PATHOGENS OF SEEDLINGS ACT AS A BIOTIC FILTER, LIMITING THE DISTRIBUTIONS OF TROPICAL TREES AND CONTRIBUTING TO BETA-DIVERSITY

by

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ABSTRACT

In Panama, a high level of spatial turnover in tree species is correlated with a rainfall gradient. Seasonal drought is known to exclude tree species typical of wetter forests from drier forests; however, the factors contributing to the converse are ambiguous. This dissertation research experimentally tested the hypothesis that pathogens attacking seedlings contribute to the exclusion of dry-forest tree species from wetter forests. We tested two related hypotheses: that the phytopathogens attacking seedlings are (i) geographically widespread and (ii) host generalists.

To test if pathogens exclude dry-forest species from wetter forests, wet- and dry-forest tree species were planted in wetter and drier forests in Panama and monitored for pathogen-caused damage and mortality. Seedlings suffered more pathogen-caused damage and mortality in the wetter forest, while dry-forest tree species suffered a greater impact from pathogen attack than wet-forest species. Together, these results support our hypothesis.

Next, fungi isolated from symptomatic seedlings were identified using molecular techniques and phylogenetic analyses. We observed 28 fungal species and found that, while diversity was greater in the wetter forest, one-third of the observed fungal species were found in both the wetter and drier forests. This suggests that some phytopathogens are geographically widespread and that the elevated impact from pathogens in wetter forests may not be the result of different pathogen communities.

Finally, we surveyed the tree species in which potential phytopathogens occurred and used inoculation experiments to assess the pathogenicity and host ranges of the potential phytopathogens. Most of the potential phytopathogens were isolated from multiple, phylogenetically distant families of trees. Similarly, in the experiments, phytopathogens were able to attack phylogenetically distant tree species. Tree species were differentially vulnerable to attack, suggesting that these generalist phytopathogens can influence plant community composition.

While specialist phytopathogens have received considerable attention for their role in the maintenance of local diversity, this work highlights the underappreciated effect of generalist pathogens on regional diversity, represents one of the few studies to experimentally assess the host ranges of seedling pathogens in the tropics, and provides the first estimate of the taxonomy, diversity, and spatial structure of tropical phytopathogens at the landscape scale.

This dissertation is dedicated to my parents and brother who have always believed in me.

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CHAPTER 1

INTRODUCTION

Background

The incredible diversity of tropical rainforests has fascinated scientists since the first natural history studies in the tropics by Alexander von Humboldt in the 1800s. Both abiotic conditions and biotic interactions are thought to contribute to the assembly of forest communities and facilitate the coexistence of seemingly similar plant species (Wright 2002, Leigh et al. 2004). Despite considerable interest, the specific mechanisms and their relative contributions to local species composition (α-diversity) and the spatial turnover of species (β-diversity) remain poorly understood for tropical plant communities. The goal of this thesis is to examine how phytopathogens of seedlings limit the geographic distributions of tree species and, thus, influence the spatial turnover of tree species in the tropical forests of Panama.

Density- or distant-dependent mortality caused by species-specific pests is a commonly cited mechanism for the maintenance of local plant diversity in the tropics (Janzen-Connell-hypothesis; Janzen 1970, Connell 1971; see also Gillett 1962). There is compelling evidence that phytopathogens are particularly important contributors to the observed demographic patterns (Mangan et al. 2010, Bagchi et al. 2014). Yet, the risk of phytopathogen attack, hereafter referred to as pathogen pressure, is influenced by

environmental variables. Thus, the role of phytopathogens in regulating plant community diversity may vary across habitats.

Environmental conditions impact the fitness of both phytopathogens and plants (Barrett et al. 2009). As such, pathogen pressure is likely to differ in relation to environmental heterogeneity. For example, elevated pathogen attack has been observed for seeds and seedlings under low light and high soil moisture conditions (Augspurger and Kelly 1984, Hersh et al. 2009, Mordecai 2012) and for clover species in areas with persistent fog and dew (Bradley et al. 2003). By extension, plant species that are adapted to environments with high pathogen pressure may be under selection for increased defenses against phytopathogens and plant species that are adapted to environments characterized by low pathogen pressure may be poorly defended and more vulnerable to disease (Coley and Barone 1996, Talley et al. 2002). Thus, phytopathogens may exclude disease-sensitive plant species from areas characterized by abiotic conditions that enable elevated pathogen pressure. By limiting the spatial distributions and abundances of certain plant species, phytopathogens may contribute to the maintenance of regional plant diversity.

In Panama, a dramatic spatial turnover in tree species correlates with a rainfall gradient (Pyke et al. 2001). Forest plots 50 km apart share only 1-15% of their tree species (Condit et al. 2002). The spatial turnover of tree species contributes to high regional diversity. Approximately 800 tree species inhabit ca. 2400 km2, well exceeding the diversity of tree species present in the U.S. and Canada (Pyke et al. 2001, Wright 2002).

Experimental assessments of drought tolerance coupled with data on tree species distributions suggest that seasonal drought excludes drought-sensitive species from the drier, Pacific forests (Engelbrecht et al. 2007, Brenes-Arguedas et al. 2009). Conversely, it is less clear why numerous tree species are excluded from or occur in very low abundances in the wetter forests.

We hypothesized that (i) the environmental conditions characterizing the wetter forests favor phytopathogen activity, that (ii) those tree species adapted to living in the wetter forests are under selective pressure to be better defended against attack while tree species typical of the drier forests are poorly defended, and that (iii) dry-forest tree species are excluded from the wetter forests by pathogen-caused seedling mortality. We focused on the seedling stage because seedling mortality directly shapes forest communities (Engelbrecht et al. 2007, Comita et al. 2010, Mangan et al. 2010, Baldeck et al. 2014, Green et al. 2014).

Our central hypothesis is contingent on two nonmutually exclusive presumptions: first, that the phytopathogens attacking seedlings are widespread geographically and, second, that they have broad host ranges. Phytopathogens are difficult to observe because of their size and parasitic nature and, despite their prevalence and importance for natural plant communities, little is known about their natural histories. Thus, we used survey-based and experimental approaches to begin to address the explicit presumptions made by our central hypothesis.

Chapter summaries

In Chapter 2 (Spear et al. 2015), we establish that a gradient in pathogen pressure correlates positively with the rainfall gradient and that the negative impact from pathogen attack is greater for tree species typical of the drier versus wetter forests. This was accomplished via a reciprocal transplant experiment in the wetter and drier forests of Panama. We monitored seedling survival and the incidence of pathogen attack for six dry- and six wet-forest tree species. Consistent with our central hypothesis, these results suggest that elevated pathogen pressure in the wetter forests contributes to the exclusion of dry-forest tree species. Host-specific pathogens should accumulate in the vicinity of their hosts. Therefore, the lack of escape from disease for seedlings of dry-forest species planted in the wetter forest, in which they do not naturally occur, suggests that the dry-forest species were attacked by phytopathogens with broad host ranges. This offers tentative support for our presumption that at least some of the phytopathogens are capable of damaging multiple host species.

In Chapter 3, we identify 28 species of potential phytopathogens inhabiting the forests spanning the rainfall gradient in Panama and we show that the phytopathogen communities are richer and more diverse in the wetter versus drier forests. However, we also show that the drier and wetter forests (ca. 45 km apart) share 33% of the observed species of phytopathogens. The fungal phytopathogens were isolated in culture from the tissue of 75 symptomatic seedlings collected from seven forests. We estimated the taxonomic placement of each fungal isolate and assigned the isolates to operational taxonomic units based on their internal transcribed spacer (ITS) region sequences. These results support our presumption that the phytopathogens attacking seedlings have

relatively wide geographic ranges and suggest that the compositional differences between the phytopathogen communities in the wetter versus drier forests may not be great enough to explain the elevated disease risk observed for seedlings in the wetter versus drier forests.

In Chapter 4, we verify the pathogenicity of six fungal species attacking seedlings in the forests of Panama and we show that the phytopathogens are able to attack tree species belonging to many families. Furthermore, we document interspecific differences in vulnerability among tree species. To achieve this, we conducted shadehouse-based inoculation experiments, during which we tested 34 fungal isolates and 36 tree species and we documented disease symptoms and mortality. We further augmented our understanding of phytopathogen host ranges by surveying the fungi residing within symptomatic seedlings collected in Panama. While the host generalism documented by our study challenges a commonly held assumption that phytopathogens in the tropics are host specific, the differences among tree species in their vulnerability to phytopathogens suggest that generalist phytopathogens can maintain forest diversity via host-specific impacts as opposed to the mechanism traditionally envisioned by the Janzen-Connell hypothesis. Furthermore, these results support our presumption that the phytopathogens attacking seedlings have broad host ranges and suggest that these generalist phytopathogens contribute to the exclusion of dry-forest tree species from the wetter forests.

Conclusions and contributions to plant ecology

The results presented here suggest that relatively widespread, generalist phytopathogens contribute to the exclusion of dry-forest tree species from the wetter forests of Panama. Research investigating pathogen-mediated impacts to plant diversity has primarily focused on (i) the maintenance of local plant diversity through distance- and density-dependent pathogen attack (Janzen–Connell hypothesis; reviewed in Comita et al. 2014) and on (ii) the threat to local plant diversity posed by exotic plants escaping the specialist pathogens that normally regulate their populations (enemy release hypothesis; reviewed in Mitchell et al. 2006). Our research represents a unique extension of this knowledge base by demonstrating that phytopathogens can limit the geographic distributions of plant species and, thus, contribute to beta diversity. This is particularly relevant in the face of habitat destruction and climate change because the conservation of biodiversity requires an understanding of the factors currently influencing where species can and cannot persist.

