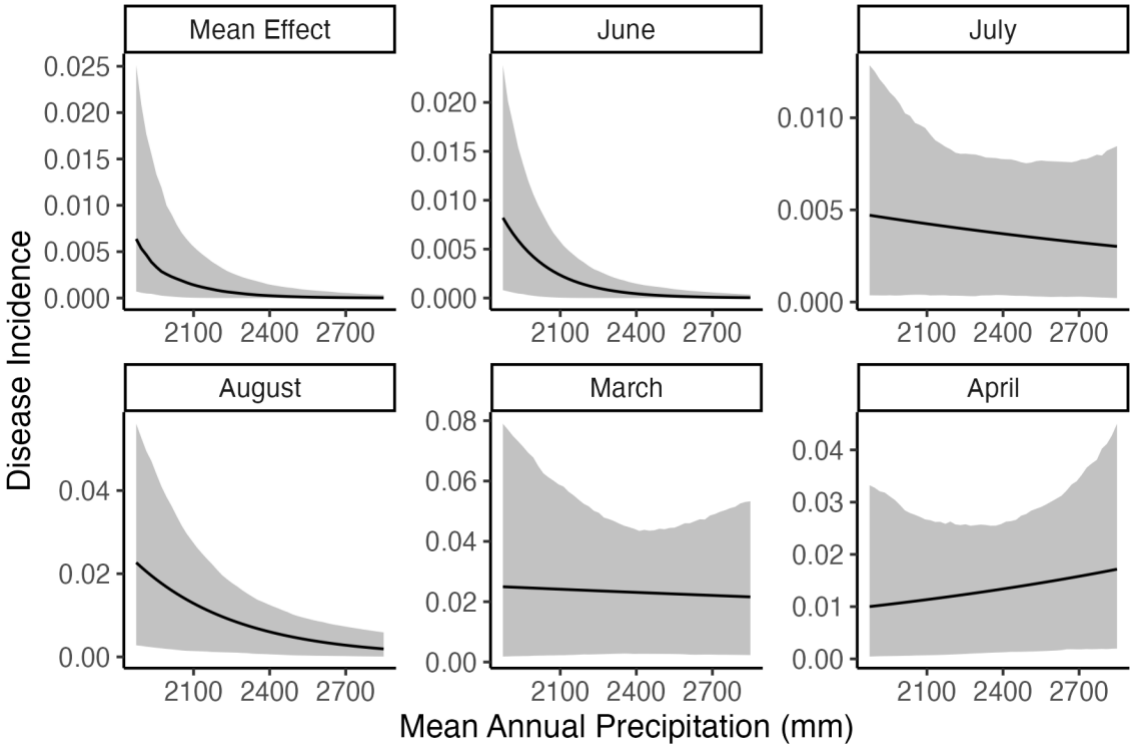


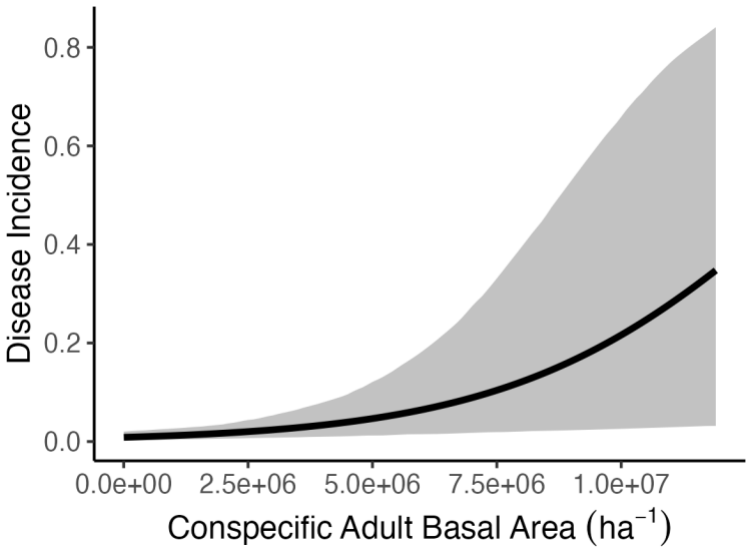
Figure 2: The relationship between mean annual precipitation (MAP, mm) and time of year on foliar disease incidence (proportion) among recently germinated tree seedlings in the forests of Central Panama. Disease incidence was low across all forest sites and censuses but varied with MAP and time of year. Incidence tended to be highest in the driest forests and decrease with increasing MAP (β = -0.65 ± 0.27, P = 0.02), however this effect was reduced in March (β = 0.59 ± 0.35, P = 0.09), and reversed in April (β = 0.87 ± 0.38, P = 0.02). Disease incidence was lowest during the initial census in June (β = -2.17 ± 0.69, P = 0.001) and increased in March (β =1.45 ± 0.33, P < 0.001) and April (β = 0.85 ± 0.34, P = 0.01). Lines represent mean parameter

values estimated using binomial GLMM and error around mean estimates represent the 95% confidence interval estimated by parametric bootstrapping. Models were fit using 10,756 observations from 272 species.

Foliar disease incidence was significantly related to local conspecific seedling density ($\beta_{Density}$ = 0.16 ± 0.08, P = 0.04), increasing on seedlings at higher conspecific densities (Fig. 4A). The effect of conspecific seedling density on disease incidence was consistent across the species surveyed; the estimated variance component for species-specific effects of conspecific density (random slope) was indistinguishable from zero (likelihood test ratio, $\chi^2_1 = 0$, P = 0.99). We found no evidence for a community-wide effect of proximity to conspecific adults on disease incidence across the 55 species analysed ($\beta_{Proximity}$ = -0.33 ± 0.37, P = 0.38, Fig. 4B). Examination of the species-specific random slopes suggested that proximity to adult conspecifics increased the risk of foliar disease for five species and decreased the risk for one species (Fig. 5), although the community-wide estimate of the variance component (random slope) was non-significant (Var = 13.5; likelihood test ratio, $\chi^2_1 = 2.46$, P = 0.16). Neither heterospecific seedling density ($\beta_{HetDensity}$ = 0.14 ± 0.13, P = 0.27) nor proximity to heterospecific adults ($\beta_{HetProximity}$ = 0.11 ± 0.07, P = 0.14) influenced disease incidence.

Table 2: Summary statistics for binomial GLMM examining the relationship between disease incidence and dispersal mechanism. Bold text denotes a statistically significant relationship ($P < 0.05$). Categorical variables (census and dispersal mechanism) were coded with sum-to-zero contrasts (effects coding) so each estimate is the difference between disease incidence in the corresponding category and the overall mean.

Explanatory Variable	Estimate	SE	*z*-value	*P*-value
Intercept	-6.71	0.72	-9.36	< 0.001
Bat	0.25	1.22	0.21	0.837
Nonvolant-Mammal	**4.89**	**1.54**	**3.18**	**0.001**
Small Bird	-1.70	1.24	-1.37	0.169
Large Bird	-1.87	1.35	-1.39	0.166
Wind	2.22	2.18	1.02	0.309
July Census	0.46	0.48	0.95	0.339
August Census	**1.17**	**0.47**	**2.45**	**0.014**
March Census	**2.36**	**0.53**	**4.49**	**< 0.001**
April Census	**1.95**	**0.53**	**3.66**	**< 0.001**



388

389 **Figure 3**: Disease incidence of tree seedlings in the forests of central Panama increases as
390 conspecific adult abundance (total basal area within 1-ha forest plot; $\beta = 1.09 \pm 0.37$, $P = 0.003$).
391 The line represents the mean parameter estimate, generated through binomial GLMM. The
392 shading represents the 95% CIs estimated via parametric bootstrapping. Models evaluated using
393 8264 observations from 92 species.

