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Do pathogens limit the distributions of tropical trees across a rainfall gradient?

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Summary

1. Organisms are adapted to particular habitats; consequently, community composition changes across environmental gradients, enhancing regional diversity. In Panama, a rainfall gradient correlates with the spatial turnover of tree species. While strong evidence suggests that tree species common in the wetter forests are excluded from the drier forests by seasonal drought, the factor(s) excluding drought-tolerant species, common in the drier forests, from the wetter forests remain ambiguous.

2. Here, we show that seedlings were significantly more likely to suffer pathogen-caused damage and mortality in the wetter forest. While seedlings of dry- and wet-forest species were equally likely to suffer pathogen attack, seedlings of dry-forest species were significantly more likely to die when attacked and tended to suffer more pathogen-caused mortality overall. Furthermore, seedlings of dry-forest species suffered pathogen-caused mortality in the forest in which they do not naturally occur and in which conspecific and/or congeneric adults are absent or rare, indicating that some pathogens are relatively widespread and/or are capable of damaging multiple host species.

3. *Synthesis.* Elevated risk of pathogen-caused damage and mortality in the wetter forests and a greater impact to host fitness from pathogen attack for seedlings of dry-forest species suggest that pathogens may enhance regional forest diversity by contributing to changes in tree species composition via the exclusion of dry-forest tree species from the wetter forests. This study highlights a potentially widespread and under explored mechanism by which pathogens shape plant communities at the landscape scale. An understanding of how species' distributions are shaped by the interplay between abiotic and biotic factors is essential for conservation biology.

Key-words: determinants of plant community diversity and structure, plant disease ecology, plant ranges, plant-pathogen interactions, precipitation gradient, regional forest diversity, seedling mortality, seedling recruitment, seedlings, tropical forest

Introduction

Biodiversity is not distributed randomly in space and a central goal of ecology is to identify these distribution patterns and their underlying processes. This is particularly relevant as global climate change reshapes the biogeographies of living organisms. Adaptations to local conditions and ecological sorting lead to the spatial turnover of species (beta-diversity) across environmental gradients, thereby enhancing regional diversity (Leigh *et al.* 2004). A classic ecological paradigm predicts that, across an environmental gradient, geographical range limits are determined by abiotic conditions at one end and by biotic pressures at the other (MacArthur 1972). Rainfall gradients in the tropics have been correlated with the turnover of plant species in space and species distributions (Veenendaal & Swaine 1998; Pyke *et al.* 2001; Baltzer *et al.* 2008). While the Isthmus of Panama is only 60 km wide, annual rainfall on the Atlantic coast is almost double that on the Pacific coast and correlates with a near-complete turnover in tree species composition (Pyke *et al.* 2001; Condit *et al.* 2002). Tree species common in the wetter forests are excluded from the drier forests by greater drought sensitivity (Engelbrecht *et al.* 2007; Brenes-Arguedas, Coley & Kursar 2009). However, it is less clear what filtering mechanism(s) exclude(s) drought-tolerant tree species common in the drier forests from the wetter forests. Here, we explored the possibility that a biotic pressure might act as such a filter.

A longstanding hypothesis predicts that pressure from plant pests, such as insects and pathogens, correlates positively with precipitation and is elevated in aseasonal forests due to lessened abiotic constraints on pest survival and reproduction (Leigh *et al.* 2004; Gilbert 2005). By extension, plant species adapted

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to living in wetter less seasonal areas may be under selective pressure to be better defended against pests (Coley & Barone 1996). Elevated pest pressure in the wetter forests could contribute to the regional turnover of tree species via the selective exclusion of poorly defended plant species. Here, we focus on plant pathogens, which are ubiquitous, diverse and have impacts that vary among plant species, making them an important structuring force in natural plant communities (Gilbert 2005). Pathogen attack is a major cause of mortality for natural seedling communities (Moles & Westoby 2004; Gilbert 2005; Mangan *et al.* 2010; Alvarez-Loayza & Terborgh 2011). We focus on the seedling stage because seedling mortality can have long-lasting effects on plant distributions, relative abundances and community composition (Comita *et al.* 2010; Mangan *et al.* 2010; Salk *et al.* 2011). Despite being a brief period of the life cycle, particularly for long-lived trees, the seedling stage represents a period of high mortality (Gilbert 2005) and strong selective pressures.

We hypothesized that, in Panama, elevated pathogen pressure in the wetter Atlantic forests acts as a filter excluding tree species typical of the drier, Pacific forests by limiting their seedling recruitment. Herein, we intend to convey a negative impact to plants when we refer to pathogen pressure. Based on our central hypothesis, we predicted that (i) there would be a greater risk of pathogen-caused damage and mortality in the wetter forest than in the drier forest regardless of tree species distribution and that (ii) seedlings of dry-forest species would be more vulnerable to pathogen attack than wet-forest species in both forests (no forest by distribution interaction). For pathogens to act as a filter in the wetter forests, seedlings of dry-forest species only need to suffer a greater impact from pathogens than seedlings of wet-forest species in the wetter forests. An alternative hypothesis is that dry-forest tree species are adapted to the pathogens that they commonly encounter in the drier forests; thus, in the drier forests, their seedlings are less impacted by pathogens than the seedlings of wet-forest species. To test these hypotheses, we established common gardens in the wetter and drier forests of central Panama (Fig. S1 in Supporting Information). We monitored seedlings of wet- and dry-forest tree species (Table S1) for pathogen-caused damage, seedling mortality and cause of death. Specifically, we examined the relative likelihood of pathogen-caused damage and mortality in the wetter versus drier forest and assessed if seedlings of dry-forest species were more likely to suffer pathogen attack than wet-forest species. Furthermore, we monitored if pathogen attack led to seedling death to evaluate if dry- and wet-forest species differ in their resistance to or tolerance of pathogen attack. The common gardens also allowed us to gauge if and to what extent seedlings experienced release from pathogen pressure when planted in a forest in which they do not naturally occur and in which conspecific and/or congeneric adults are absent or rare.