Furthermore, in spite of their importance and ubiquity, knowledge of the identities, distributions, host ranges, and host-specific impacts of phytopathogens in natural systems is limited. To our knowledge, Chapter 3 represents the first estimation of the taxonomy, diversity, and spatial structure of tropical phytopathogens across a rainfall gradient and at a landscape scale. Additionally, Chapter 4 is one of three studies to have both identified and examined the host specificities of the phytopathogens killing seedlings in the tropics (Augspurger and Wilkinson 2007, Schweizer et al. 2013).

Future directions and recommendations

In Chapter 3, we show that there is species overlap between the phytopathogen communities in the wetter and drier forests, which suggests that higher pathogen pressure in wetter versus drier forests may not be a product of different pathogen communities. An alternative hypothesis is that the seedlings in the wetter forests may be at a greater risk of pathogen attack because of the interaction between the abiotic environment and disease development. To test this hypothesis, abiotic conditions, such as light and water availability, could be artificially manipulated in the forests to attempt to "rescue" seedlings from pathogen attack in the wetter forests by mimicking dry-forest conditions and increase pathogen attack of seedlings in the drier forests by mimicking wet-forest conditions.

Together, the results presented in Chapters 2 and 4 suggest that generalist phytopathogens attacking seedlings enhance regional forest diversity by limiting the geographic distributions of certain tree species. To determine if the generalist phytopathogens contribute to the maintenance of local diversity, it is necessary to establish if a competition-defense tradeoff exists among coexisting host plants. For multihost phytopathogens to enhance plant community diversity, superior competitors need to suffer a greater impact from shared phytopathogens than inferior competitors (Mordecai 2011). Thus, future research should include competition experiments between coexisting species. The observed relative abundances and spatial distributions of plant species should then be related to their competitive abilities and disease sensitivities.

Furthermore, in a temperate system, different combinations of co-infection resulted in unequal effects on seedling survival among plant species (Hersh et al. 2012).

Our inoculation experiments were not designed to evaluate the effects of co-infection, which may be a key variable in understanding how generalist phytopathogens influence plant community diversity (Benítez et al. 2013). Thus, we recommend that future work investigating phytopathogens in the tropics combine survey-based and experimental studies to evaluate host-specific impacts of co-infection by different combinations of phytopathogens.

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CHAPTER 2

DO PATHOGENS LIMIT THE DISTRIBUTIONS OF TROPICAL TREES ACROSS A RAINFALL GRADIENT?

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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: $F = 0.114$, $P = 0.743$).

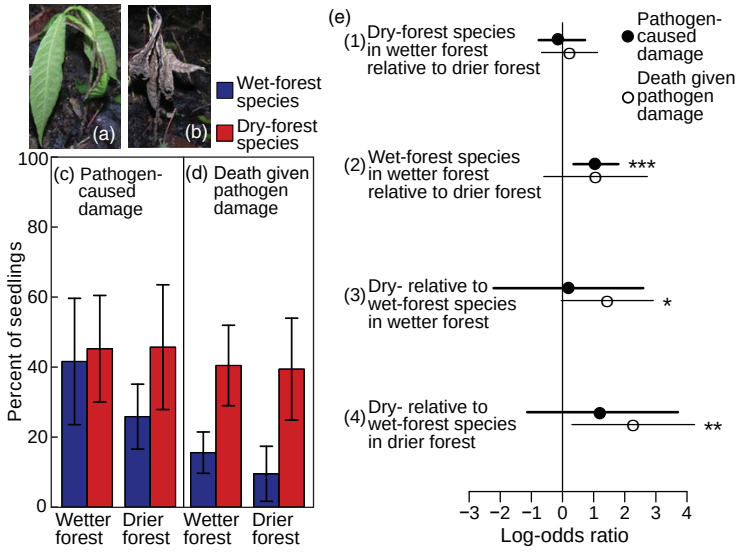

Fig. 1. Photos of a seedling of *Anacardium excelsum* that (a) suffered pathogen-caused damage and (b) was dead within seven days. Percent of seedlings with (c) pathogen-caused damage and (d) for which damage was lethal. Percentages were calculated by averaging forest by species percentages (species 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 (*$P \leq 0.05$, **$P \leq 0.01$, ***$P \leq 0.001$).

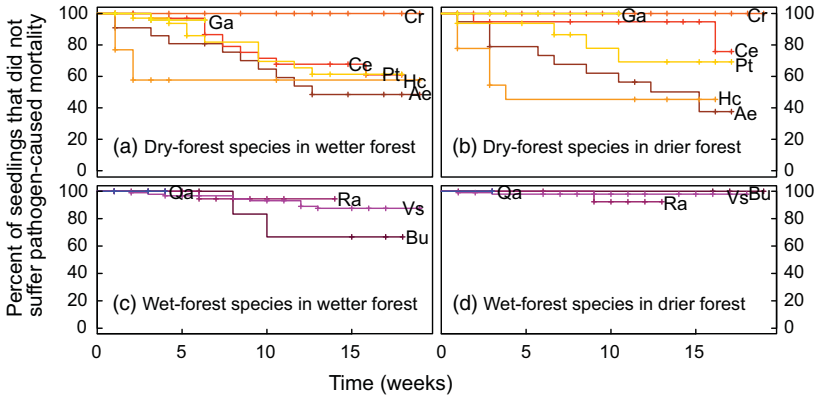

Fig. 2. Pathogen-caused mortality (Kaplan–Meier survivorship curves) for the six dry- (a, b) and four of the six wet-forest tree species (c, d) in the wetter (a, c) and drier (b, d) forests. Species codes are at the end of each curve (see Table S1 for full names). Survivorship curves were not plotted 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).

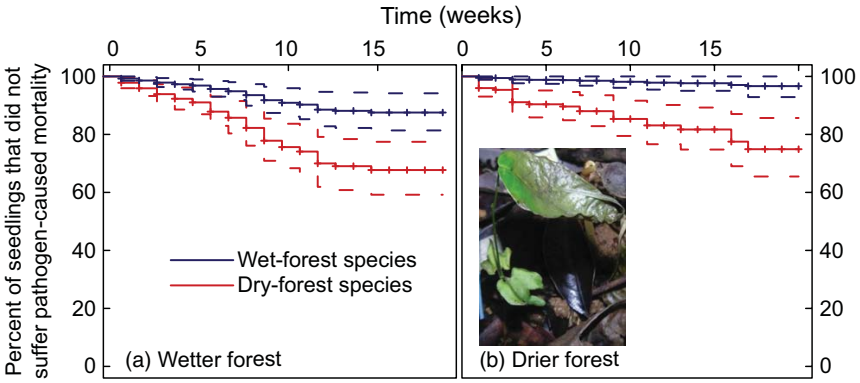

Fig. 3. Pathogen-caused mortality was greater (a) in the wetter forest than (b) in the drier forest and was greater for seedlings of dry-forest species (red lines) than for wet-forest species (blue lines). The survival curves (solid) and 95% confidence intervals (dashed) are the estimates from a Cox 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.

Table S1. The 12 dry- and wet-forest tree species tested. Species name, two-letter code, family (refer to the Tropicos database at tropicos.org for the authorities), mean (±SE) seed wet weights ($n \geq 25$), cross-isthmus distribution (Condit, Pérez & Daguerre 2011), shade-tolerance guild for the seedling stage (LD = light demanding, IST = intermediate shade tolerance, ST = shade tolerant, or some intermediary) based on previously published classifications and indices, presence of conspecific (CS) or congeneric (CG) adults in our wetter (WF) or drier forest (DF) sites (Condit *et al.* 2013; Smithsonian Tropical Research Institute) and seed collection details (site, number of source trees, collection from ground vs. tree). PNS = Parque Nacional Soberanía (moist forest), PNM = Parque Natural Metropolitano (drier forest; transitional between moist and dry forests), BCI = Barro Colorado Island (moist forest), PNSL = Parque Nacional San Lorenzo (wetter forest; transitional between moist and wet forests), and BCNM = Barro Colorado Nature Monument - Gigante Peninsula (moist forest).

Species	Code	Family	Mean (±SE) seed wet wt. (g)	Dist.	Shade-tolerance guild	Conspecific (CS) or congeneric (CG) adults observed in wetter (WF) or drier forest (DF) sites	Seed coll. site	Trees	Coll. from
Anacardium excelsum	Ae	Anacardiaceae	2.19 ± 0.11	dry	LD[a] - IST[b]	CS in DF[l]	PNS	> 5	ground
Castilla elastica	Ce	Moraceae	0.34 ± 0.01	dry	LD[c] - IST[d]	CS in DF[l]	PNM	> 3	ground
Cojoba rufescens	Cr	Fabaceae	0.27 ± 0.01	dry	ST[e]	CS in DF[l]	BCI	2	tree
Genipa americana	Ga	Rubiaceae	0.19 ± 0.004	dry	ST[a]	CS in DF, CG in WF	BCI	1	ground
Hymenaea courbaril	Hc	Fabaceae	4.53 ± 0.08	dry	ST[f]	CS in DF	BCI	1	tree
Protium tenuifolium	Pt	Burseraceae	0.22 ± 0.01	dry	ST[g,h]	CG in WF	BCI	2	tree
Brosimum utile	Bu	Moraceae	6.03 ± 0.26	wet	IST[i]	CS in WF	PNSL	1	tree
Carapa guianensis	Cg	Meliaceae	42.89 ± 2.23	wet	ST[j]	CS in WF	PNSL	1	tree
Pourouma bicolor	Pb	Urticaceae	0.48 ± 0.03	wet	LD[g] – IST[i]	CS in WF	BCNM	2	ground
Quararibea asterolepis	Qa	Malvaceae	1.1 ± 0.05	wet	ST[g,h]	none	BCI	> 5	ground
Randia armata	Ra	Rubiaceae	0.82 ± 0.02	wet	ST[a,g]	none	BCI	1	ground