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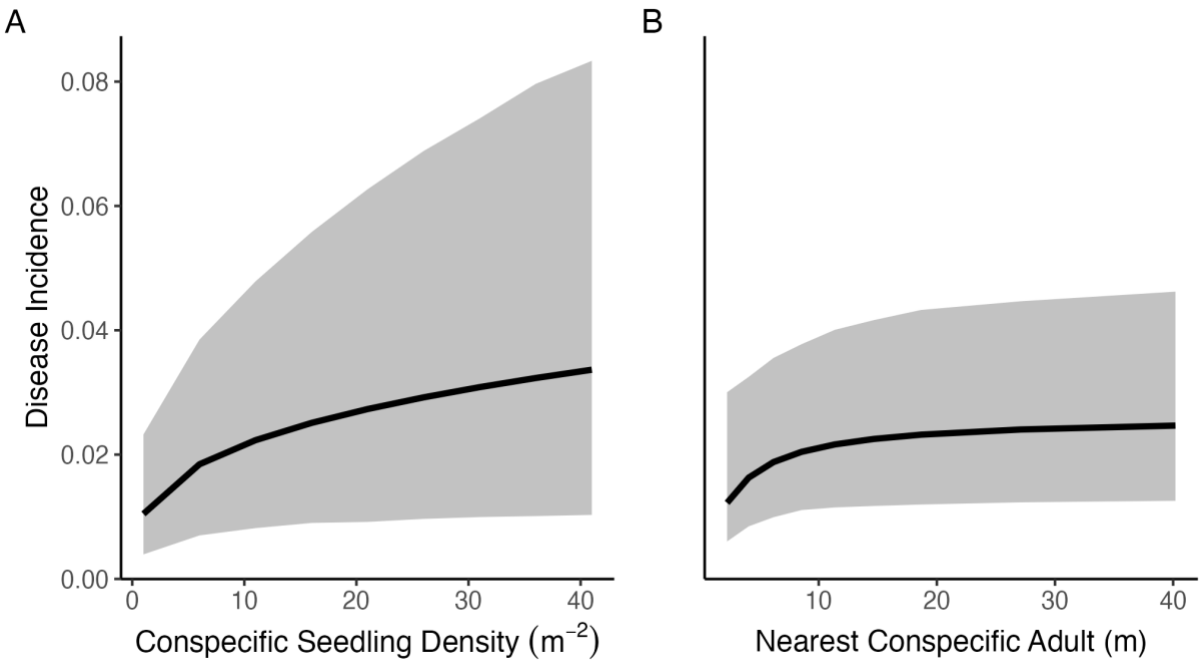
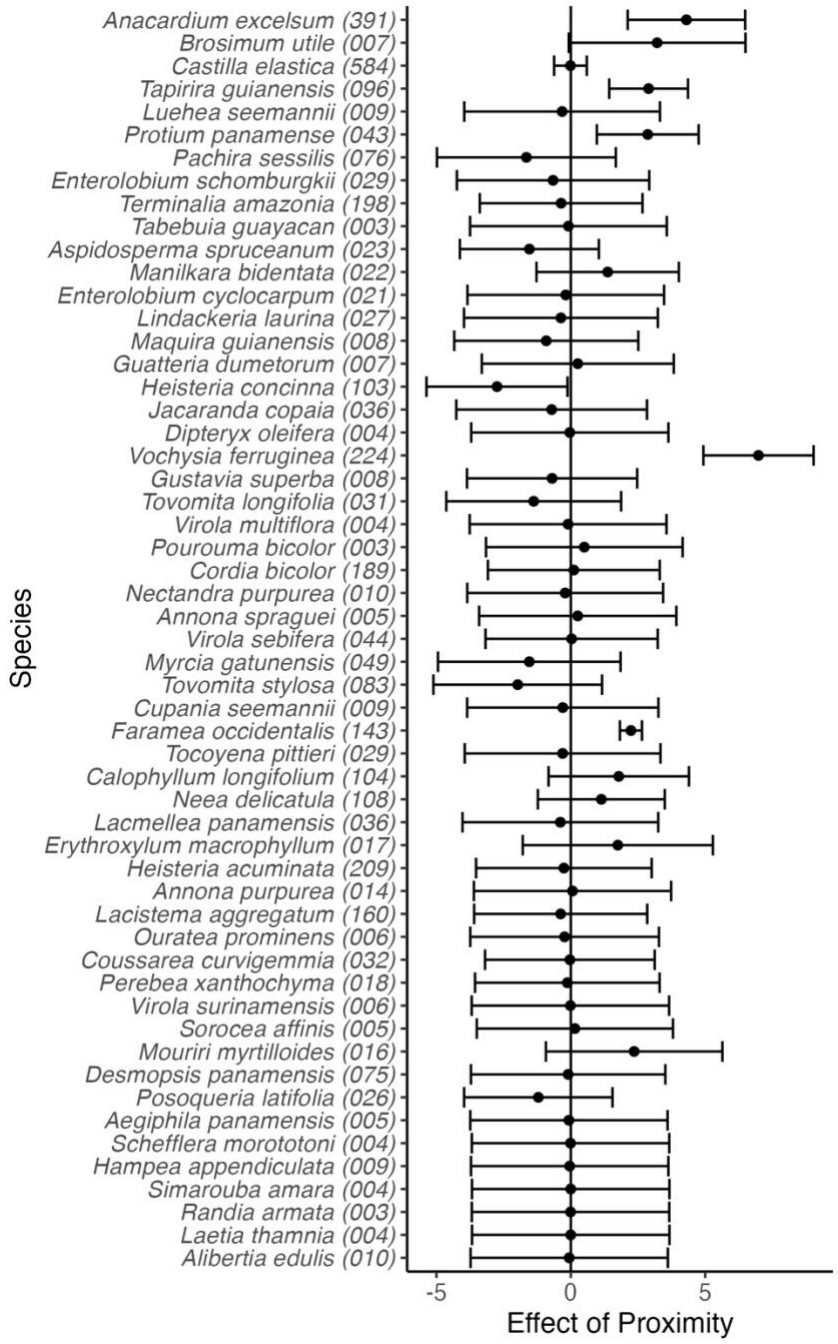


Figure 4: Disease incidence of tree seedlings in the forests of central Panama increases as conspecific seedling density increases ($\beta = 0.16 \pm 0.08$, $P = 0.04$, A) and has no clear relationship with proximity to the nearest conspecific adult ($\beta = -0.33 \pm 0.37$, $P = 0.38$, B). Lines represent the mean parameter estimates, generated through binomial GLMM. The shading represents the 95% CIs estimated via parametric bootstrapping. Models of density (proximity) evaluated 3492 (3389) observations from 52 (55) species.



405

406 **Figure 5:** The variation in the species-level slopes suggests that five species (*A. excelsum, T.*

407 *guianensis, V. ferruginea, P. panamense, and F. occidentalis*) experience higher disease

408 incidence than average when closer to a conspecific adult, while one species, *H. concinna*,

409 experiences below-average disease risk when closer to a conspecific adult. The community-wide

410 estimate for the variance component was itself non-significant (Var = 13.5; likelihood ratio test,

411 $\chi^2_1 = 2.46$, $P = 0.16$). The species are arranged in descending order of how common they are in
412 the forest as adults (the sum of site-wide basal area). The number of seedling observations of
413 each species is in parenthesis. Points represent the mean estimated deviance from the overall
414 community effect of proximity on disease while bars are the standard deviation around the mean
415 prediction.