Materials and methods

STUDY SITES AND SPECIES

Common gardens were planted in two lowland forest sites in central Panama (Fig. S1 a,b). Our wetter forest site is located near the Atlantic coast in Santa Rita Arriba (SRA) (9°20′03.71″ N, 79°46′39.96″ W, elev 200–250 m). SRA receives ≥3000 mm of rain year^{-1} with a dry season of ca. 67 days (Santiago *et al.* 2004). Our wetter forest site, located on private property (ca. 32 ha), is mixed-age and evergreen. Our drier forest site is located near the Pacific coast in Parque Natural Metropolitano (PNM) (8°59′36.62″ N, 79°32′36.17″ W, elev 50–95 m). PNM receives ≤1800 mm of rain year^{-1} with a dry season of ca. 129 days (Santiago *et al.* 2004). PNM's forest (ca. 232 ha) is mixed-age and semi-deciduous. Based on a transect and an informal survey, our wetter forest site is considerably more diverse than our drier forest site. No formal, forest inventory plot has been established in our wetter forest site; however, two 1-ha plots located in the forests of SRA had a mean tree species richness of 162 (≥10 cm dbh) (Condit *et al.* 2005). In contrast, only 36 tree species (≥10 cm dbh) were documented in a 1-ha forest inventory plot in PNM (Santiago *et al.* 2004).

We tested 12 tree species, representing nine families. The tree species were categorized as having either a wet- or dry-forest distribution based on their presence and/or abundance in the wetter versus drier forests (Condit, Pérez & Daguerre 2011) (Table S1). The dry- and wet-forest species that we tested are distributed over the phylogeny with no clear phylogenetic separation (Fig. S2). Based on previously published classifications and indices, we assigned each tree species to a shade-tolerance guild (LD = light demanding, IST = intermediate shade tolerance, ST = shade tolerant or some intermediary; Table S1). Our classifications are for the seedling stage as that is the focal life stage in our study and because light requirements often change with ontogeny. Tree species fall along a continuum of shade tolerances and, while some tree species can be clearly assigned to a specific shade-tolerance guild, many have intermediate shade tolerances and their classification is less straightforward (Wright *et al.* 2003). Both the dry- and wet-forest species used in our study represent a range of shade tolerances, and none of the species is considered to be a pioneer (Fig. S2; Table S1). To compare the mean shade tolerance of the seedlings of our dry- versus wet-forest species, we assigned a numerical value to each shade-tolerance guild represented by our tree species (LD – IST = 1, IST = 2, ST = 3). For the tree species tested in our experiment, there was no difference in the degree of shade tolerance for tree species typical of dry (M = 2.33, SD = 1.03) and wet forests (M = 2.5, SD = 0.84) (Wilcoxon rank sum test: $W = 17$, $P = 0.923$). No difference in shade tolerance for dry- versus wet-forest plant species is consistent with the results of other Panama-based studies (Engelbrecht *et al.* 2007; Brenes-Arguedas *et al.* 2011).

Five of the six dry-forest tree species and none of the wet-forest tree species have been observed in our drier forest site (PNM) (Condit *et al.* 2013; Smithsonian Tropical Research Institute; Table S1). Due to the low diversity of the drier forest, conspecific adult trees of three of our six dry-forest tree species, *Anacardium excelsum, Castilla elastica* and *Cojoba rufescens,* were present and abundant near our common gardens in PNM (E. Spear, pers. obs.). *Anacardium excelsum* and *C. elastica* are two of the dominant tree species in PNM, representing 13% and 11%, respectively, of the 318 trees (≥10 cm dbh) documented in a 1-ha forest inventory plot (Smithsonian Tropical Research Institute). Three of the six wet-forest tree species and none of the dry-forest tree species have been observed in our wetter forest site (Condit *et al.* 2013; Table S1). No conspecific adults of the wet-forest tree species were observed near our common gardens in either forest.

COMMON GARDEN EXPERIMENT

We established 30 common gardens in each forest. The locations of the common gardens were haphazardly selected along a ~0.5 km path and the locations represented a variety of understorey light environ-

ments based on subjective estimates. Seedlings were covered by hardware-wire exclosures (0. 6 m tall, 1 m^2; Fig. S1c) to minimize deaths from vertebrates and falling debris. Vegetation <12 cm in height and leaf litter were left in the exclosures. Leaf litter was cleared from the top of each exclosure weekly to avoid unnatural shading.

Recently emerged seedlings are particularly vulnerable to pathogen attack (Fig. S3; Augspurger 1983; Agrios 2005). To study this vulnerable developmental stage, seeds were planted directly in the forest. Seeds were collected in the forests bordering the Panama Canal from late May through the beginning of September 2010 (Table S1). Planting seeds rather than seedlings also allows for surface sterilization (2 min in 70% ethanol, 2 min in 10% commercial bleach and 2 min in 70% ethanol; following Meyer *et al.* 2008) before planting and ensures similar ontogenetic stages across species. Surface-sterilized seeds were planted as soon as possible after collection to maximize germination success by minimizing storage time (see Table S2 for species-specific planting dates). We planted the seeds of a given tree species in our drier and wetter forest sites in the same week and, to the best of our ability, on two consecutive dates (i.e. seeds of that species were planted in our drier forest site in one day and in our wetter forest site in the following day).