Virola surinamensis	Vs	Myristicaceae	2.01 ± 0.03	wet	ST [a,k]	CG in WF	BCI	> 3	tree & ground

[a]Kitajima *et al.* 2012. While Kitajima *et al.* (2012) classified *A. excelsum* as a gap-dependent species, 61% of the *A. excelsum* seedlings that they planted in the shaded understory survived for 90 days after planting (vs. 94% in a gaps); based on those survivorship percentages and comparisons with the survivorship of more light-demanding species (e.g., *Jacaranda copaia*), we argue that *A. exclesum* falls between light demanding and intermediate shade tolerant.
[b]Fournier 2002a. According to Fournier (2002a), *Anacardium excelsum* seedlings are shade tolerant during their early stages of development and require more light for survival and growth during later stages of development.
[c]McCarthy & Kobe 2008.
[d]Myers & Kitajima 2007. Based on the proportion of seedlings of *C. elastica* that survived for 1 year in the shaded understory (57.6 ± 5.6%) and comparisons with the survivorship of more light-demanding species in the Myers and Kitajima (2007) study, we argue that *C. elastica* falls between light demanding and intermediate shade tolerance.
[e]Kursar *et al*. 2009.
[f]Markesteijn *et al.* 2011.
[g]Comita *et al.* 2007.
[h]Coley 1983.
[i]Santiago & Wright 2007.
[j]Fournier 2002b. According to Fournier (2002b), *C. guianensis* seedlings are shade tolerant during the early stages of development.
[k]Howe 1990.
[l]Adults abundant near seedling plots.

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Table S2. Species-specific planting and germination data for the 12 tree species used in the reciprocal transplant experiment. Two-letter code for tree species name (full names in Table S1), cross-isthmus distribution (drier vs. wetter forests), approximate planting times, week(s) when planted (Jan 1 = Julian week 1) and mean week planted for each species, the median (min-max) week of germination for each species, the median (min-max) number of seeds of each species planted in each common garden (1,960 seeds in total), the median (min-max) number of seedlings of each species in each common garden and the total number of seedlings/total number of seeds planted and percentage of seeds germinated of each species in the drier and wetter forest sites. Seeds were equally likely to germinate in the two forests (GLMM: Odds ratio wetter:drier (95% confidence interval) = 1.05 (0.68, 1.63), $P = 0.82$). The wet-forest tree species used in this study tended to have lower germination success than the dry-forest tree species, but not significantly so (GLMM: OR dry:wet (95% CI) = 2.64 (0.86, 8.12), $P = 0.09$). Germination success was not the focus of this study and causes of germination failure were not tracked; therefore, the results of the analyses of germination success cannot be accurately interpreted. We planted the seeds of a given tree species in the 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 the drier forest site in one day and in the wetter forest site in the following day). Furthermore, whenever possible, we planted wet- and dry-forest tree species concurrently (on the same dates). 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, median week of germination did not differ for wet- versus dry-forest species (Wilcoxon rank sum test: $W = 8.5$, $P = 0.148$). Finally, 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.

Tree sp.	Dist.	Approx. planting times	Week(s) Planted	Mean week planted	Median (min-max) week of germination	Median (min-max) seeds per garden	Median (min-max) seedlings per garden	Total seedlings/seeds (% germ) in drier forest	Total seedlings/seeds (% germ) in wetter forest
Ae	dry	mid - late Jun	25, 26	25.5	27 (26-32)	2 (1-3)	1 (0-2)	19/66 (29%)	22/66 (33%)
Ce	dry	mid - late Jun	25, 26	25.5	29 (27-37)	3 (2-4)	1 (0-4)	22/80 (28%)	36/81 (44%)
Hc	dry	late Jun	26	26	32 (26-46)	1 (1-2)	0 (0-2)	20/41 (49%)	13/42 (31%)
Cr	dry	late Jun, late Jul, mid Aug	26, 30, 34	30	35 (27-44)	3 (2-5)	2 (0-5)	62/94 (66%)	49/94 (52%)
Pt	dry	late Jun - mid Jul	27, 28, 29	28	30 (28-39)	3 (2-3)	1 (0-3)	16/88 (18%)	42/90 (47%)
Ga	dry	late Aug	36	36	43 (36-44)	2 (2-3)	1 (0-3)	30/61 (49%)	37/60 (62%)

Pb	wet	mid Jun	25	25	38.5 (34-43)	2 in all	0 (0-1)	4/60 (7%)	2/60 (3%)
Vs	wet	mid - late Jun	25, 26, 27	26	33 (27-40)	8 (7-10)	3 (0-8)	96/230 (42%)	96/230 (42%)
Bu	wet	late Jun	26	26	30 (28-35)	3 (3-4)	0 (0-2)	5/103 (5%)	10/105 (10%)
Ra	wet	late Jul	30	30	38 (33-44)	2 (1-2)	1 (0-2)	21/50 (42%)	29/51 (57%)
Cg	wet	mid Aug	33	33	38 (33-44)	1 (0-1)	0 (0-1)	5/18 (28%)	1/19 (5%)
Qa	wet	mid Sept	38	38	44 (43-46)	3 (1-3)	1 (0-3)	56/87 (64%)	32/85 (38%)

Table S3. Densities of seedling-sized plants in the common gardens and naturally occurring in the (a) drier and (b) wetter forests. For all common gardens, the total density of planted seedlings and the density of conspecific, planted seedlings were at or below natural densities of seedling-sized plants.

	All seedling-sized plants		Most abundant species	
	Max (m^{-2})	Average (m^{-2})	Max (m^{-2})	Average (m^{-2})
(a) Drier forest				
Common garden	21	12	6	3
Natural	24	12	12	5
(b) Wetter forest				
Common garden	22	12	8	4
Natural	68	32	58	10

Table S4. Species-by-species variability in pathogen-caused mortality and damage for the 12 tree species tested (see Table S1 for full names). For each species, the columns detail their cross-isthmus distribution, their species code, the total number of seedlings in both forests, the total number and percentage of seedlings that were killed by pathogens in both forests, the total number and percentage of seedlings that were damaged by pathogens in both forests and, considering only those seedlings that suffered pathogen-caused damage, the percentage for which that pathogen-caused damage resulted in death.

Tree species distribution	Tree species	Total no. of seedlings	Total no. (%) of seedlings killed by pathogens*	Total no. (%) of seedlings damaged by pathogens*	Of those with pathogen-caused damage (*n* = 272), proportion for which it was lethal
Drier forests	Ae	41	21 (51%)	39 (95%)	54%
	Ce	58	12 (21%)	32 (55%)	38%
	Cr	111	0 (0)	12 (11%)	0
	Ga	67	1 (1%)	4 (6%)	25%
	Hc	33	13 (39%)	20 (61%)	65%
	Pt	58	14 (24%)	26 (45%)	54%
Wetter forests	Bu	15	2 (13%)	9 (60%)	22%
	Cg	6	0 (0)	2 (33%)	0
	Pb	6	0 (0)	1 (17%)	0
	Qa	88	0 (0)	4 (5%)	0
	Ra	50	2 (4%)	8 (16%)	25%
	Vs	192	12 (6%)	115 (60%)	10%
	All species	725	77 (11%)	272 (38%)	28%

Table S5. Informative pairwise comparisons for (a) likelihood of pathogen-caused damage, (b) likelihood of pathogen-caused death given pathogen-caused damage and (c) overall risk of pathogen-caused death. The log-odds ratios compare the relative odds of an outcome (e.g., pathogen-caused damage) given a change in an independent variable (e.g., wetter forest relative to the drier forest). Positive log-odds values indicate a positive relationship between the likelihood of the outcome and the change in the independent variable and *vice versa*. A hazard ratio greater than one indicates an increased hazard of pathogen-caused death.

	(a) Pathogen-caused damage Log-odds ratio (95% CI)	(b) Death given pathogen-caused damage Log-odds ratio (95% CI)	(c) Pathogen-caused death Hazard ratio (95% CI)
(1) DS.WF-DS.DF	-0.02 (-0.76, 0.72) NS	0.23 (-0.67, 1.12) NS	1.32 (0.69, 2.54) NS
(2) WS.WF-WS.DF	1.07 (0.35, 1.8)***	1.06 (-0.6, 2.72) NS	4.39 (0.91, 21.16)*
(3) DS.WF-WS.WF	0.19 (-2.21, 2.59) NS	1.44 (-0.04, 2.91)*	2.24 (0.25, 20.24) NS
(4) DS.DF –WS.DF	1.28 (-1.14, 3.7) NS	2.27 (0.3, 4.24)**	7.45 (0.6, 93.04)*

DS = dry-forest tree species, WS = wet-forest tree species, WF = wetter forest, DF = drier forest

*****$P \leq 0.05$, ******$P \leq 0.01$; *******$P \leq 0.001$, NS not significant

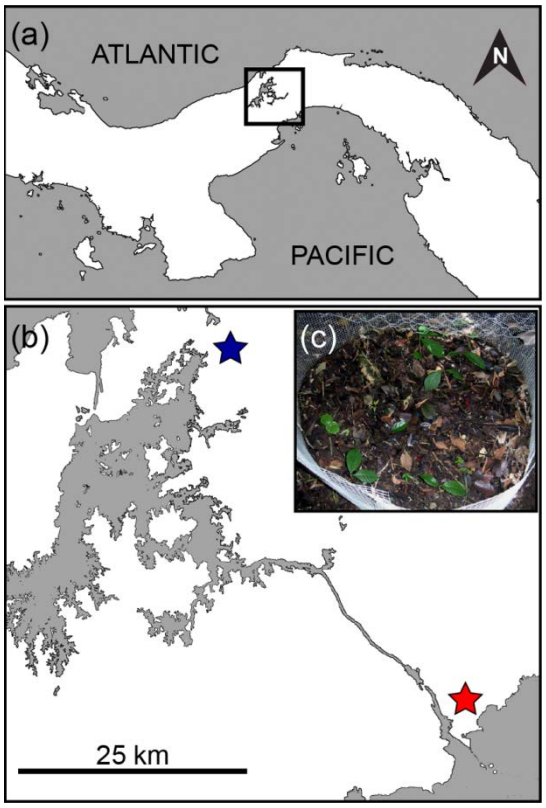


Fig. S1. The study sites (a) in the Republic of Panama, (b) located in the wetter (blue) and drier (red) forests and (c) a common garden in our drier forest site.