416 Although we found a general effect that disease incidence decreased with increasing
417 MAP, MAP did not explain variation among sites in the effects of either conspecific seedling
418 density ($\beta_{MAP \bullet Density} = 0.07 \pm 0.06$, $P = 0.26$) or conspecific adult proximity ($\beta_{MAP \bullet Proximity} = -0.20$
419 ± 0.23, $P = 0.38$) on disease incidence.

420 **Discussion:**

421 We conducted surveys of seedling disease across a regional precipitation gradient in the forests
422 of central Panama to characterize the effects of mean annual precipitation, conspecific seedling
423 density, distance to nearest adult conspecific, and species characteristics on disease incidence in
424 the young seedling community. Although we sought to characterize disease caused by both
425 soilborne and foliar pathogens, we almost exclusively recorded foliar disease. Pathogens are
426 often thought to be more prevalent in wetter areas (Gillett, 1962; Givnish, 1999; Milici et al.,
427 2020), but foliar disease incidence was highest in the forest sites with lowest annual precipitation
428 and during the dry season. Foliar disease incidence was also non-randomly distributed; seedlings
429 were more likely to exhibit foliar pathogen damage as conspecific seedling density increased, if
430 they were a common species, or if they were dispersed by non-flying mammals. Overall, this
431 study suggests foliar pathogens help shape seedling community composition and diversity,
432 particularly in drier tropical forests and during the dry season.

(i) Foliar pathogen incidence varies throughout the year and along the regional precipitation gradient.

Pathogens have long been thought to produce more infections as mean annual precipitation increases (Gillett, 1962; Givnish, 1999; Milici et al., 2020), but we show that apart from the late dry season, foliar disease incidence is actually most prevalent in the driest sites. This relationship was significant, and consistent through the wet season, despite the study including only five sites. Differences in defence allocation among the species assemblages across the precipitation gradient may explain this relationship. Available soil moisture is an important driver of tree species distribution across Panama (Condit et al., 2013; Engelbrecht et al., 2007), and as a result there is a high degree of community turnover along the precipitation gradient. In Panama, fast-growing light demanding tree species, more characteristic of the seedling community in our drier sites, have decreased chemical defence allocation and higher rates of attack by insect herbivores (Coley, 1983) compared to slow-growing shade tolerant species, more characteristic of the seedling community in our wetter sites (Coley, 1983; Gripenberg et al., 2018). Better defended wet-forest species may be better able to resist pathogen infection despite the moist climate that favours pathogen transmission and germination (Barrett et al., 2009; Garrett, 1970; Swinfield et al., 2012). Evidence for a trade-off between growth and defence has been found in many ecological communities, including the precipitation gradient studied here, suggesting it may be a universal phenomenon (Coley, 1983; Coley et al., 1985; Coley & Barone, 1996; Fine et al., 2006; Lind et al., 2013). A growth-defence trade-off was the best predictor of foliar pathogen incidence for grassland species (Cappelli et al., 2020), and may explain why seedlings of drier forest species were more susceptible to pathogen damage and death than wetter forest species in an experiment in two of the forest sites included in this study (Spear et al., 2015, *but see* Gaviria

& Engelbrecht, 2015). Alternatively, increased foliar pathogen incidence in dry forests may be the result of a gradient in insect herbivore interactions. Foliar pathogens often require damage by insect herbivores to infect a host (García-Guzmán & Dirzo, 2001), which decrease in prevalence as mean annual precipitation increases across these sites in Panama (Weissflog et al., 2017). Thus, decreased rates of herbivory may cause fewer opportunities for foliar pathogens to infect seedlings as mean annual precipitation increases, which would result in a decreased foliar pathogen disease incidence in areas of high mean annual precipitation.

Foliar pathogen incidence among recently germinated seedlings was initially low, increased as the wet season progressed and into the dry season. This pattern is likely caused by a combination of exposure period and dry season drought-stress. In the seasonal tropical forest studied here, most seedlings germinate early in the wet season (Garwood, 1983), and it takes time for the new leaves to be colonized by fungi (Arnold & Herre, 2003; Piepenbring et al., 2015). Thus, the seedlings in the early wet season may have not yet been exposed to foliar pathogens or had time to develop symptoms of infection. Additional increases in symptomatic seedlings during the dry season could be owed to drought stress, which prior studies have shown affects a large proportion of the seedling species at our sites (Engelbrecht et al., 2007; Engelbrecht & Kursar, 2003; Gaviria et al., 2017) as well as many other seasonal tropical forest regions (Comita & Engelbrecht, 2014). Drought stress can compromise the plant immune response, making plants more susceptible to infections in a process known as “predisposition” (Desprez-Loustau et al., 2006; Schoeneweiss, 1975). A positive effect of drought on fungal pathogen development within hosts is well-documented (e.g., Ghanbary et al., 2017, 2021; Jactel et al., 2012; Sinha et al., 2019); even beneficial fungi, such as foliar endophytes, can become pathogenic when their hosts are drought-stressed (Slippers & Wingfield, 2007). Under climate

change, increased seasonality of precipitation in Central American forests is predicted to intensify dry seasons (Condit, 1998a; Kirtman et al., 2013; Lintner et al., 2012). Our finding that foliar pathogen incidence is highest during the dry season for all sites along the precipitation gradient, suggests that dry-season intensification could lead to increases in foliar pathogen damage among seedlings. Given the established role of pathogens in influencing seedling survival, growth, and diversity (Bagchi et al., 2014; Mangan et al., 2010) such intensification of disease could affect community composition of tropical forest tree assemblages (Condit, 1998a; Milici et al., 2020; Thompson et al., 2010).

(ii) Dispersal mechanism and rarity affect foliar pathogen incidence

It is generally accepted that escaping disease is an important benefit of dispersal (Augspurger, 1983; Howe et al., 1985; Russo, 2005; Sugiyama et al., 2018), but few studies have examined the relationship between dispersal mechanism and subsequent disease incidence (e.g., Fricke et al., 2013). Non-volant-mammal-dispersed species had much higher rates of foliar disease incidence than species dispersed by other means (e.g., wind or birds), which complements a study in the Peruvian Amazon that found higher seed pathogen incidence on primate-dispersed tree species (Pringle et al., 2007). While different enemies (e.g., insects) might be prevalent on tree species with alternative dispersal modes, the concordance between this study and Pringle et al. (2007) may suggest that tree species dispersed by non-flying mammals are particularly vulnerable to pathogens. The majority of seeds fall near their parents, regardless of dispersal mode (Muller-Landau et al., 2008), so the pattern of disease incidence on first-year seedlings described here predominantly reflects the fates of undispersed seeds and seedlings. Although further work would be necessary to confirm a greater vulnerability of non-flying-mammal-dispersed species to pathogens, it might imply that dispersal is particularly important

for the health of seedlings from these species compared to species dispersed by other means. Higher disease susceptibility might contribute to the reduced recruitment observed in many non-volant mammal dispersed species in defaunated forests in Panama and elsewhere in the tropics (Effiom et al., 2013; Kurten et al., 2015; Terborgh et al., 2008).