Because fruiting times differed among species and seeds were planted as they were collected, seeds of different species were planted at different times and, for five of the 12 tree species, seeds were planted on multiple dates (see Table S2 for additional details). Whenever possible, we planted wet- and dry-forest tree species concurrently (on the same dates) and, in fact, there is no difference in the mean week planted for the wet- versus dry-forest species tested (Wilcoxon rank sum test: $W = 15.5$, $P = 0.746$). Additionally, there is no difference in the median week germinated for wet- versus dry-forest species (Wilcoxon rank sum test: $W = 8.5$, $P = 0.148$). Furthermore, because of the spread of germination times for a given species, seeds planted earlier in the experiment often germinated at the same time as seeds planted later in the experiment (Table S2).

Seeds were planted just below the soil surface at haphazard locations within the exclosures, and the location of each seed was marked. Seed availability varied among species and, as possible, we planted multiple seeds per species in each common garden to maximize the number of seedlings (i.e. sample sizes; Table S2). We were only able to collect 37 seeds of *Carapa guianensis*, so seeds were planted in a random subset of the common gardens in both forest sites (18 of the 30 gardens in our drier forest site and 19 of the 30 gardens in our wetter forest site). Similarly, if the number of seeds collected was greater than a multiple of 60 (30 gardens per forest*2 forests), the extra seeds were planted in a random subset of common gardens in both forests (Table S2). Due to varied seed availability and varied germination success, the number of seedlings per species often varied among the common gardens and some gardens lacked seedlings of a certain species (Table S2). For all common gardens, total seedling density and the density of conspecific seedlings were at or below natural densities (Table S3). For both forest sites, we determined the natural density of all seedling-sized plants and the most abundant morphospecies by establishing quadrats (1 m^2) adjacent to 15 common gardens per forest.

Our study was conducted during the rainy season (Jun–Nov 2010) because we were specifically interested in investigating how pathogen-caused damage and mortality impact seedling establishment and we wanted to limit seedling deaths due to extraneous factors, including drought. Furthermore, previous studies have established that seasonal drought excludes wet-forest plant species from the drier forests (Engelbrecht *et al.* 2007; Brenes-Arguedas, Coley & Kursar 2009); therefore, that was not an objective of this study.

OBSERVATIONAL CENSUSES

Germination, pathogen-caused damage, seedling mortality and cause of mortality were recorded during weekly surveys ($n = 21$) and during the final harvests (Oct 24–Nov 16, 2010). Short census intervals were essential for accurately identifying the cause of death because pathogen infection can progress from initial symptoms to seedling decomposition within a week (Figs 1a,b and S4; Augspurger 1983). Furthermore, we were interested in tracking the appearance of pathogen-caused damage and the fitness impact of that damage (i.e. if pathogen attack did or did not result in death). Seedling mortality was categorized as pathogen, herbivore, missing or unknown. Mortality was categorized as unknown if the seedling was found dead with no prior notes about its condition and the cause of death was not immediately apparent. A subset of symptomatic seedlings were harvested to culture the putative fungal pathogen(s). The methods and analyses of the cultures will be reported separately. The final harvests were staggered by species relative to germination times. The number of censuses varied among species because of differences in seed availability, seed germination and final harvest dates. For some species, the number of censuses varied between forests (e.g. Ga, Fig. 2 a,b) because seeds of the species germinated earlier in one forest than the other.

DATA ANALYSIS

All statistical analyses were performed in R v. 3.0.2 (R Development Core Team 2013). The experimental design included two fixed effects: forest type (drier or wetter) and tree species distribution (dry- or wet-forest) and two random effects: species identity (12 species) and location within a given forest (30 per forest). The number of seedlings per species varied widely and for two species, *Carapa guianensis* and *Pourouma bicolor*, there were five or less seedlings per forest (Table S2). Unless otherwise noted, all species were retained in the statistical analyses because species were grouped according to their distribution for the desired comparisons, the variation associated with differences among species was partitioned into the random effect 'species identity', and the inclusion of *C. guianensis* and *P. bicolor* did not qualitatively change the results. Both fixed effects were retained in all models because the comparisons were planned and both random effects were included in all models because they were part of the study design. Our dependent variables were risk of pathogen-caused mortality, likelihood of pathogen-caused damage and likelihood of pathogen-caused death given that a seedling suffered pathogen-caused damage.

Since a forest by distribution interaction is not necessary for our central hypothesis and because those forest by distribution interactions which were marginally significant or significant did not support the alternative hypothesis presented in the introduction (see Results and Discussion), the forest by distribution interaction term was dropped and all subsequent models included only the main effects of forest (ignoring distribution) and of distribution (ignoring forest). To explore how one predictor variable modified the effect of the other, pairwise contrasts of interest were tested using the 'glht' function in the 'multcomp' package (Hothorn, Bretz & Westfall 2008). The *P*-values were not corrected for multiple comparisons because the comparisons were planned in an experimental context (Quinn & Keough 2002).

Nonparametric Wilcoxon rank sum tests were used to compare the shade tolerance, mean week planted and median week germinated of dry- versus wet-forest tree species. Based on Levene tests, the homogeneity of variances assumption was met for all three comparisons (shade tolerance: $F = 0.094$, $P = 0.765$, mean week planted: $F = 0.415$, $P = 0.534$ and median week germinated: $F = 0.114$, $P = 0.743$).