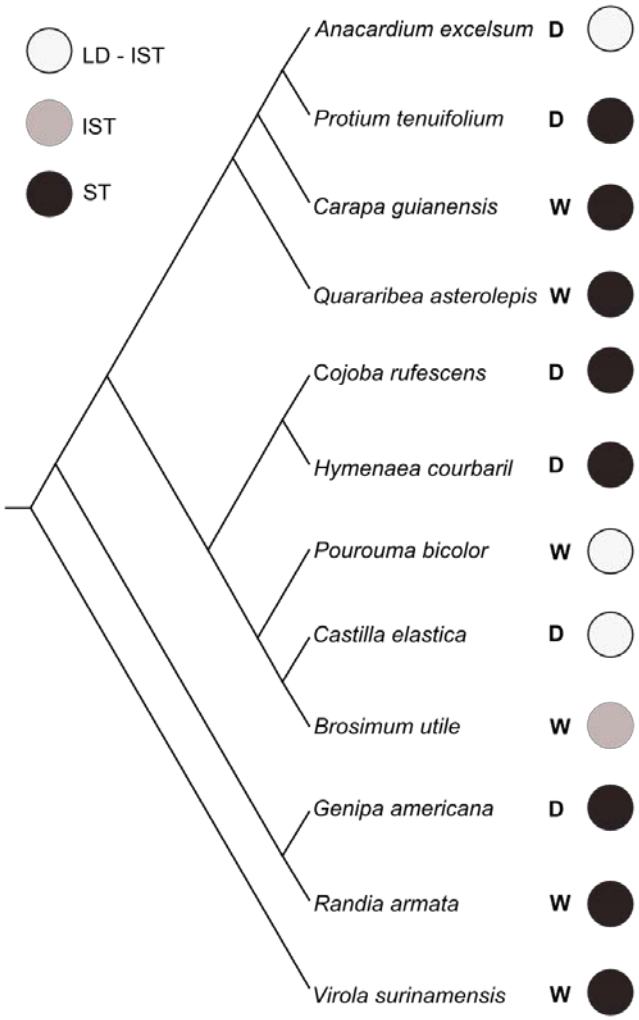


Fig. S2. Cladogram depicting the evolutionary relationships among the 12 tree species used in our reciprocal transplant experiment. This was constructed based on an Angiosperm Phylogeny Group III-derived megatree (R20120829) in the online software Phylomatic v3 (Webb & Donoghue 2005) and visualized using TreeView X v0.5.0 (Page 1996); branch lengths are arbitrary. The dry-forest (D) and wet-forest (W) tree species that were used are distributed over the phylogeny with no clear phylogenetic separation. The circles indicate the shade-tolerance guild for the seedling stage for each species (LD – IST (open) = light demanding to intermediate shade tolerance, IST (grey) = intermediate shade tolerance, ST (black) = shade tolerant; see Table S1 for classification details).

References cited in Fig. S2

Page, R.D.M. (1996) Treeview: an application to display phylogenetic trees on personal computers. *Computer Applications in the Biosciences*, **12**, 357–358.

Webb, C.O. & Donoghue, M.J. (2005) Phylomatic: tree assembly for applied phylogenetics. *Molecular Ecology Notes*, **5**, 181-183.

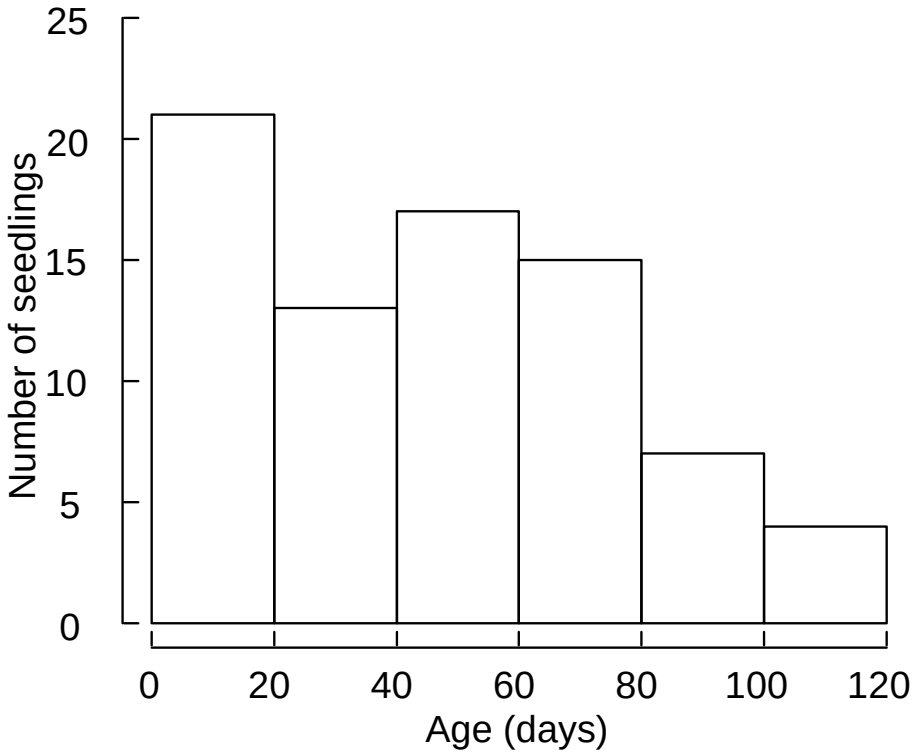


Fig. S3. Frequency distribution of ages for the seedlings killed by pathogens (*n* = 77). The median age was 42 days.

Fig. S4. Time-lapse images of an *Anacardium excelsum* seedling that suffered pathogen-caused mortality in the forest of Barro Colorado Island, Panama. The seedling had noticeable foliar necrosis on June 29, 2011 and was dead on July 4, 2011. The seedling was not visible in the July 3rd images.

CHAPTER 3

PHYLOGENETIC RELATIONSHIPS AND SPATIAL DISTRIBUTIONS OF PHYTOPATHOGENS OF SEEDLINGS ACROSS A RAINFALL GRADIENT IN PANAMA

Abstract

In spite of their hypothesized role in structuring plant communities, few studies have identified the phytopathogens impacting wild plant communities or described the spatial distributions of the phytopathogens. Across a precipitation gradient in Panama (ca. 60 km), seedlings are more likely to suffer pathogen-caused damage and mortality in wetter forests. To explore the mechanism(s) responsible for this spatial variation in disease prevalence, we identified fungi that are likely phytopathogens, explored if phytopathogen richness and diversity are correlated with precipitation, and compared the communities of phytopathogens inhabiting the wetter versus drier forests. Specifically, we isolated 90 fungal isolates from symptomatic seedlings collected from forests spanning the rainfall gradient and, based on their internal transcribed spacer (ITS) region sequences, we estimated the taxonomic placement of each isolate and assigned the isolates to operational taxonomic units. The isolates represent 28 fungal species. Genus-level taxonomic placement could be confidently assigned for 73% of the isolates and the five genera most frequently isolated were *Mycoleptodiscus*, *Glomerella*, *Bionectria*,

Diaporthe, and *Calonectria.* We found that the community of phytopathogen species is richer and more diverse in the wetter versus drier forests. Despite these differences, the wetter and drier forests share 33% of the observed species of phytopathogens. This suggests that the elevated disease risk for seedlings in the wetter forests relative to the drier forests may not be the product of compositional differences in the phytopathogen communities. To our knowledge, this study represents the first estimate of the taxonomy, diversity, and spatial structure of tropical phytopathogens across a rainfall gradient and at a landscape scale.

Introduction

Phytopathogens, pathogens that cause disease in plant hosts, have received considerable attention as a leading cause of yield loss in agricultural systems. Increasingly, phytopathogens are receiving attention for regulating the abundance and distribution of plants in natural systems (Gilbert 2002, Mordecai 2011, Bagchi et al. 2014). Phytopathogens are hypothesized to promote local coexistence by preventing any one tree species from becoming overly common (Janzen-Connell effects; Gillett 1962, Janzen 1970, Connell 1971). Conversely, they can inhibit coexistence and potentially decrease local diversity by debilitating inferior competitors and, thereby, amplifying fitness differences between species (Mordecai 2011). Recently, phytopathogens have also been implicated in the spatial turnover of plant species by restricting plant species' ranges across environmental gradients (Defossez et al. 2011, Spear et al. 2015), suggesting that phytopathogens contribute to regional forest diversity.