Foliar pathogen incidence, by being higher in seedlings of abundant species, may contribute to the persistence of rare species in the community. Our findings suggest that rarity confers an advantage to seedlings, likely due to the build-up of host-specific pathogens in areas where adults of a given species are locally abundant, which can cause a community compensatory trend (CCT; Liang et al., 2016; Y. Liu et al., 2015). A CCT has been documented in the seedling community of a Bornean rainforest (Webb & Peart, 1999) and has been attributed to soilborne pathogens in a subtropical forest in China (Liang et al., 2016; Y. Liu et al., 2015). Foliar disease incidence increases with species abundance in a temperate grassland (Mitchell et al., 2002) and contributes to a CCT in Puerto Rican tree seedlings (Bachelot et al., 2017). If the patterns of foliar disease we observed affect survival patterns, then the contribution of foliar pathogens to a CCT may be a widespread phenomenon that contributes to plant species coexistence by advantaging the survival and recruitment of rare species.

(iii) Foliar pathogen incidence is density- but not distance-dependent

Evidence from tropical to temperate forests has established that soilborne pathogens can mediate NCDD (Augspurger, 1983; Domínguez-Begines et al., 2021; Mangan et al., 2010; Packer & Clay, 2000) and are likely important to stabilizing species coexistence. As such, soilborne pathogens have been the focus of many studies, but the importance of foliar pathogens has been highlighted by a growing body of work (Bagchi et al., 2014; X. Liu et al., 2022; this study). We did not identify the foliar pathogens observed in this study, but previous research

(Piepenbring et al., 2011; Spear, 2017; Spear & Broders, 2021) suggests that they likely belonged to the Ascomycota (e.g., *Fusarium spp.* and *Colletotrichum spp.*). Many of the symptoms we observed were consistent with tropical tar spot fungi (Phyllochorales), a common fungal pathogen in the forests of Panama (Piepenbring et al., 2011). We show that these foliar pathogens result in negative conspecific density-dependent infection, suggesting that the contributions of foliar pathogens to NCDD should not be ignored. If the NCDD pattern in pathogen infection we report here reduces survival and growth, then foliar pathogens could also contribute to stabilizing coexistence by natural enemies as predicted by the Janzen-Connell hypothesis and modern coexistence theory (Chesson, 2000; Connell, 1971; Janzen, 1970).

Necrotrophic foliar pathogens represent the majority of pathogens that a plant encounters through the year and can reduce seedling performance later into plant development than soilborne pathogens (Rottstock et al., 2014). Foliar pathogens may even produce stronger patterns of NCDD and contribute more strongly to seedling mortality than soilborne pathogens (Bagchi et al., 2014; Bayandala et al., 2016, 2017; X. Liu et al., 2022). Our study shows that pathogen infection, which likely increases seedling mortality risk, occurs non-randomly in space and across species. Non-random patterns of mortality at the early life stages have been shown to be particularly important in shaping community structure and diversity in tropical tree communities (Green et al., 2014). Without tracking the fate of infected seedlings, we can only speculate about the long-term consequences for tree-community diversity of the patterns of conspecific-density dependent infection by foliar pathogens observed in this study. Nevertheless, our work provides evidence that foliar pathogens may contribute to maintaining the diversity of tropical trees through negative conspecific density dependent infection of seedlings, in addition to soilborne pathogens.

The Janzen-Connell hypothesis (Connell, 1971; Janzen, 1970) suggests that distance-dependent patterns of infection at the young seedling stage will increase tree diversity. However, our data indicated that foliar pathogens are not more prevalent near adult conspecific trees and, therefore, are unlikely to maintain tree community diversity as predicted by the Janzen-Connell hypothesis. Negative conspecific distance-dependent disease incidence was only evident for a small number of the species evaluated and the average effect over the 55 species in the analysis was weak, which is consistent with past research from Panama on the effects of distance dependence during the seed-to-seedling transition (Marchand et al., 2020). For this community, distance dependence is widespread at the later seedling stage (Murphy et al., 2017), which suggests the pressures that cause distance-dependent recruitment may differ from those that affect the youngest seedlings. Compared to soilborne pathogens, foliar pathogens may be less likely to result in distance-dependent disease. Their airborne spores, dispersing from adult trees in the in the canopy above, can spread over a wide area so that the spores reaching a seedling could originate from trees far away (Jauss et al., 2021). Thus, the airborne spores that encounter and infect seedlings may originate from trees spread over a large area and, therefore, their composition may be unrelated to the identities of the nearest adult trees. Conversely, the spores of soilborne pathogens do not move large distances (Oliveira & Bell, 2022), concentrating them near parent trees and making them more likely to cause distance-dependent disease.

Conclusions:

Previous work has demonstrated that pathogen attack on tropical forest seedlings can be density-dependent in individual plant species (Augspurger, 1983; Augspurger & Kelly, 1984; Bell et al., 2006) and pathogens can increase the species diversity of seedling assemblages (Bagchi et al., 2014; Krishnadas et al., 2018; Mangan et al., 2010). Here, we document patterns of disease on a

large number of tree species, finding that the conspecific-density dependence of disease incidence on seedlings previously observed in small suites of species holds at the community scale. If these patterns influence recruitment, they are likely to explain pathogen effects on plant diversity in community-level experiments (Bagchi et al., 2014; Krishnadas et al., 2018; Mangan et al., 2010). Furthermore, whereas soil-borne pathogens have previously been associated with density-dependent attack on plant communities (Augspurger & Wilkinson, 2007; Bell et al., 2006; Mangan et al., 2010), we show that attack by foliar pathogens can also be density dependent. Finally, we show that foliar pathogens are most prevalent in drier conditions, in forests with the least annual precipitation and during the dry season. Thus, the more intense dry seasons predicted for many tropical forests under climate change may lead to an increase in foliar disease on seedlings. The implications of such increased disease for tree communities are difficult to predict, but the importance of pathogens for tree community diversity means the effects could have important consequences for tropical forest diversity.

References:

Arnold, A. E., & Herre, E. A. (2003). Canopy cover and leaf age affect colonization by tropical fungal endophytes: Ecological pattern and process in Theobroma cacao (Malvaceae). *Mycologia*, *95*(3), 388–398. https://doi.org/10.1080/15572536.2004.11833083

Augspurger, C. K. (1983). Seed Dispersal of the Tropical Tree, Platypodium Elegans, and the Escape of its Seedlings from Fungal Pathogens. *Journal of Ecology*, *71*(3), 759–771. https://doi.org/10.2307/2259591

Augspurger, C. K. (1984). Seedling Survival of Tropical Tree Species: Interactions of Dispersal Distance, Light-Gaps, and Pathogens. *Ecology*, *65*(6), 1705–1712. JSTOR. https://doi.org/10.2307/1937766

Augspurger, C. K., & Kelly, C. K. (1984). Pathogen Mortality of Tropical Tree Seedlings: Experimental Studies of the Effects of Dispersal Distance, Seedling Density, and Light Conditions. *Oecologia*, *61*(2), 211–217.