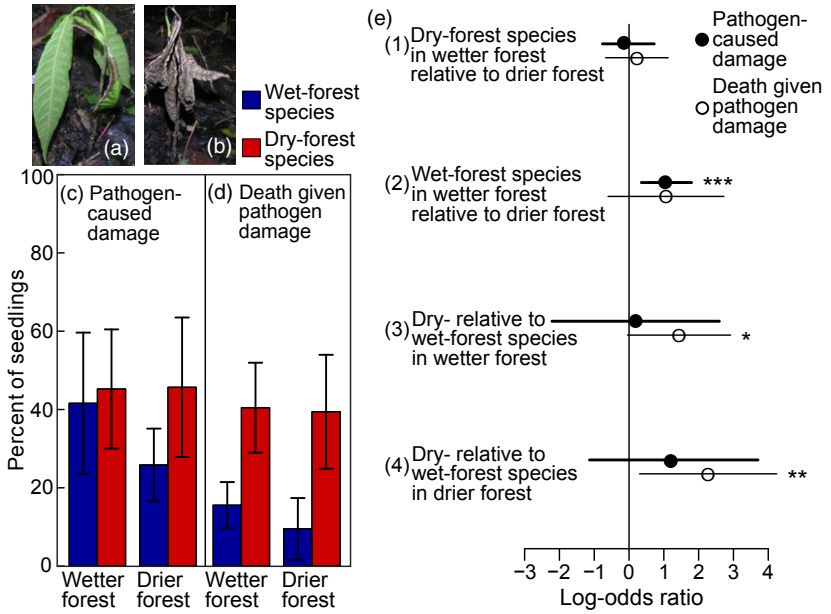

Fig. 1. Photos of a seedling of *Anacardium excelsum* that (a) suffered pathogen-caused damage and (b) was dead within seven days. Percent of seedlings with (c) pathogen-caused damage and (d) for which damage was lethal. Percentages were calculated by averaging forest by species percentages (species with five or less seedlings per forest were excluded, which did not change the trends). Error bars denote one standard error. (e) Log-odds ratios and their 95% confidence intervals from the GLMMs for pathogen-caused damage (filled circles) and death given pathogen-caused damage (open circles) (Table S5a,b). While seedlings of dry-forest species were not significantly more likely to suffer pathogen-caused damage than seedlings of wet-forest species (panel c and filled circles 3 and 4 in panel e; Table 1a), they were significantly more likely to die if they suffered pathogen damage (panel d and open circles 3 and 4 in panel e; Table 1b). Asterisks identify significant effects (*$P \leq 0.05$, **$P \leq 0.01$, ***$P \leq 0.001$).

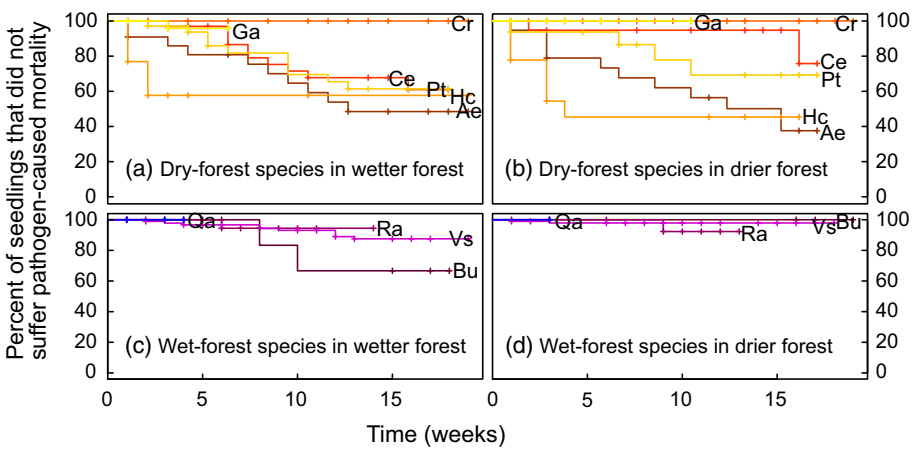

Fig. 2. Pathogen-caused mortality (Kaplan–Meier survivorship curves) for the six dry- (a, b) and four of the six wet-forest tree species (c, d) in the wetter (a, c) and drier (b, d) forests. Species codes are at the end of each curve (see Table S1 for full names). Survivorship curves were not plotted for the two species with five or less seedlings per forest. The curves include all seedlings with known start/stop dates, and tick marks indicate censored observations. Time varied among species because of differences in seed availability, germination and harvest dates. The number of censuses varied between forests for species that germinated earlier in one forest than the other (e.g. Ga in panels a and b).

A mixed-effects Cox proportional hazards model [‘coxme’ function, ‘coxme’ package (Therneau 2012)] was used to analyse pathogen-caused mortality (n = 630 seedlings with known germination and last observed dates) because this approach can partition out the variance attributable to differences among species and locations within a given forest via the use of random effects and because it can include right-censored data (e.g. seedlings alive at the experiment’s completion, killed by something other than a pathogen or harvested for pathogen isolation). Right-censored data are informative because we know that they did not suffer pathogen-caused mortality before the last census in which they were observed alive (i.e. the time of pathogen-caused mortality would have been at least greater than the time that the seedling was last observed); thus, these data contribute to the survivorship curves and estimates of risk of pathogen-caused mortality. Furthermore, accounting for seedlings lost from the study (e.g. seedlings for which cause of death could not be assigned or that went

Table 1. Log-odds ratios and 95% confidence intervals estimated by generalized linear mixed models (GLMMs) considering the main effects of forest type and tree species distribution for (a) pathogen-caused damage (n = 725) and (b) pathogen-caused mortality given pathogen damage (n = 272). Both models included the random effects 'species identity' and 'location within a given forest.' The intercept values for the two GLMMs are (a) pathogen-caused damage = −1.31 (−2.72, 0.1) and (b) death given pathogen-caused damage = −2.27 (−3.28, −1.25). The intercepts represent the average response at the baseline conditions (forest type: drier, tree species distribution: wet) and the log-odds ratios represent the effects of the alternative conditions relative to the baseline conditions. Positive log-odds values indicate a positive relationship between the likelihood of the outcome (e.g. pathogen-caused damage) and the predictor variable (e.g. forest type) and *vice versa*