Despite their importance and ubiquity, basic knowledge of phytopathogen communities (e.g., identities and distributions) and their interactions with plants (e.g., host ranges and host-specific impacts) in natural systems is limited (but see Davidson 2000, Augspurger and Wilkinson 2007, Gilbert and Webb 2007). Knowledge of the identities, richness, diversity, and distributions of phytopathogens is essential for understanding spatial variation in host-pathogen interactions (e.g., why plants are at a greater risk of pathogen attack in certain habitats) and the associated consequences for plant communities.

Across a precipitation gradient spanning the Isthmus of Panama (ca. 60 km), tree seedlings are more likely to suffer pathogen-caused damage and mortality in wetter, less seasonal forests (Spear et al. 2015). This study begins to explore if and how phytopathogen community composition contributes to the elevated risk of pathogen-caused damage and mortality observed in the wetter forests. Many fungal taxa have large geographic ranges (Tedersoo et al. 2014); therefore, we hypothesized that at least some phytopathogens are widespread, which could lead to overlap of pathogen species between the wetter and drier forests. We also hypothesized that the less seasonal, wetter forests support a greater richness and diversity of phytopathogens than the drier forests, which are characterized by a longer dry season and a greater frequency and duration of dry spells during the wet season (Condit 1998, Engelbrecht et al. 2006).

To (i) identify phytopathogens, (ii) explore if phytopathogen community richness and diversity are correlated with precipitation, and (iii) compare the phytopathogen communities in wetter versus drier forests, we isolated fungi from symptomatic tissue of diseased seedlings collected from forest sites spanning the rainfall gradient. We

sequenced the internal transcribed spacer (ITS) region to infer the taxonomic placement of the isolated fungi. We focused on phytopathogens that attack seedlings because mortality during the seedling phase represents a major bottleneck that directly shapes plant communities (Engelbrecht et al. 2007, Comita et al. 2010, Mangan et al. 2010, Baldeck et al. 2014, Green et al. 2014). While oomycetes and bacteria also represent important phytopathogens, our study focuses on fungi, which are responsible for the majority of plant disease (Kirk et al. 2001) and are known to be major agents of seedling mortality in the tropics (Gilbert 2005).

Methods

Sample collection

We collected 75 seedlings with observable pathogen damage from seven forest sites across a rainfall gradient in Panama (Fig 3.1, Table 3.1). The sites span the Isthmus of Panama which is characterized by a north to south rainfall gradient (Fig 3.1, Table 3.1). Total annual rainfall increases from ≤1800 mm of rain $year^{-1}$ on the drier, Pacific side of the Isthmus to ≥3000 mm of rain $year^{-1}$ on the wetter, Atlantic side (Condit 1998, Santiago et al. 2004). The rainfall is highly seasonal and total annual rainfall is influenced by the duration of the dry season (ca. 129 days on the drier side and ca. 67 days on the wetter side) and the frequency and duration of dry spells during the wet season (Condit 1998, Engelbrecht et al. 2006). This rainfall gradient is correlated with a distinct turnover of plant species to the extent that there is almost no overlap in the 50 most common species in the Pacific and Atlantic forests (Pyke et al. 2001). Based on their mean annual precipitation, we categorized the forest sites as drier, mid, or wetter (Table 3.1). The

majority of seedlings were collected from the two drier forests (37 seedlings, 7 from FC and 29 from PNM) and the wetter forest (28 seedlings from SRR) (Table 3.1). In addition to the 75 seedlings collected from forest sites, two symptomatic seedlings were collected from a shadehouse in Gamboa, Panama. The identities (estimated taxonomic placements) of the three fungal isolates from the two seedlings collected from the shadehouses are reported here but those three isolates are not included in the ecological analyses.

The seedlings were collected during the rainy seasons of four years (2007, 2010 - 2012). Symptomatic seedlings were obtained in two ways: 1) opportunistic collection of naturally occurring, symptomatic seedlings and 2) seedling baits. Baiting phytopathogens with plants is a traditional method in phytopathology that capitalizes on the parasitic nature of pathogens to separate them from the numerous other organisms in the soil (Beales 2012). The fungi associated with the diseased seedlings were isolated in pure culture (see below). To maximize the isolation of phytopathogens, fungi were isolated from symptomatic seedlings rather than dead seedlings or soil. Characteristic symptoms of pathogen attack included dark, sunken necrotic lesions on the roots and stem, foliar necrosis, and collapse of the stem at the soil line (i.e., damping off) (Agrios 2005) and, in some cases, the biotic agent was observed on the seedling (e.g., mycelia).

Based on evidence that the likelihood of sharing a phytopathogen decreases with increasing evolutionary distance between tree species (Gilbert and Webb 2007), we collected symptomatic seedlings of tree species spread across the phylogeny to “capture” a representative sample of phytopathogens in each forest. The collected seedlings represent 21 tree species and 11 families (Table 3.2). When possible, we collected: (i) seedlings of multiple tree species from the same site, (ii) seedlings of given tree species

from multiple forest sites, and (iii) multiple seedlings of a given tree species at a single site. However, the number of seedlings collected per tree species ranged from one to 18 and not all species were collected from all forest sites. Seven of the tree species belong to the family Fabaceae and 32 of the 77 collected seedlings were Fabaceae. The disproportionate sampling of Fabaceae was likely because this family is extremely common, but it may also have occurred if Fabaceae are more vulnerable to pathogen attack than the species in other families.

For the seedling baits, seeds of 19 of the 21 tree species (*Calophyllum longifolium* and *Dipteryx oleifera* were not included because seeds were unavailable; Table 3.2) were planted in common gardens in 30 to 40 haphazardly-selected locations per drier (FC in 2007; PNM in 2010 and 2012) and wetter (SRR in 2007, 2010, 2012) forest site and monitored for symptoms of pathogen attack. In 2007, seeds were germinated in a greenhouse and then the seedlings were transplanted into the forests at FC and SRR. In 2010 and 2012, seeds were planted directly in the forests to allow for surface sterilization before planting (as described in Spear et al. 2015).

Tissue processing and isolation of fungi in pure culture

Collected seedlings were transported to the lab to isolate the fungi associated with symptomatic tissue. Each seedling was first rinsed under running tap water. Tissue (leaf, stem, and/or root) was then excised from the advancing margin of disease, where the causative pathogen is likely to be more abundant or active than secondary, saprophytic colonizers. Excised tissue pieces were surface sterilized via sequential immersions in 70% EtOH (2 min), 10% commercial bleach (Clorox, with predilution concentration of

5.25% NaClO; 2 min), and 95% EtOH (30 s) and plated on a nutrient medium commonly used to culture a wide variety of fungi [malt extract agar (MEA)] with an antibiotic (chloramphenicol) to prevent bacterial contamination (following Gilbert and Webb 2007). To isolate fungi into pure culture, hyphal growth emerging from the plated plant tissue was transferred to a new plate of medium. When two morphologically distinct fungi emerged from a single tissue piece, each was transferred to a separate plate. All plates were maintained in an air-conditioned lab. Living vouchers of these fungal isolates are stored as agar slants and as agar plugs suspended in sterile distilled water with the International Cooperative Biodiversity Group (ICBG) at the Smithsonian Tropical Research Institute (STRI), Panama City, Panama.

While we isolated fungi directly from symptomatic tissue to maximize the likelihood of isolating the disease-causing fungus or fungi, fungi isolated from symptomatic plant tissue are not necessarily the causative pathogens. Saprotrophic fungi often colonize recently killed plant tissue and they may outgrow the actual, disease-causing pathogen in culture. Traditionally, phytopathologists establish causation by inoculating healthy plants with the isolate in question to generate the symptoms originally observed and by re-isolating the phytopathogen (fulfilling Koch's postulates; Agrios 2005). Due to the considerable time, labor, and resources that would be required to experimentally evaluate the pathogenicity of 93 fungal isolates from 21 different host tree species, we did not conduct proof of pathogenicity in this study. While we did estimate the taxonomic placements of each of the fungal isolates (methods described in subsequent sections), fungi cannot be reliably classified as pathogens based on their taxonomic affiliations because members of a given genus often represent a range of lifestyles,

including pathogens, mutualistic endophytes, and saprophytes (Delaye et al. 2013). As such, we cautiously refer to all fungi isolated in this study as “phytopathogens”, hereafter, and all interpretations and discussions of the phytopathogen communities should be treated with similar caution.

Molecular analyses

DNA from 93 isolates was used for molecular identification based on the nuclear ribosomal internal transcribed spacer (ITS) region. The ITS region (ca. 600 base pairs) is the accepted fungal DNA barcode (Schoch et al. 2012). Fragments of fungal mycelia were collected from each isolate and preserved in DNA (SDS) extraction buffer for up to one year. As symptomatic seedlings were collected over multiple years and because of funding constraints, the generation of DNA sequence data was completed over multiple years by multiple labs (the Arnold Lab at the University of Arizona, the ICBG at STRI, and the molecular research lab at STRI's Naos Marine Laboratories). The extraction of DNA from mycelia, PCR amplification, and bidirectional sequencing methods followed each lab’s specific protocols and those specifics will not be reported here (but see Table 3.3 for details about the primer pairs used).