Augspurger, C. K., & Wilkinson, H. T. (2007). Host Specificity of Pathogenic Pythium Species: Implications for Tree Species Diversity. *Biotropica*, *39*(6), 702–708. https://doi.org/10.1111/j.1744-7429.2007.00326.x

Bachelot, B., Uriarte, M., McGuire, K. L., Thompson, J., & Zimmerman, J. (2017). Arbuscular mycorrhizal fungal diversity and natural enemies promote coexistence of tropical tree species. *Ecology*, *98*(3), 712–720. https://doi.org/10.1002/ecy.1683

Bagchi, R., Gallery, R. E., Gripenberg, S., Gurr, S. J., Narayan, L., Addis, C. E., Freckleton, R. P., & Lewis, O. T. (2014). Pathogens and insect herbivores drive rainforest plant diversity and composition. *Nature*, *506*(7486), 85–88. https://doi.org/10.1038/nature12911

Barrett, L. G., Kniskern, J. M., Bodenhausen, N., Zhang, W., & Bergelson, J. (2009). Continua of specificity and virulence in plant host–pathogen interactions: Causes and consequences. *New Phytologist*, *183*(3), 513–529. https://doi.org/10.1111/j.1469-8137.2009.02927.x

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2015). Fitting Linear Mixed-Effects Models Using **lme4**. *Journal of Statistical Software*, *67*(1). https://doi.org/10.18637/jss.v067.i01

Bayandala, Fukasawa, Y., & Seiwa, K. (2016). Roles of pathogens on replacement of tree seedlings in heterogeneous light environments in a temperate forest: A reciprocal seed sowing experiment. *Journal of Ecology*, *104*(3), 765–772. https://doi.org/10.1111/1365-2745.12552

617 Bayandala, Masaka, K., & Seiwa, K. (2017). Leaf diseases drive the Janzen–Connell mechanism
618 regardless of light conditions: A 3-year field study. *Oecologia*, *183*(1), 191–199.
619 https://doi.org/10.1007/s00442-016-3757-4
620 Bell, T., Freckleton, R. P., & Lewis, O. T. (2006). Plant pathogens drive density-dependent
621 seedling mortality in a tropical tree. *Ecology Letters*, *9*(5), 569–574.
622 https://doi.org/10.1111/j.1461-0248.2006.00905.x
623 Bever, J. D., Dickie, I. A., Facelli, E., Facelli, J. M., Klironomos, J., Moora, M., Rillig, M. C.,
624 Stock, W. D., Tibbett, M., & Zobel, M. (2010). Rooting theories of plant community
625 ecology in microbial interactions. *Trends in Ecology & Evolution*, *25*(8), 468–478.
626 https://doi.org/10.1016/j.tree.2010.05.004
627 Cappelli, S. L., Pichon, N. A., Kempel, A., & Allan, E. (2020). Sick plants in grassland
628 communities: A growth-defense trade-off is the main driver of fungal pathogen
629 abundance. *Ecology Letters*, *23*(9), 1349–1359. https://doi.org/10.1111/ele.13537
630 Chesson, P. (2000). Mechanisms of Maintenance of Species Diversity. *Annual Review of*
631 *Ecology and Systematics*, *31*(1), 343–366.
632 https://doi.org/10.1146/annurev.ecolsys.31.1.343
633 Coley, P. D. (1983). Herbivory and Defensive Characteristics of Tree Species in a Lowland
634 Tropical Forest. *Ecological Monographs*, *53*(2), 209–234.
635 https://doi.org/10.2307/1942495
636 Coley, P. D., & Barone, J. A. (1996). HERBIVORY AND PLANT DEFENSES IN TROPICAL
637 FORESTS. *Annual Review of Ecology and Systematics*, *27*(1), 305–335.
638 https://doi.org/10.1146/annurev.ecolsys.27.1.305

639 Coley, P. D., Bryant, J. P., & Chapin, F. S. (1985). Resource Availability and Plant
640 Antiherbivore Defense. *Science*, *230*(4728), 895–899.
641 https://doi.org/10.1126/science.230.4728.895
642 Comita, L. S., & Engelbrecht, B. M. J. (2014). Drought as a driver of tropical tree species
643 regeneration dynamics and distribution patterns. In D. A. Coomes, D. F. R. P. Burslem,
644 & W. D. Simonson (Eds.), *Forests and Global Change* (1st ed., pp. 261–308). Cambridge
645 University Press. https://doi.org/10.1017/CBO9781107323506.013
646 Comita, L. S., Muller-Landau, H. C., Aguilar, S., & Hubbell, S. P. (2010). Asymmetric Density
647 Dependence Shapes Species Abundances in a Tropical Tree Community. *Science, New*
648 *Series*, *329*(5989), 330–332.
649 Comita, L. S., & Stump, S. M. (2020). Natural Enemies and the Maintenance of Tropical Tree
650 Diversity: Recent Insights and Implications for the Future of Biodiversity in a Changing
651 World. *Annals of the Missouri Botanical Garden*, *105*(3), 377–392.
652 https://doi.org/10.3417/2020591
653 Condit, R. (1998a). Ecological Implications of Changes in Drought Patterns: Shifts in Forest
654 Composition in Panama. In A. Markham (Ed.), *Potential Impacts of Climate Change on*
655 *Tropical Forest Ecosystems* (pp. 273–287). Springer Netherlands.
656 https://doi.org/10.1007/978-94-017-2730-3_12
657 Condit, R. (1998b). *Tropical Forest Census Plots: Methods and Results from Barro Colorado*
658 *Island, Panama and a Comparison with Other Plots* (1st ed.). Springer-Verlag Berlin
659 Heidelberg, Landes Bioscience, Georgetown, USA. 10.1007/978-3-662-03664-8

660 Condit, R., Engelbrecht, B. M. J., Pino, D., Pérez, R., & Turner, B. L. (2013). *Species*
661 *distributions in response to individual soil nutrients and seasonal drought across a*
662 *community of tropical trees*. 5.
663 Condit, R., Pérez, R., Aguilar, S., & Lao, S. (2022). Census data from 65 tree plots in Panama
664 (1994-2015) [Dataset]. *Dryad*. https://doi.org/10.15146/mdpr-pm59
665 Connell, J. H. (1971). On the role of natural enemies in preventing competitive exclusion in
666 some marine animals and in rain forest trees. *Dynamics of Populations*, 298–312.
667 Dalling, J. W., Davis, A. S., Schutte, B. J., & Elizabeth Arnold, A. (2011). Seed survival in soil:
668 Interacting effects of predation, dormancy and the soil microbial community: Seed
669 survival in soil. *Journal of Ecology*, *99*(1), 89–95. https://doi.org/10.1111/j.1365-
670 2745.2010.01739.x
671 Davison, A. C., & Hinkley, D. V. (1997). *Bootstrap Methods and their Application*. Cambridge
672 University Press. https://doi.org/10.1017/CBO9780511802843
673 Desprez-Loustau, M.-L., Marçais, B., Nageleisen, L.-M., Piou, D., & Vannini, A. (2006).
674 Interactive effects of drought and pathogens in forest trees. *Annals of Forest Science*,
675 *63*(6), 597–612. https://doi.org/10.1051/forest:2006040
676 Detto, M., Visser, M. D., Wright, S. J., & Pacala, S. W. (2019). Bias in the detection of negative
677 density dependence in plant communities. *Ecology Letters*, *22*(11), 1923–1939.
678 https://doi.org/10.1111/ele.13372
679 Domínguez-Begines, J., Ávila, J. M., García, L. V., & Gómez-Aparicio, L. (2021). Disentangling
680 the role of oomycete soil pathogens as drivers of plant–soil feedbacks. *Ecology*, *102*(8).
681 https://doi.org/10.1002/ecy.3430