	(a) Pathogen-caused damage	(b) Death given pathogen-caused damage
Main effects		
Forest type (wetter: drier)	0.55 (0.09, 1.01)*	0.44 (−0.17, 1.06) NS
Tree distribution (dry: wet)	0.69 (−1.23, 2.61) NS	1.69 (0.58, 2.8)**
Random effects		
Species identity	Var = 2.6, SD = 1.61	Var = 0.34, SD = 0.58
Location in a given forest	Var = 0.23, SD = 0.48	Var < 0.001, SD < 0.001

NS, Not significant
*$P \leq 0.05$, **$P \leq 0.01$

missing) is necessary to avoid biased results. Cox proportional hazards models ['coxph' function, 'survival' package (Therneau 2013)], without random effects, were used to plot survival. The proportional hazards assumption was met for both variables [forest type: $r = 0.137$, $\chi^2 = 1.513$, $P = 0.219$, tree species distribution: $r = -0.092$, $\chi^2 = 0.654$, $P = 0.419$; 'cox.zph' function, 'survival' package (Therneau 2013)]. Results are reported as hazard ratios (HR). An HR greater than one indicates an increased hazard of pathogen-caused mortality, and an HR less than one indicates a decreased hazard of pathogen-caused mortality.

Generalized linear mixed models [GLMMs, 'glmer' function, 'lme4' package (Bates *et al.* 2013)], assuming binomial error distributions and logit link functions, were used to analyse the proportion of seeds that germinated (n = 694; the response variable was the proportion of seeds per species per common garden that germinated; in total, 1960 seeds were planted), the proportion of seedlings that suffered pathogen-caused damage (n = 725; presence/absence, not indicative of severity) and, of the seedlings with pathogen-caused damage, the proportion that ultimately suffered pathogen-caused mortality (n = 272). Binary response GLMMs were preferable to time-to-event models for analysing pathogen-caused damage because below-ground infection was not observable until the final harvest so time to pathogen-caused damage could not be reliably modelled. The coefficients (β) estimated by the logistic regressions are the estimated relative changes in the log odds of an outcome (e.g. pathogen-caused damage) given a change in an independent variable (e.g. forest type). Negative log-odds values indicate a negative relationship between the likelihood of the outcome and the independent variable and *vice versa.* Log odds are plotted in Fig. 1 and reported in Tables 1 and S5a,b. For ease of interpretation, log odds were exponentiated to odds ratios (OR) in the main text and the legend of Table S2. An OR greater than one indicates greater odds and an OR less than one indicates lower odds.

Results

PATHOGENS WERE THE PRIMARY CAUSE OF SEEDLING MORTALITY IN BOTH FORESTS

Within the 21-week study period, 38% of 725 seedlings had observable damage that was characteristic of pathogens (e.g. foliar, stem and/or root necrosis, sunken lesions, collapse from stem necrosis or a slimy, waterlogged appearance; Agrios 2005) and 11% were obviously killed by pathogens. In some cases, the biotic disease agent was visible (e.g. mycelia). Among individual tree species, the proportion of seedlings with pathogen-caused damage ranged from 5% to 95% and the proportion killed by pathogens ranged from 0% to 51% (Table S4). Pathogens caused the majority of seedling deaths in both forests (in the drier forest: 8% of all seedlings were killed by pathogens; of seedling deaths, 56% were caused by pathogens, 10% were caused by herbivores, 30% were missing and 4% were unknown; in the wetter forest: 13% of all seedlings were killed by pathogens; of seedling deaths, 44% were caused by pathogens, 16% were caused by herbivores, 38% were missing and 2% were unknown). No seedlings were killed by large, vertebrate herbivores, falling debris or drought because seedlings were protected by wire exclosures and the study was conducted during the wet season.

PATHOGEN PRESSURE IS ELEVATED IN THE WETTER FOREST RELATIVE TO THE DRIER FOREST

A greater proportion of seedlings were damaged by pathogens in the wetter forest than in the drier forest (44% vs. 31%). Ignoring species distribution, seedlings were 74% more likely to suffer pathogen-caused damage in the wetter forest than in the drier forest (GLMM, $P = 0.018$; Table 1a). Similarly, seedlings were 65% more likely to suffer pathogen-caused mortality in the wetter forest (COXME, $P = 0.038$; Fig. 3; Table 2). For risk of pathogen-caused mortality, there was a marginally significant forest by distribution interaction (COXME, $P = 0.084$) and, for likelihood of pathogen-caused damage, there was a significant forest by distribution interaction (GLMM, $P = 0.005$). Pairwise contrasts exploring how the forest effect differs for wet- versus dry-forest tree species revealed that only wet-forest species are significantly more likely to suffer pathogen-caused damage and mortality in the wetter forest (Fig. 1e; Table S5a,c). On the whole, seedlings of dry-forest species suffered relatively high levels of pathogen-caused damage and mortality in both forests (Figs 1c, 2a,b and red lines in 3). In fact, seedlings of dry-forest species were at a greater risk of pathogen-caused mortality in the drier forest than seedlings of wet-forest species (Table S5c).