Forward and reverse sequence reads were assembled using Sequencher 5.2 (Gene Codes, Ann Arbor, MI, USA). For seven isolates, either only the forward read successfully amplified or the forward and reverse reads failed to form a consensus region of DNA, resulting in unidirectional reads. For those seven isolates, we used the forward read to estimate taxonomic placement and assign membership in operational taxonomic units because: (i) only a forward read was successfully amplified for some isolates, (ii)

we used a variety of primers and the binding sites for the forward primers ITS1-F and ITS5 are close together, and (iii) the forward read includes only a small portion of the less variable small subunit (SSU) genic region of the rDNA (Fig. 3.2, Table 3.3). All ITS sequences were manually trimmed and edited in Sequencher 5.2. Sequence data will be made publicly available in the National Center for Biotechnology Information (NCBI) GenBank database.

Phylogenetic analyses

For preliminary identification, 86 edited consensus sequences and seven unidirectional, forward reads were referenced against the 172,000+ fungal ITS DNA sequences in the GenBank database (Schoch et al. 2012) via the NCBI's nucleotide Basic Local Alignment Search Tool (BLASTn) algorithm (accessed September 2014; Altschul et al. 1990).

Given the problems and limitations of the taxonomic assignments of sequences deposited in GenBank (e.g., low-quality, unidentified, and incorrectly named sequences and the lack of sequences for numerous described fungi; Kang et al. 2010, Schoch et al. 2012), we used maximum likelihood and Bayesian phylogenetic analyses to estimate the taxonomic placements of the fungal isolates with greater confidence (following Higginbotham et al. 2014). To guide this process, all 93 sequences were aligned as one group using the web-based Multiple Sequence Comparison by Log- Expectation (MUSCLE) tool (Edgar 2004) and, in the resulting alignment, we identified clusters of sequences that aligned well to one another (N = 33; Table 3.4).

Clusters of sequences that aligned easily were treated as groups of apparently closely related strains and each group was analyzed as a distinct dataset. Datasets contained one to 13 of the 93 fungal sequences. For each dataset, all sequences were referenced against sequences in GenBank and the top 50 BLASTn matches for each sequence were downloaded. The top hits for all isolates in a given dataset were compiled and then redundant sequences and sequences from potentially misidentified strains and/or unvouchered specimens were removed from the compilation. Whenever possible, we included at least one sequence from a reliable culture collection (e.g., ATCC - American Type Culture Collection, Manassas, Virginia) in each dataset. Based on the named sequences in each dataset, we selected outgroups by reviewing the literature. Sequence data for the outgroups were acquired from GenBank. Each dataset was then aligned individually in MUSCLE and the alignments were trimmed to relatively consistent starting and ending points in Mesquite 2.75 (Maddison and Maddison 2011).

To develop phylogenetic hypotheses using maximum likelihood (ML) and Bayesian methods, it is necessary to specify a model of nucleotide substitution. For each dataset, different models of substitution were compared in R ver. 3.1.1 (R Core Team 2014) using the ‘modelTest’ function in the package ‘phangorn’ (Schliep 2011). For 30 of the 33 datasets, the general time reversible (GTR) model with gamma distributed rate variation among sites (G) and a proportion of invariable sites (I) had the most support. For trees AB, AD, and H (see Appendix), the best model of substitution was GTR+I.

Phylogenetic trees were inferred by (i) maximum likelihood (support determined by 100 bootstrap replicates; starting tree generated by a fast ML stepwise-addition algorithm) using the Genetic Algorithm for Rapid Likelihood Inference web service

(GARLI 2.1) hosted at molecularevolution.org (Zwickl 2006, Bazinet et al. 2014) and by (ii) Bayesian methods (5 million generations, four chains, two runs, random starting trees, sampling every 1,000th tree, and the first 25 % of samples from the cold chain discarded as burn-in) using MrBayes ver. 3.2.2 (Ronquist and Huelsenbeck 2003) accessed via the CIPRES Science Gateway web portal (Miller et al. 2010). The trees were visualized in FigTree ver. 1.4.2 (Rambaut 2007). The topologies reflect the majority rule consensus trees based on maximum likelihood analyses and support for each clade is presented as ML bootstrap values (≥50%) and Bayesian posterior probabilities (≥50%). The resulting trees (labeled A-AG) are included in the Appendix.

Many fungi are pleomorphic, meaning that a single fungus can produce several types of spores (sexual and asexual) at different times. For many of these pleomorphic fungi, the teleomorph state (sexual spores) and the anamorph state(s) (asexual spores or no spores) have been given different Latin binomials. Recent molecular studies have clarified connections between teleomorphs and anamorphs (Kirk et al. 2001). For sequences with high similarity to anamorphic fungi, we examined the top BLAST matches for named teleomorphs and consulted the 9th edition of the Dictionary of Fungi (Kirk et al. 2001) and published phylogenetic studies. Herein, we refer to the fungi by their teleomorph names but their associated anamorphic names can be found in Table 3.4.

Assigning operational taxonomic units

Using the program Sequencher 5.2, the 93 ITS sequences were assembled into operational taxonomic units (OTUs) based on different thresholds of sequence similarity and at least 40% sequence overlap. Only those sequences ≥ 350 bp were included and

seven of the 93 sequences clustered into OTUs were forward, unidirectional reads. As stated above, unidirectional reads were only used when only the forward read successfully amplified or the forward and reverse reads failed to overlap to form a consensus region of DNA.

Sequence similarity is only a proxy for delineating taxonomic units. Past studies have used different thresholds, usually 95-97% sequence similarity, for species delineation (O'Brien et al. 2005, U'Ren et al. 2009, Hersh et al. 2012). There is a wide range of intraspecific and interspecific variation reported in the literature for the ITS rDNA sequence. As described in O'Brien et al. (2005), >99% ITS sequence similarity has been observed for different species of fungi (i.e., different species may differ in <1% of their ITS sequences). Conversely, ≤90% ITS sequence similarity has been observed for members of the same species. As the amount of sequence similarity within versus between biological species of tropical fungal phytopathogens is unknown and relaxing or tightening the threshold of sequence similarity has the potential to alter observed richness and diversity and between community comparisons, we assembled the sequences into OTUs for each of a range of similarity values (90, 95, 97, and 99%). However, based on Arnold and Lutzoni (2007) and U'Ren et al. (2009), we consider 95% sequence similarity to be a proxy for species and refer to OTUs defined by 95% sequence similarity as "species" hereafter.

Ecological analyses

We extrapolated accumulation curves for operational taxonomic units (OTUs) defined by 90-99% ITS sequence similarity using the "specaccum" function (Oksanen et

al. 2013). The curves incorporate fungi from all seedlings collected from the forest sites (N = 75 seedlings) and represent the mean OTU accumulation of 100 randomizations of seedling order derived from the observed richness. We plotted the abundance distributions based on Fisher's log-series for OTUs defined by 90-99% ITS sequence similarity using the “fisherfit” function (Oksanen et al. 2013).

Here, we report observed OTU richness (S_{obs}), the number of singletons and their contribution to the observed richness, abundance-based estimates of extrapolated OTU richness [Chao1 (classic formula) and Abundance Coverage-based Estimator (ACE)], and diversity (Fisher’s alpha) for: (i) all fungi isolated from symptomatic seedlings collected from the forest sites, (ii) the assemblage of fungi isolated from symptomatic seedlings collected from the drier forests, and (iii) the assemblage of fungi isolated from symptomatic seedlings collected from the wetter forest. We report both diversity and richness values and multiple estimates to allow for comparison with existing and future studies. For the abundance-based estimates of OTU richness, the bias-corrected formula for Chao1 was used when Chao’s estimated coefficient of variation (CV), used to characterize the degree of heterogeneity among species discovery probabilities, was less than 0.5. For datasets with a CV > 0.5, the classic formula for Chao1 was used instead of the bias-corrected formula because the bias-corrected formula becomes imprecise when CV > 0.5 (Colwell 2013). Diversity was measured by Fisher’s alpha (α) because its assumption that the abundance of species fits a log-series distribution normalizes for sample size, making it reasonably robust for comparing unequal sample sizes (Leigh 1999). Furthermore, Fisher’s alpha is less sensitive to small sample sizes than other diversity indices (e.g., Simpson’s index; Condit et al. 1996).

To explore the relationship between annual precipitation and phytopathogen community composition and the spatial distributions of phytopathogens, we compared the phytopathogens isolated from diseased seedlings collected from the drier forests with the phytopathogens isolated from diseased seedlings collected from the wetter forests using community similarity indices. Only 12 seedlings (represented by 12 fungal isolates) were collected from the four forest sites categorized as mid-range annual precipitation so that category was not included in the analyses. To compare the similarity of communities (species overlap) of phytopathogens in the drier and wetter forests, we first used the classic and commonly used Jaccard index of similarity, which is based on incidence data (i.e., presence/absence, ignoring relative abundance), to allow for comparison with other studies. The Jaccard index of similarity ranges from 0 to 1, with 0 indicating no species overlap and 1 indicating full species overlap. Second, we used Chao's abundance-based Jaccard similarity index, which was developed to reduce the bias (generally, an underestimation of similarity) associated with small sample sizes, unequal sampling, and diverse assemblages with a large proportion of rare species by accounting for unseen species (Chao et al. 2005). Third, we used a nonparametric analysis of variance using distance matrices (adonis) with 999 permutations and Chao as the method to calculate pairwise distances (Chao takes into account unseen species) to test for differences in the two groups' centroids. Because 'adonis' is sensitive to dispersion or spread effects (Anderson 2001), we tested whether dispersion differs between wetter and drier forests (homogeneity of variance) using the 'betadisper' function with Chao as the distance method and the bias correction for small and unequal sample sizes. The phytopathogen communities in the drier and wetter forests have homogenous dispersions (average

distance to median: drier forests = 0.65, wetter forest = 0.67; $F_{1,23} = 0.75$, $P = 0.395$). The homogeneity of dispersion results are qualitatively similar for 90, 97, and 99% sequence similarity. The significance of the fitted model was analyzed with a standard parametric ANOVA. For the adonis and betadisper analyses, OTU frequencies were summed for each host species by precipitation category combination (e.g., *Anacardium excelsum* seedlings collected from drier forests would be one experimental unit).