682 Eck, J. L., Stump, S. M., Delavaux, C. S., Mangan, S. A., & Comita, L. S. (2019). Evidence of
683 within-species specialization by soil microbes and the implications for plant community
684 diversity. *Proceedings of the National Academy of Sciences*, 201810767.
685 https://doi.org/10.1073/pnas.1810767116
686 Effiom, E. O., Nuñez-Iturri, G., Smith, H. G., Ottosson, U., & Olsson, O. (2013). Bushmeat
687 hunting changes regeneration of African rainforests. *Proceedings of the Royal Society B:*
688 *Biological Sciences*, *280*(1759), 20130246. https://doi.org/10.1098/rspb.2013.0246
689 Engelbrecht, B. M. J., Comita, L. S., Condit, R., Kursar, T. A., Tyree, M. T., Turner, B. L., &
690 Hubbell, S. P. (2007). Drought sensitivity shapes species distribution patterns in tropical
691 forests. *Nature*, *447*(7140), 80–82. https://doi.org/10.1038/nature05747
692 Engelbrecht, B. M. J., & Kursar, T. A. (2003). Comparative drought-resistance of seedlings of 28
693 species of co-occurring tropical woody plants. *Oecologia*, *136*(3), 383–393.
694 https://doi.org/10.1007/s00442-003-1290-8
695 Fine, P. V. A., Miller, Z. J., Mesones, I., Irazuzta, S., Appel, H. M., Stevens, M. H. H.,
696 Sääksjärvi, I., Schultz, J. C., & Coley, P. D. (2006). The growth-defense trade-off and
697 habitat specialization by plants in Amazonian forests. *Ecology*, *87*(sp7), S150–S162.
698 https://doi.org/10.1890/0012-9658(2006)87[150:TGTAHS]2.0.CO;2
699 Fricke, E. C., Simon, M. J., Reagan, K. M., Levey, D. J., Riffell, J. A., Tomás A. Carlo, &
700 Tewksbury, J. J. (2013). When condition trumps location: Seed consumption by fruit-
701 eating birds removes pathogens and predator attractants. *Ecology Letters*, *16*(8), 1031–
702 1036. https://doi.org/10.1111/ele.12134

García-Guzmán, G., & Dirzo, R. (2001). Patterns of leaf-pathogen infection in the understory of a Mexican rain forest: Incidence, spatiotemporal variation, and mechanisms of infection. *American Journal of Botany*, *88*(4), 634–645. https://doi.org/10.2307/2657063

Garrett, S. D. (1970). *Pathogenic root-infecting fungi*. Cambridge University Press.

Garwood, N. (2009). *Seedlings of Barro Colorado Island and the Neotropics*. Cornell University Press, Ithaca.

Garwood, N. C. (1983). Seed Germination in a Seasonal Tropical Forest in Panama: A Community Study. *Ecological Monographs*, *53*(2), 159–181. https://doi.org/10.2307/1942493

Gaviria, J., & Engelbrecht, B. M. J. (2015). Effects of Drought, Pest Pressure and Light Availability on Seedling Establishment and Growth: Their Role for Distribution of Tree Species across a Tropical Rainfall Gradient. *PLOS ONE*, *10*(11), e0143955. https://doi.org/10.1371/journal.pone.0143955

Gaviria, J., Turner, B. L., & Engelbrecht, B. M. J. (2017). Drivers of tree species distribution across a tropical rainfall gradient. *Ecosphere*, *8*(2), e01712. https://doi.org/10.1002/ecs2.1712

Ghanbary, E., Fathizadeh, O., Pazhouhan, I., Zarafshar, M., Tabari, M., Jafarnia, S., Parad, G. A., & Bader, M. K.-F. (2021). Drought and Pathogen Effects on Survival, Leaf Physiology, Oxidative Damage, and Defense in Two Middle Eastern Oak Species. *Forests*, *12*(2), 247. https://doi.org/10.3390/f12020247

Ghanbary, E., Tabari Kouchaksaraei, M., Mirabolfathy, M., Modarres Sanavi, S. A. M., & Rahaie, M. (2017). Growth and physiological responses of *Quercus brantii* seedlings

inoculated with *Biscogniauxia mediterranea* and *Obolarina persica* under drought stress. *Forest Pathology*, *47*(5), e12353. https://doi.org/10.1111/efp.12353

Gilbert, G. S., Foster, R. B., & Hubbell, S. P. (1994). Density and distance-to-adult effects of a canker disease of trees in a moist tropical forest. *Oecologia*, *98*(1), 100–108. https://doi.org/10.1007/BF00326095

Gilbert, G. S., Harms, K. E., Hamill, D. N., & Hubbell, S. P. (2001). Effects of seedling size, El Niño drought, seedling density, and distance to nearest conspecific adult on 6-year survival of Ocotea whitei seedlings in Panamá. *Oecologia*, *127*(4), 509–516. https://doi.org/10.1007/s004420000616

Gillett, J. B. (1962). Pest pressure, an underestimated factor in evolution. In D. Nichols (Ed.), *Taxonomy and geography* (pp. 37–46). The Systematics Association.

Givnish, T. J. (1999). On the causes of gradients in tropical tree diversity. *Journal of Ecology*, *87*(2), 193–210. https://doi.org/10.1046/j.1365-2745.1999.00333.x

Green, P. T., Harms, K. E., & Connell, J. H. (2014). Nonrandom, diversifying processes are disproportionately strong in the smallest size classes of a tropical forest. *Proceedings of the National Academy of Sciences*, *111*(52), 18649–18654. https://doi.org/10.1073/pnas.1321892112

Gripenberg, S., Rota, J., Kim, J., Wright, S. J., Garwood, N. C., Fricke, E. C., Zalamea, P., & Salminen, J. (2018). Seed polyphenols in a diverse tropical plant community. *Journal of Ecology*, *106*(1), 87–100. https://doi.org/10.1111/1365-2745.12814

Hazelwood, K., Beck, H., & Paine, C. E. T. (2021). Negative density dependence in the mortality and growth of tropical tree seedlings is strong, and primarily caused by fungal pathogens. *Journal of Ecology*, *109*(4), 1909–1918. https://doi.org/10.1111/1365-2745.13615

748 Howe, H. F., Schupp, E. W., & Westley, L. C. (1985). Early Consequences of Seed Dispersal for
749 a Neotropical Tree (Virola surinamensis). *Ecology*, *66*(3), 781–791.
750 https://doi.org/10.2307/1940539
751 Howe, H. F., & Smallwood, J. (1982). Ecology of Seed Dispersal. *Annual Review of Ecology*
752 *and Systematics*, *13*(1), 201–228. https://doi.org/10.1146/annurev.es.13.110182.001221
753 Hubbell, S. P. (1998). The maintenance of diversity in a neotropical tree community: Conceptual
754 issues, current evidence, and the challenges ahead. In F. Dallmeier & J. A. Comiskey
755 (Eds.), *Forest biodiversity, research, monitoring and modeling.* (Vol. 20, pp. 17–44).
756 UNESCO and Parthenon Publishing.
757 Hülsmann, L., Chisholm, R. A., & Hartig, F. (2021). Is Variation in Conspecific Negative
758 Density Dependence Driving Tree Diversity Patterns at Large Scales? *Trends in Ecology*
759 *& Evolution*, *36*(2), 151–163. https://doi.org/10.1016/j.tree.2020.10.003
760 Jactel, H., Petit, J., Desprez-Loustau, M.-L., Delzon, S., Piou, D., Battisti, A., & Koricheva, J.
761 (2012). Drought effects on damage by forest insects and pathogens: A meta-analysis.
762 *Global Change Biology*, *18*(1), 267–276. https://doi.org/10.1111/j.1365-
763 2486.2011.02512.x
764 Janzen, D. H. (1970). Herbivores and the Number of Tree Species in Tropical Forests. *The*
765 *American Naturalist*, *104*(940), 501–528.
766 Jauss, R.-T., Nowack, A., Walden, S., Wolf, R., Schaffer, S., Schellbach, B., Bonkowski, M., &
767 Schlegel, M. (2021). To the canopy and beyond: Air dispersal as a mechanism of
768 ubiquitous protistan pathogen assembly in tree canopies. *European Journal of*
769 *Protistology*, *80*, 125805. https://doi.org/10.1016/j.ejop.2021.125805