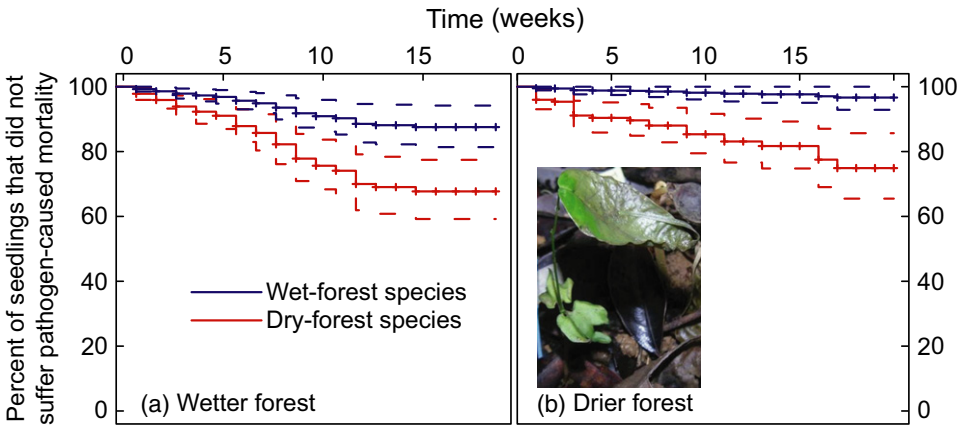

Fig. 3. Pathogen-caused mortality was greater (a) in the wetter forest than (b) in the drier forest and was greater for seedlings of dry-forest species (red lines) than for wet-forest species (blue lines). The survival curves (solid) and 95% confidence intervals (dashed) are the estimates from a Cox proportional hazards model (n = 630, including all seedlings with known start/stop dates, regardless of if symptomatic or not). Tick marks indicate censored observations. Only forest type is a significant predictor (COXME, P = 0.038; Table 2). Photo is a *Protium tenuifolium* seedling with pathogen-caused damage.

Table 2. Overall risk of pathogen-caused mortality based on a mixed-effects Cox proportional hazards model considering the main effects of forest type and tree species distribution (n = 630, including all seedlings with known start/stop dates, regardless of if symptomatic or not). Species identity and location within a given forest were included as random effects. A hazard ratio (HR) greater than one indicates increased hazard of pathogen-caused mortality, and an HR less than one indicates decreased hazard of pathogen-caused mortality

	HR	β	SE(β)	z-Value	P
Main effects					
Forest type (wetter: drier)	1.65	0.50	0.24	2.07	0.038
Tree distribution (dry: wet)	3.27	1.18	0.88	1.35	0.180
Random effects					
Species identity	Var = 1.40, SD = 1.18				
Location in a given forest	Var < 0.001, SD = 0.02				

WET- AND DRY-FOREST SPECIES ARE DIFFERENTIALLY IMPACTED BY PATHOGENS

In general, seedlings of dry-forest species tended to suffer more pathogen-caused mortality than seedlings of wet-forest species [ignoring forest type: 17% vs. 4% (not significant), in the wetter forest: 18% vs. 8% (not significant) and in the drier forest: 15% vs. 2% (P = 0.05)] (Figs 2 and 3; Tables 2 and S 5c). However, there was interspecific variation in the proportion of seedlings that suffered pathogen-caused mortality, with seedlings of two dry-forest species, *Genipa americana* and *Cojoba rufescens,* suffering minimal to no pathogen-caused mortality (Fig. 2a,b; Table S4). Consequently, tree species distribution was not a significant predictor of the risk of pathogen-caused mortality (Table 2).

Seedlings of dry-forest species tended to suffer more pathogen-caused damage than wet-forest species but not significantly so (Fig. 1c; Tables 1a and S5a). Yet, dry- and wet-forest species did significantly differ in their fitness impact from pathogen-caused damage (Fig. 1d; Tables 1b and S5b). Pathogen-caused damage was approximately five times more likely to be lethal for seedlings of dry-forest species than for wet-forest species (GLMM, P = 0.003; Table 1b). There was no forest by distribution interaction for likelihood of pathogen-caused death given pathogen-caused damage (GLMM, P = 0.277).

DRY-FOREST SPECIES EXPERIENCED LITTLE TO NO ESCAPE FROM PATHOGENS IN THE FOREST IN WHICH THEY DO NOT NATURALLY OCCUR

Seedlings of dry-forest species suffered relatively high levels of pathogen-caused damage and mortality in both the wetter and drier forests (Figs 1c, 2a,b and 3). In contrast, seedlings of wet-forest species suffered less pathogen-caused damage and minimal pathogen-caused mortality in our drier forest site (Figs 1c,e, 2d and 3b). In our wetter forest site, seedlings of two wet-forest species, *Virola surinamensis* and *Brosimum utile*, suffered moderate pathogen-caused mortality (Fig. 2c). Conspecific adults of five of the six dry-forest tree species that we tested have been observed in our drier forest site (Condit *et al.* 2013; Smithsonian Tropical Research Institute; Table S1) and three of those species were present and abundant near our common gardens in our drier forest site (E. Spear, pers. obs.). For two of the dry-forest species that suffered high pathogen-caused mortality in the drier forest, *Hymenaea courbaril* and *Protium tenuifolium* (Fig. 2b), no adults were observed near our gardens. While none of the dry-forest species have been observed in our wetter forest site (Condit *et al.* 2013; Table S1), four of the six dry-forest species suffered high seedling mortality (Fig. 2a). In terms of the wet-forest tree species that we tested, although at least one adult of *B. utile* has been observed at our wetter forest site (Condit *et al.* 2013) and congeneric adults of *V. surinamensis* were observed in the vicinity of our common gardens in our wetter forest site, no conspecific adults of the wet-forest species were observed near our common gardens in our wetter forest site (E. Spear, pers. obs.).