Whether phytopathogens in tropical forests are host specific or host generalized remains to be determined; however, recognizing that a given fungus can exist as both an asymptomatic endophyte and a disease-causing pathogen (Alvarez-Loayza et al. 2011), there is compelling evidence for host generalism from studies of endophytes of tropical grasses (Higgins et al. 2011), fungi associated with tropical seeds (Kluger et al. 2008), fungal pathogens of tropical leaves (Gilbert and Webb 2007), wood-decaying fungi (Ferrer and Gilbert 2003), and oomycota and fungal pathogens of tropical seedlings (Augspurger and Wilkinson 2007, Schweizer et al. 2013). To account for the possibility that the species identities of the seedlings collected influenced the phytopathogens detected and because seedlings of given species were not necessarily collected from forests representing both precipitation categories, we used three datasets to compare phytopathogen community similarity between the wetter and drier forests. The first dataset includes all phytopathogens from all seedlings collected from the wetter and drier forests ($N = 78$ fungal isolates, $N = 17$ tree species) regardless of whether seedlings of a given tree species were collected in both categories of precipitation. For that dataset, seedlings of 13 tree species were collected from the drier forests and seedlings of 15 tree species were collected in the wetter forest. The second dataset includes all

phytopathogens (*N* = 64 fungal isolates) from seedlings of host tree species for which at least one seedling was collected in both the wetter and drier forests (*N* = 9 tree species). The third dataset includes phytopathogens from seedlings of a single species, *Dalbergia retusa*, collected in a drier forest (PNM, *N* = 11 seedlings and 12 fungal isolates) and a wetter forest (SRR, *N* = 7 seedlings and 8 fungal isolates) in one year (2012). In addition to controlling for species identity, the third dataset removes potential year-to-year variation (the full study was conducted over four years) and between-drier-forest variation (for the full study, seedlings were collected from two drier forests and one wetter forest).

Ecological analyses were performed in R ver. 3.1.1 (R Core Team 2014) using the package 'vegan' (Oksanen et al. 2013) and in EstimateS (Colwell 2013). Community overlap was visualized using the web application BioVenn (Hulsen et al. 2008).

Results

Identities of the phytopathogens

Culturable fungi were recovered from 77 seedlings with observable symptoms of pathogen attack, yielding 93 isolates (Table 3.4). Initial investigations of taxonomic placement were made by referencing our sequence data against those sequences deposited in GenBank via BLASTn. Five of the 93 isolates had a top match to uncultured fungal clones, 39 had a top match to cultured but unidentified fungi (i.e., no taxonomic information was provided), and 30 had a top match to strains from unpublished studies (Table 3.4). Fifty-two of the 93 isolates had top matches to named strains, 40 to genus, one to order, nine to class, and two to phylum (Table 3.4). The top matches to named

strains tentatively suggested placement in the Sordariomycetes (Diaporthales, Hypocreales, and Xylariales), Dothideomycetes (Botryosphaeriales and Capnodiales), and Eurotiomycetes (Eurotiales).

We used phylogenetic analyses to assign taxonomic placements with greater assurance. Encouragingly, identifications based on the top BLAST hits agreed with our phylogenetically-informed estimations for 39 of the 93 isolates (Table 3.4). Of the 39 congruencies, in 6 cases our phylogenetic analyses provided greater taxonomic resolution than the top BLAST hits (Table 3.4). Still, there were some incongruities. For six of our isolates, their top BLAST hits appear to be misidentified at the genus-level based on our phylogenetic analyses (Table 3.4). For eight of our isolates, their top BLAST hits were identified to genus and, based on our phylogenetic analyses, we could only confidently assign family-level placements (Table 3.4). Finally, of the 41 top hits that had no taxonomic information, we were able assign 34 to genus, one to family, and five to class, as a result of our phylogenetic analyses.

Based on our phylogenetic estimations, all 93 fungal isolates belong to the phylum (division) Ascomycota and its largest subphylum, Pezizomycotina (Spatafora et al. 2006). Class could be confidently assigned for 75% of the isolates (70 of 93). For those fungal isolates for which class could be confidently assigned, most are Sordariomycetes (90%) and the rest are Dothideomycetes (6%) and Eurotiomycetes (4%). We tentatively classified an additional 23 isolates as Sordariomycetes, 13 of which are estimated to be members of the genus *Mycoleptodiscus* (Magnaporthaceae) and eight of which are estimated to be members of the genus *Glomerella* (Glomerellaceae). Both genera have uncertain placement within the Sordariomycetes (Kirk et al. 2001). The other

two isolates (07.TB262 and 07.TB50) tentatively classified as Sordariomycetes were not closely related to any reliable, named sequences in GenBank (Table 3.4; see tree G in Appendix). Isolates 07.TB262 (532 bp) and 07.TB50 (527 bp) had 94 and 95% sequence similarity, respectively, to their closest, cultured matches in GenBank, identified as leaf litter ascomycetes (Table 3.4).

The 93 fungal isolates represent nine orders and 12 families (Fig. 3.3, Table 3.4). Sixty-eight of the 93 isolates could confidently be assigned to 17 genera (Fig. 3.3, Table 3.4, Appendix). The Hypocreales was the most commonly observed order (41% of the isolates), followed by the Magnaporthales (14% of isolates) (Fig. 3.3). The five genera most frequently isolated were *Mycoleptodiscus*, *Glomerella*, *Bionectria*, *Diaporthe*, and *Calonectria* (Fig. 3.3, Tables 3.4 and 3.5). Considering the two most frequently isolated genera, the 13 *Mycoleptodiscus* sp. isolates were isolated from seedlings collected from four forest sites, representing all three categories of precipitation (dry, mid, and wet), and seven tree species, representing six tree families (Table 3.4). The eight *Glomerella* sp. isolates were isolated from three forest sites, all three categories of precipitation, and three tree species representing two tree families (Table 3.4). Five of the eight *Glomerella* isolates were isolated from *Dalbergia retusa* (Fabaceae) seedlings (Table 3.4).

The phytopathogens that we isolated from diseased seedlings are related to fungi isolated by Panama-based studies of pathogens of seeds of four Neotropical tree species (e.g., isolate 2010.65b2; see tree AE in Appendix; Kluger et al. 2008; U'Ren et al. 2009) and endophytes in tropical grasses (e.g., isolate 2012.151S; see tree E in Appendix; Higgins et al. 2011), ferns, and trees (Higginbotham et al. 2013).

Richness and diversity of phytopathogens considering all forests

For all sequence similarity thresholds for assembling operational taxonomic units (OTUs), the species accumulation curves are nonasymptotic, which is indicative of incomplete sampling and high diversity. Curve steepness increases with increased sequence similarity stringency (Fig. 3.4). The 90 fungal isolates isolated from 75 seedlings represent 19 to 43 OTUs, for 90 to 99% sequence similarity, respectively, and diversity, as measured by Fisher's α, ranges from 7.35 to 32.29 (Table 3.6). As expected, the proportion of rare OTUs increases as the threshold for sequence similarity is increased. For 90-99% sequence similarity, the number of singleton OTUs ranges from six to 27 (32 - 63% of observed OTUs), respectively (Table 3.6). For abundance-based richness estimators, the extrapolated richness values exceed observed species richness for all levels of sequence similarity, ranging from 21.5 to 94.3 OTUs for 90 to 99% sequence similarity, respectively (Table 3.4).

Based on 95% sequence similarity, the 90 fungal isolates represent 28 species of phytopathogens. The frequency with which a fungal species was isolated ranges from once to 13 times. Most of the species are rare and relatively few are abundant. We isolated 10 of the 28 species only once and eight of the 28 species only twice (Fig. 3.5b, Table 3.4). The two most frequently isolated species include 28% of the 90 isolates (Table 3.4).

Richness and diversity of phytopathogen communities in drier vs. wetter forests

The observed and estimated richness of phytopathogens is greater in the wetter forest (20 phytopathogen species from 35 isolates and 28 seedlings, with an estimated species richness (ACE) of 38.32) than in the drier forests (16 phytopathogen species from 43 isolates and 36 seedlings, with an estimated species richness (ACE) of 19.59) (Table 3.7). A greater proportion of the phytopathogen species are represented by singletons in the wetter forests than in the drier forests (60% versus 31%) and the community of phytopathogen species observed in the wetter forest is more diverse than that observed in the drier forest [Fisher's α: 9.23 (SD = 2.56) versus 19.39 (SD = 5.87)] (Table 3.7). These richness and diversity trends are consistent across sequence similarity thresholds (Table 3.7).