770 Kirtman, B., Power, S. B., Adedoyin, A. J., Boer, G. J., Bojariu, R., Camilloni, I., Doblas-Reyes,
771 F., Fiore, A. M., Kimoto, M., Meehl, G., Prather, M., Sarr, A., Schar, C., Sutton, R., van
772 Oldenborgh, G. J., Vecchi, G., & Wang, H.-J. (2013). Chapter 11 - Near-term climate
773 change: Projections and predictability. In IPCC (Ed.), *Climate Change 2013: The*
774 *Physical Science Basis. IPCC Working Group I Contribution to AR5*. Cambridge
775 University Press.
776 Krishnadas, M., Bagchi, R., Sridhara, S., & Comita, L. S. (2018). Weaker plant-enemy
777 interactions decrease tree seedling diversity with edge-effects in a fragmented tropical
778 forest. *Nature Communications*, *9*(1). https://doi.org/10.1038/s41467-018-06997-2
779 Kurten, E. L., Wright, S. J., & Carson, W. P. (2015). Hunting alters seedling functional trait
780 composition in a Neotropical forest. *Ecology*, *96*(7), 1923–1932.
781 https://doi.org/10.1890/14-1735.1
782 Lebrija-Trejos, E., Hernández, A., & Wright, S. J. (2023). Effects of moisture and density-
783 dependent interactions on tropical tree diversity. *Nature*, *615*(7950), 100–104.
784 https://doi.org/10.1038/s41586-023-05717-1
785 Liang, M., Liu, X., Gilbert, G. S., Zheng, Y., Luo, S., Huang, F., & Yu, S. (2016). Adult trees
786 cause density-dependent mortality in conspecific seedlings by regulating the frequency of
787 pathogenic soil fungi. *Ecology Letters*, *19*(12), 1448–1456.
788 https://doi.org/10.1111/ele.12694
789 Lind, E. M., Borer, E., Seabloom, E., Adler, P., Bakker, J. D., Blumenthal, D. M., Crawley, M.,
790 Davies, K., Firn, J., Gruner, D. S., Stanley Harpole, W., Hautier, Y., Hillebrand, H.,
791 Knops, J., Melbourne, B., Mortensen, B., Risch, A. C., Schuetz, M., Stevens, C., &
792 Wragg, P. D. (2013). Life-history constraints in grassland plant species: A growth-

defence trade-off is the norm. *Ecology Letters*, *16*(4), 513–521. https://doi.org/10.1111/ele.12078

Lintner, B. R., Biasutti, M., Diffenbaugh, N. S., Lee, J., Niznik, M., & Findell, K. L. (2012). Amplification of wet and dry month occurrence over tropical land regions in response to global warming. *Journal of Geophysical Research: Atmospheres*, *117*(D11), 2012JD017499. https://doi.org/10.1029/2012JD017499

Liu, X., Parker, I. M., Gilbert, G. S., Lu, Y., Xiao, Y., Zhang, L., Huang, M., Cheng, Y., Zhang, Z., & Zhou, S. (2022). Coexistence is stabilized by conspecific negative density dependence via fungal pathogens more than oomycete pathogens. *Ecology*, *103*(12). https://doi.org/10.1002/ecy.3841

Liu, Y., Fang, S., Chesson, P., & He, F. (2015). The effect of soil-borne pathogens depends on the abundance of host tree species. *Nature Communications*, *6*(1), 10017. https://doi.org/10.1038/ncomms10017

Liu, Y., & He, F. (2019). Incorporating the disease triangle framework for testing the effect of soil-borne pathogens on tree species diversity. *Functional Ecology*, *33*(7), 1211–1222. https://doi.org/10.1111/1365-2435.13345

Mangan, S. A., Schnitzer, S. A., Herre, E. A., Mack, K. M. L., Valencia, M. C., Sanchez, E. I., & Bever, J. D. (2010). Negative plant–soil feedback predicts tree-species relative abundance in a tropical forest. *Nature*, *466*(7307), 752–755. https://doi.org/10.1038/nature09273

Marchand, P., Comita, L. S., Wright, S. J., Condit, R., Hubbell, S. P., & Beckman, N. G. (2020). Seed-to-seedling transitions exhibit distance-dependent mortality but no strong spacing effects in a Neotropical forest. *Ecology*, *101*(2), e02926. https://doi.org/10.1002/ecy.2926

Marden, J. H., Mangan, S. A., Peterson, M. P., Wafula, E., Fescemyer, H. W., Der, J. P., dePamphilis, C. W., & Comita, L. S. (2017). Ecological genomics of tropical trees: How local population size and allelic diversity of resistance genes relate to immune responses, cosusceptibility to pathogens, and negative density dependence. *Molecular Ecology*, *26*(9), 2498–2513. https://doi.org/10.1111/mec.13999

McCarthy-Neumann, S., & Kobe, R. K. (2008). Tolerance of soil pathogens co-varies with shade tolerance across species of tropical tree seedlings. *Ecology*, *89*(7), 1883–1892.

Milici, V. R., Dalui, D., Mickley, J. G., & Bagchi, R. (2020). Responses of plant–pathogen interactions to precipitation: Implications for tropical tree richness in a changing world. *Journal of Ecology*, *108*(5), 1800–1809. https://doi.org/10.1111/1365-2745.13373

Milici, V.R., Comita, L.S., & Bagchi, R. (2024). Foliar disease incidence in a tropical seedling community is density dependent and varies along a regional precipitation gradient [Data set]. In Journal of Ecology. Zenodo. https://doi.org/10.5281/zenodo.10456097

Milici, V.R. (2024). valeriemilici/PanamaSeedlingSurveys:PanamaSeedlingSurveyAnalysis (v1.0.0). Zenodo. https://doi.org/10.5281/zenodo.10456335

Mitchell, C. E., Tilman, D., & Groth, J. V. (2002). Effects of Grassland Plant Species Diversity, Abundance, and Composition on Foliar Fungal Disease. *Ecology*, *83*(6), 1713–1726. https://doi.org/10.1890/0012-9658(2002)083[1713:EOGPSD]2.0.CO;2

Muller-Landau, H. C., Wright, S. J., Calderón, O., Condit, R., & Hubbell, S. P. (2008). Interspecific variation in primary seed dispersal in a tropical forest. *Journal of Ecology*, *96*(4), 653–667. https://doi.org/10.1111/j.1365-2745.2008.01399.x