Discussion

PATHOGENS WERE THE PRIMARY CAUSE OF SEEDLING MORTALITY IN BOTH FORESTS

During our 5-month study, pathogens caused the majority of seedling deaths in both forests, which is consistent with previous evidence that pathogen attack is a major cause of mortality for seedlings under natural conditions (Moles & Westoby 2004; Gilbert 2005; Alvarez-Clare & Kitajima 2009; Mangan *et al.* 2010; Alvarez-Loayza & Terborgh 2011). Pathogen-caused mortality was highly variable among species (from 0% to 51%). Killing some species more than others may facilitate coexistence, which would support the hypothesis that pathogens play a central role in maintaining forest diversity. Finally, consistent with Alvarez-Clare & Kitajima (2009), the rate at which seedlings were killed by pathogens remained relatively constant during our experiment (Fig. 3). This suggests that our study captured the actual patterns and differences between forests and tree species distributions.

PATHOGEN PRESSURE IS ELEVATED IN THE WETTER FOREST RELATIVE TO THE DRIER FOREST

We observed a greater overall risk of pathogen-caused mortality and damage for seedlings in the wetter forest (Fig. 3a; Tables 1a and 2), supporting our prediction of a gradient in pathogen pressure that correlates positively with the precipitation gradient. This pattern was only significant for seedlings of wet-forest species (Table S5a,c), whereas seedlings of dry-forest species suffered equally high pathogen-caused mortality and damage in both forests (Figs 1c, 2a,b and 3). Seedlings of dry-forest species may have suffered equally high pathogen-caused mortality and damage in the drier forest because the presence and high abundance of conspecific adults may have exposed them to an accumulation of specialist pathogens. On a local scale, it has been shown that seedlings experience more pathogen-caused damage and mortality near conspecific adults (Gilbert 2002; Petermann *et al.* 2008; Mangan *et al.* 2010), presumably resulting from a build-up of host-specialized pathogens (Janzen-Connell effects; Connell 1971; Janzen 1970). Under the same logic, it is possible that relatively generalized, multihost pathogens attacked the dry-forest species in the wetter forest where conspecific and, in some cases, congeneric adults of dry-forest species were absent.

We hypothesize that several mutually compatible mechanisms could generate elevated pathogen pressure in the wetter forests. First, seedlings could experience more pathogen-caused damage and mortality in wetter forests because limited dispersal of pathogens and/or environmental filtering could result in different pathogen communities in the wetter versus drier forests (Gilbert 2002). For many but not all plant diseases, incidence and severity increase with more rain and higher relative humidity (e.g. *Pythium*-caused seedling damping off versus powdery mildews, respectively; Agrios 2005); thus, future work could compare the incidence and severity of different types of pathogens (soil-borne versus airborne and biotrophic versus necrotrophic) in the wetter versus drier forests. Secondly, independent of compositional differences, the pathogen communities could differ in their aggressiveness. Reciprocal selection, or a co-evolutionary arms race, between the trees and their pathogens in the wetter forest could select for pathogens that are better able to infect and damage host trees (Gilbert 2002). Thirdly, the higher annual rainfall, shorter dry season (Condit 1998; Pyke *et al.* 2001) and higher relative humidity (Santiago *et al.* 2004) characterizing the wetter forests may provide an abiotic environment that favours pathogens by being more conducive to reproduction, dispersal and/or infection (Gilbert 2005; Barrett *et al.* 2009; Hersh, Vilgalys & Clark 2012; Swinfield *et al.* 2012). A fourth possibility is that the poorer soils (Brenes-Arguedas *et al.* 2008; Condit *et al.* 2013) and lower understorey light levels (Brenes-Arguedas *et al.* 2011) of the wetter forests stress seedlings and make them more susceptible to disease (Agrios 2005; Barrett *et al.* 2009). Finally, any or all of these mechanisms could be interacting additively or synergistically to generate the elevated pathogen pressure observed in the wetter forest.

WET- AND DRY-FOREST SPECIES ARE DIFFERENTIALLY IMPACTED BY PATHOGENS

We posited that dry-forest species may be poorly defended relative to wet-forest species because they are adapted to an environment characterized by relatively low pathogen pressure and experience weaker selection for defences against pathogens (Coley & Barone 1996) and that, because they are poorly defended, they are more susceptible to pathogen attack. In general, seedlings of dry-forest species did tend to suffer more pathogen-caused mortality (Fig. 3). Not surprisingly given the inherent differences among tree species in the traits influencing disease vulnerability, not all dry-forest species suffered more pathogen-caused mortality than wet-forest species (Fig. 2). It is highly likely that other plant traits, in addition to distribution, are important.

While seedlings of dry-forest species were not more likely to be damaged by pathogens, pathogen-caused damage was significantly more likely to result in death for dry-forest species than for wet-forest species (Fig. 1d,e). Dry-forest species were more likely to die when attacked regardless of forest (Fig. 1d,e; Table S5b). This suggests that dry- and wet-forest species do not differ in their resistance to pathogen attack but do differ in their tolerance of pathogen attack. A greater fitness impact experienced by seedlings of dry- versus wet-forest species may reflect an intrinsically inferior ability to halt or slow infection because of lower investment in constitutive defences or a lesser capacity to detect and suppress pathogens via induced defences. In fact, Santiago *et al.* (2004) demonstrated that dry-forest species tend to have shorter lived and less defended leaves than wet-forest species. A greater impact to host fitness may also reflect an inferior ability to compensate for lost tissue (Strauss & Agrawal 1999).

DO PATHOGENS EXCLUDE DRY-FOREST SPECIES FROM WETTER FORESTS?

Our results suggest that there is a greater risk of pathogen-caused damage and mortality in the wetter forests and that seedlings of dry-forest species tend to suffer more pathogen-caused mortality than wet-forest species. Together, these results suggest that pathogens could act as a biotic filter limiting the recruitment of some dry-forest species in the wetter forests. Furthermore, a greater fitness impact from pathogen attack for surviving seedlings of dry-forest species could translate into higher mortality later in life (Mangan *et al.* 2010) or lower lifetime fecundity, reducing their persistence. Pathogens may be a weaker filter than the drought filter acting in the drier forests and, indeed, more tree species are restricted to the wetter forests than to the drier forests (Condit 1998).

Seedlings of dry-forest species tended to suffer more pathogen-caused mortality than wet-forest species in both forests (Fig. 3). The fact that seedlings of dry-forest species were at a greater risk of pathogen-caused mortality in the drier forest than seedlings of wet-forest species is consistent with our prediction that seedlings of dry-forest species are more vulnerable to pathogen attack than wet-forest species in both forests and is in opposition with the alternative hypothesis that dry-forest tree species are adapted to and, thus, more resistant to the pathogens that they commonly encounter in the drier forests (Table S5). For pathogens to act as a filter and limit the establishment of dry-forest species in the wetter forests, dry-forest species only need to suffer more pathogen-caused mortality than wet-forest species in the wetter forests (i.e. no forest by distribution interaction is necessary). In the drier forests, dry-forest species dominate, even though they are more sensitive to pathogens than wet-forest species, because wet-forest species are drought-intolerant. Thus, regional turnover of tree species occurs because seedlings of wet-forest species suffer high mortality in the drier forests due to seasonal drought (Engelbrecht *et al.* 2007; Brenes-Arguedas, Coley & Kursar 2009) and, in part, because seedlings of dry-forest species tend to suffer relatively more pathogen-caused mortality in the wetter forests.

In such a complex and diverse system, it is unrealistic to assume that pathogens are the only determining factor. The ensemble of abiotic and biotic factors that may be sorting tree species across the rainfall gradient include nutrient availability (Brenes-Arguedas *et al.* 2008; Condit *et al.* 2013), light (Brenes-Arguedas *et al.* 2011), herbivores (Brenes-Arguedas, Coley & Kursar 2009) and differences among species in their inherent growth rates (Brenes-Arguedas *et al.* 2008, 2011; Brenes-Arguedas, Coley & Kursar 2009). It is difficult to disentangle their relative contributions to community assembly because all of these factors are likely to interact and their relative contributions are likely to change in different habitats. It has been hypothesized that there is a trade-off between drought tolerance and competitive ability and that, while drought-tolerant plants are physiologically capable of growing in wetter areas, their lower growth rates lead to poor competitive ability (Brenes-Arguedas *et al.* 2008, 2011; Brenes-Arguedas, Coley & Kursar 2009). Lower competitive ability and a greater fitness impact from disease may act in combination to ultimately exclude dry-forest species from the wetter forests.

DRY-FOREST SPECIES EXPERIENCED LITTLE TO NO ESCAPE FROM PATHOGENS IN THE FOREST IN WHICH THEY DO NOT NATURALLY OCCUR

In our study, seedlings of four of the six dry-forest species that we tested suffered high pathogen-caused mortality in the wetter forest, in which none of the dry-forest tree species have been observed (Condit *et al.* 2013; Fig. 2a; Table S1). This lack of escape from disease suggests that some pathogens are relatively widespread and/or are capable of damaging multiple host species. Evidence that plant-associated fungi have geographically limited dispersal (Gonthier *et al.* 2001; Gilbert 2002; Peay *et al.* 2012) suggests that pathogens may not be widespread. Assuming limited dispersal and given the relative rarity of tree species in diverse tropical forests, selection should favour pathogens with broad host ranges (May 1991). Multihost pathogens can promote coexistence and enhance diversity if infection by a shared pathogen differentially affects each host (Hersh, Vilgalys & Clark 2012; Sedio & Ostling 2013) and if the abiotic environmental factors that modulate plant–pathogen interactions vary in space (Benítez *et al.* 2013). The possibility that some pathogens are relatively widespread and/or are capable of damaging multiple host species underscores the fact that the distributions and host specificities of pathogens remain critical lacunae in our understanding of how plant–pathogen interactions shape plant community composition and diversity.

Conclusions

In summary, we show that tree seedlings are more likely to be damaged and killed by pathogens in wetter forests than in drier, more seasonal forests and that seedlings of dry-forest tree species tend to suffer a greater negative impact from pathogens, potentially limiting the recruitment of some dry-forest tree species in wetter forests. There is increasing evidence of the biotic regulation of species distributions, and our results suggest that seedling pathogens may be an important, albeit little explored, biotic factor restricting the distributions of trees across a rainfall gradient and, thereby, enhancing regional forest diversity. An understanding of the mechanisms shaping beta-diversity (species turnover) across landscape-scale gradients is essential for disentangling the factors responsible for the impressive diversity of tropical forests.

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Data accessibility

Data supporting the results presented in this paper are available from the Dryad Digital Repository (Spear, Coley & Kursar 2014).

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Supporting Information

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

Table S1. Descriptions of the 12 tree species tested, the occurrence of conspecific or congeneric adults in our wetter or drier forest sites, and seed collection details.

Table S2. Species-specific planting and germination data for the 12 tree species tested.

Table S3. Densities of seedling-sized plants in our common gardens and naturally occurring in our wetter and drier forest sites.

Table S4. Species-by-species variability in pathogen-caused mortality and damage.

Table S5. Pairwise comparisons for likelihood of pathogen-caused damage, likelihood of death given pathogen-caused damage and overall risk of pathogen-caused death.

Figure S1. A map of the study sites in the Republic of Panama and a photo of one of the common gardens.

Figure S2. A cladogram depicting the evolutionary relationships among the 12 tree species tested, their cross-isthmus distributions (drier versus wetter forests), and their shade-tolerance guilds for the seedling stage.

Figure S3. Frequency distribution of ages for the seedlings killed by pathogens.

Figure S4. Time-lapse images of a seedling that suffered pathogen-caused damage and death.