836 Murphy, S. J., Wiegand, T., & Comita, L. S. (2017). Distance-dependent seedling mortality and
837 long-term spacing dynamics in a neotropical forest community. *Ecology Letters*, *20*(11),
838 1469–1478. https://doi.org/10.1111/ele.12856
839 Nathan, R., & Muller-Landau, H. C. (2000). Spatial patterns of seed dispersal, their determinants
840 and consequences for recruitment. *Trends in Ecology & Evolution*, *15*(7), 278–285.
841 https://doi.org/10.1016/S0169-5347(00)01874-7
842 Oliveira, T. S. D., & Bell, R. W. (Eds.). (2022). *Subsoil Constraints for Crop Production*.
843 Springer International Publishing. https://doi.org/10.1007/978-3-031-00317-2
844 Packer, A., & Clay, K. (2000). Soil pathogens and spatial patterns of seedling mortality in a
845 temperate tree. *Nature*, *404*(6775), 278–281. https://doi.org/10.1038/35005072
846 Piepenbring, M., Hofmann, T. A., Kirschner, R., Mangelsdorff, R., Perdomo, O., Justavino, D.
847 R., & Trampe, T. (2011). Diversity patterns of neotropical plant parasitic microfungi.
848 *Ecotropica*, *17*, 27–40.
849 Piepenbring, M., Hofmann, T. A., Miranda, E., Cáceres, O., & Unterseher, M. (2015). Leaf
850 shedding and weather in tropical dry-seasonal forest shape the phenology of fungi –
851 Lessons from two years of monthly surveys in southwestern Panama. *Fungal Ecology*,
852 *18*, 83–92. https://doi.org/10.1016/j.funeco.2015.08.004
853 Pringle, E. G., Álvarez-Loayza, P., & Terborgh, J. (2007). Seed characteristics and susceptibility
854 to pathogen attack in tree seeds of the Peruvian Amazon. *Plant Ecology*, *193*(2), 211–
855 222. https://doi.org/10.1007/s11258-006-9259-4
856 Richards, P. W., Walsh, R. P. D., Baillie, I. C., & Greig-Smith, P. (1996). *The tropical*
857 *rainforest: An ecological study* (2nd ed.). Cambridge University Press.

Rottstock, T., Joshi, J., Kummer, V., & Fischer, M. (2014). Higher plant diversity promotes higher diversity of fungal pathogens, while it decreases pathogen infection per plant. *Ecology*, *95*(7), 1907–1917. https://doi.org/10.1890/13-2317.1

Rozendaal, D. M. A., Brienen, R. J. W., Soliz-Gamboa, C. C., & Zuidema, P. A. (2010). Tropical tree rings reveal preferential survival of fast-growing juveniles and increased juvenile growth rates over time. *New Phytologist*, *185*(3), 759–769. https://doi.org/10.1111/j.1469-8137.2009.03109.x

Russo, S. E. (2005). Linking seed fate to natural dispersal patterns: Factors affecting predation and scatter-hoarding of Virola calophylla seeds in Peru. *Journal of Tropical Ecology*, *21*(3), 243–253. https://doi.org/10.1017/S0266467405002312

Schoeneweiss, D. F. (1975). Predisposition, stress, and plant disease. *Annual Review of Phytopathology*, *13*(1), 193–211.

Sinha, R., Irulappan, V., Mohan-Raju, B., Suganthi, A., & Senthil-Kumar, M. (2019). Impact of drought stress on simultaneously occurring pathogen infection in field-grown chickpea. *Scientific Reports*, *9*(1), 5577. https://doi.org/10.1038/s41598-019-41463-z

Slippers, B., & Wingfield, M. J. (2007). Botryosphaeriaceae as endophytes and latent pathogens of woody plants: Diversity, ecology and impact. *Fungal Biology Reviews*, *21*(2–3), 90–106. https://doi.org/10.1016/j.fbr.2007.06.002

Spear, E. R. (2017). Phylogenetic relationships and spatial distributions of putative fungal pathogens of seedlings across a rainfall gradient in Panama. *Fungal Ecology*, *26*, 65–73. https://doi.org/10.1016/j.funeco.2016.12.004

879 Spear, E. R., & Broders, K. D. (2021). Host-generalist fungal pathogens of seedlings may
880 maintain forest diversity via host-specific impacts and differential susceptibility among
881 tree species. *New Phytologist*, *231*(1), 460–474. https://doi.org/10.1111/nph.17379
882 Spear, E. R., Coley, P. D., & Kursar, T. A. (2015). Do pathogens limit the distributions of
883 tropical trees across a rainfall gradient? *Journal of Ecology*, *103*(1), 165–174.
884 https://doi.org/10.1111/1365-2745.12339
885 Sugiyama, A., Comita, L. S., Masaki, T., Condit, R., & Hubbell, S. P. (2018). Resolving the
886 paradox of clumped seed dispersal: Positive density and distance dependence in a bat-
887 dispersed species. *Ecology*, *0*(0). https://doi.org/10.1002/ecy.2512
888 Swinfield, T., Lewis, O. T., Bagchi, R., & Freckleton, R. P. (2012). Consequences of changing
889 rainfall for fungal pathogen-induced mortality in tropical tree seedlings: Fungal
890 Pathogen-Induced Mortality in Tropical Tree Seedlings. *Ecology and Evolution*, *2*(7),
891 1408–1413. https://doi.org/10.1002/ece3.252
892 Terborgh, J. (2020). At 50, Janzen–Connell Has Come of Age. *BioScience*, *70*(12), 1082–1092.
893 https://doi.org/10.1093/biosci/biaa110
894 Terborgh, J., Nuñez-Iturri, G., Pitman, N. C. A., Valverde, F. H. C., Alvarez, P., Swamy, V.,
895 Pringle, E. G., & Paine, C. E. T. (2008). Tree recruitment in an empty forest. *Ecology*,
896 *89*(6), 1757–1768. https://doi.org/10.1890/07-0479.1
897 Thompson, S., Alvarez-Loayza, P., Terborgh, J., & Katul, G. (2010). The effects of plant
898 pathogens on tree recruitment in the Western Amazon under a projected future climate: A
899 dynamical systems analysis. *Journal of Ecology*, *98*(6), 1434–1446.

Velásquez, A. C., Castroverde, C. D. M., & He, S. Y. (2018). Plant–Pathogen Warfare under Changing Climate Conditions. *Current Biology*, *28*(10), R619–R634. https://doi.org/10.1016/j.cub.2018.03.054

Visser, M. D., Schnitzer, S. A., Muller-Landau, H. C., Jongejans, E., de Kroon, H., Comita, L. S., Hubbell, S. P., & Wright, S. J. (2018). Tree species vary widely in their tolerance for liana infestation: A case study of differential host response to generalist parasites. *Journal of Ecology*, *106*(2), 781–794. https://doi.org/10.1111/1365-2745.12815

Webb, C. O., & Peart, D. R. (1999). Seedling density dependence promotes coexistence of Bornean rain forest trees. *Ecology*, *80*(6), 2006–2017. https://doi.org/10.1890/0012-9658(1999)080[2006:SDDPCO]2.0.CO;2

Weissflog, A., Markesteijn, L., Lewis, O. T., Comita, L. S., & Engelbrecht, B. M. J. (2017). Contrasting patterns of insect herbivory and predation pressure across a tropical rainfall gradient. *Biotropica*. https://doi.org/10.1111/btp.12513

Wright, S. J., Calderón, O., Hernandéz, A., Detto, M., & Jansen, P. A. (2016). Interspecific associations in seed arrival and seedling recruitment in a Neotropical forest. *Ecology*, *97*(10), 2780–2790. https://doi.org/10.1002/ecy.1519

Wright, S. J., Kitajima, K., Kraft, N. J., Reich, P. B., Wright, I. J., Bunker, D. E., Condit, R., Dalling, J. W., Davies, S. J., & Diaz, S. (2010). Functional traits and the growth–mortality trade-off in tropical trees. *Ecology*, *91*(12), 3664–3674.