Seedlings of all tree species were not harvested from both the drier and wetter forests and seedlings were harvested over multiple years. Therefore, to minimize the potential variation introduced by differences in host tree species identities in the wetter versus drier forests, we reanalyzed community richness and diversity based on a subset of fungal isolates (N = 64) that were isolated only from the nine tree species for which at least one seedling of each species was collected in both categories of precipitation. Consistent with the full dataset, the community of phytopathogen species observed in the wetter forest is richer and more diverse than that observed in the drier forest [observed (estimated) richness: 18 (44.56) vs. 16 (23); Fisher's α: 25.89 (SD = 10.35) vs. 10.24 (SD = 2.74)] for the 64 isolates from the nine tree species collected in both categories of precipitation (Table 3.8). To control for host species and year, we reanalyzed community

richness and diversity using a subset of fungal isolates (N = 20) that were isolated from seedlings of a single species, *Dalbergia retusa*, collected in one drier forest (PNM) and one wetter forest (SRR) in one year (2012). In contrast with the larger datasets with fungal isolates from seedlings of nine or more tree species, the community of phytopathogen species observed in the drier forest is richer and more diverse than that observed in the wetter forest [observed (estimated) richness: 7 (11.18) vs. 5 (8.68); Fisher's α: 7.02 (SD = 3.67) vs. 5.7 (SD = 3.8)] based on 12 isolates from *D. retusa* seedlings collected from the drier forest and eight isolates from *D. retusa* seedlings from the wetter forest. However, conclusions about differences in diversity based on the isolates from *D. retusa* seedlings should be treated with caution given the very small sample sizes.

Similarity of phytopathogen communities in drier vs. wetter forests

Despite differences in richness and diversity, the communities of phytopathogens in the wetter and drier forests are moderately similar sharing nine of the 27 observed species (classic Jaccard similarity coefficient = 0.33; Fig. 3.3, Table 3.9). Taking into consideration rare and unseen species, the similarity of the two communities is even greater. The wetter and drier forests share an estimated 14.8 species with a Chao-Jaccard abundance-based similarity index of 0.55 (SD = 0.13) (Table 3.9). Consistent with the full dataset, the communities of phytopathogens in the wetter and drier forests are moderately similar based on the subset of fungal isolates (N = 64) including only the nine tree species for which at least one seedling of each species was collected in both the

wetter and drier forests [9 of the 25 phytopathogen species are shared; classic Jaccard similarity index = 0.36; Chao-Jaccard abundance-based similarity index = 0.76 (SD = 0.21); Table 3.10]. Finally, an overlap in phytopathogen species between the wetter and drier forests still exists when controlling for host species and year. For phytopathogens isolated from seedlings of a single species, *Dalbergia retusa*, collected in one drier forest (PNM) and one wetter forest (SRR) in one year (2012), five phytopathogen species are unique to the drier forests, three species are unique to the wetter, and two species are shared between the two precipitation categories (classic Jaccard similarity index = 0.2; Chao-Jaccard abundance-based similarity index = 0.38 (SD = 0.23).

When comparing community composition for species of phytopathogens isolated from all seedlings collected from wetter and drier forests, annual precipitation explains a very small proportion of the sample variation and is not a significant predictor of phytopathogen community structure (adonis: $F_{1,24} = 0.99$, $P = 0.471$, $R^2 = 0.04$). The same is true for the other sequence similarity thresholds (90, 97, and 99%; results not reported here).

For those fungal isolates for which genus-level could be confidently assigned, eight of the 17 genera observed in the study are shared between the wetter and drier forests. *Mycoleptodiscus* is the most commonly isolated genus in both the wetter and drier forests. Five genera were only isolated from seedlings collected from the wetter forest (singletons of *Gibberella*, *Mycosphaerella*, and *Talaromyces*, two *Hypocrea* isolates, and three *Calonectria* isolates) and two genera were only isolated from seedlings collected from the drier forests (one *Emericella* isolate and four *Pestalotiopsis* isolates).

Discussion

Identities of the phytopathogens

All 93 isolates belong to the phylum (division) Ascomycota, which is the largest phylum of Fungi (Kirk et al. 2001). These make up the majority of described fungi and are ecologically diverse, including saprophytes involved in decomposition and nutrient cycling, mutualistic endophytes, and pathogens of plants (Kirk et al. 2001). Similarly, the vast majority of our fungal isolates (90%) are estimated to be members of the class Sordariomycetes, one of the largest classes in the Ascomycota (Kirk et al. 2001). Finally, the five genera most commonly isolated from diseased seedlings, *Mycoleptodiscus*, *Glomerella*, *Bionectria*, *Diaporthe*, and *Calonectria* (Fig. 3.3, Tables 3.4 and 3.5), include species known to be phytopathogens. Moreover, these genera overlap with seedling pathogens in a temperate forest (specifically, *Gomerella*, *Bionectria*, and *Diaporthe*; Hersh et al. 2012).

Those isolates not closely related to any reliable, named sequences in GenBank (e.g., 07.TB262 and 07.TB50; see tree G in Appendix; Table 3.4) may represent unknown taxa or described taxa that have not yet been sequenced. Further analyses are required to clarify their taxonomic affiliations.

Based on their internal transcribed spacer (ITS) region sequences, the fungi that we isolated from tree seedlings with pathogen-caused damage are related to fungi isolated during Panama-based studies of seed-infecting fungi (Kluger et al. 2008, U'Ren et al. 2011) and endophytes in tropical grasses (Higgins et al. 2011), ferns, and trees (Higginbotham et al. 2013). Given that seeds and seedlings are linked life stages and that both are in close contact with soil, it is not surprising that seeds and seedlings are infected

by related fungi. Yet, there is no overlap between the host plant species in the seed studies and in our study. While the maintenance of local plant community diversity by phytopathogens has frequently been ascribed to host-specific phytopathogens in accordance with the Janzen-Connell hypothesis (Comita et al. 2014), this result suggests that plant-associated fungi have wide host ranges. In our study, host generalism is further supported by the fact that some of the fungal phytopathogens that we isolated from tree seedlings have high sequence similarity with endophytic fungi isolated from Neotropical grasses (e.g., isolate 2012.151S; see tree E in Appendix; Table 3.4; Higgins et al. 2011). Considering only the fungi isolated in this study, the two most commonly isolated species, *Mycoleptodiscus* sp. and Nectriaceae sp., were each isolated from seven tree species, representing six families (see species 2 and 5 based on 95% sequence similarity in Table 3.4). While the scope of this study is limited, these observations lead to the hypothesis that generalized phytopathogens may be common in tropical forests. Given the relative rarity of tree species in diverse tropical forests and the passive dispersal of fungal phytopathogens, selection should favor phytopathogens capable of attacking multiple hosts (May 1991). Likewise, there is experimental evidence that phytopathogens in tropical forests are able to attack multiple host species (Augspurger and Wilkinson 2007, Gilbert and Webb 2007, Schweizer et al. 2013).

Many fungi colonize the living tissue of plants asymptomatically and sometimes as mutualistic endophytes (e.g., Arnold et al. 2003). However, the same fungi can become antagonistic phytopathogens under different environmental conditions, when a host's health is compromised, or in a different host species (Delaye et al. 2013, Stergiopoulos and Gordon 2014). For example, the fungal endophyte, *Diplodia mutila*, is

a common, asymptomatic endophyte in the mature plants of *Iriartea deltoidea* and in *I. deltoidea* seedlings in the shaded understory, but it causes disease in *I. deltoidea* seedlings growing in high light environments (Alvarez-Loayza et al. 2011). Therefore, it is not extraordinary that the phytopathogens isolated in our study are closely related to fungi isolated from apparently healthy plant tissue. The fact that a given fungus can have positive, neutral, or negative effects on its host(s) depending on host identity, host condition, or abiotic factors further complicates our ability to designate fungi as pathogenic.

Sampling efficacy

The species accumulation curves indicate statistically incomplete sampling and 19 (68%) of the 28 phytopathogen species detected were singletons or doubletons, which means that our conclusions about richness, diversity, community similarity, and distributions should be treated as tentative and that more intensive sampling is needed (more forest sites and host species). Culture-based techniques for detecting phytopathogens and evaluating their distributions are time- and resource-intensive. Furthermore, a direct comparison of culturing and culture-free, environmental PCR suggests that culturing underestimates fungal diversity and distorts the taxonomic composition of fungal communities (Arnold et al. 2007), as some plant-associated fungi cannot be cultured and some fungi grow slowly in culture and, thus, go unobserved relative to fungi that grow quickly in culture.

Recently, culture-independent molecular methods have detected staggering fungal diversity relative to that detected by traditional culture-based methods (e.g., O'Brien et al.

2005). Even accounting for differences in sampling effort, the community richness of phytopathogens in our culture-based study is dramatically less than the richness observed by a culture-independent, molecular survey of fungal endophyte communities associated with a single host species across a rainfall gradient in Hawaii (40-257 fungal species per tree and >42,000 species overall (Zimmerman and Vitousek 2012). New sequencing technology would provide higher resolution data for the phytopathogen communities in the forests spanning the rainfall gradient. While molecular techniques can overcome some of the labor and time limitations of culture-based surveys, a serious limitation of DNA sequence-based methods is that ecological functions cannot be assigned to the detected organisms. Therefore, both culture-based and culture-independent molecular sampling should be used in combination.

Richness and diversity of phytopathogens considering all forests

For all 90 fungal isolates (isolated from 75 symptomatic seedlings, representing 21 tree species, that were collected from seven forests), we observed 28 phytopathogen species (ACE estimated richness of 37.32; Fisher's $\alpha = 13.93$, SD = 2.96; Table 3.6). The richness of phytopathogen species observed in our study (28 species, based on 95% sequence similarity, from 75 seedlings) is lower than that observed in a culture-based study of seedling pathogens in a temperate forest (130 species, based on 96% sequence similarity, from 293 seedlings; Hersh 2009); however, more seedlings were collected for fungal isolation in the temperate study. Furthermore, in the temperate study, fungi were isolated from dead and dying seedlings as opposed to symptomatic and dying seedlings in

our study. This suggests that the temperate study may include both saprobes and pathogens, which may have contributed to the higher observed richness. Thus, we cannot make any inferences about the diversity of the phytopathogen communities in temperate versus tropical forests.

The phytopathogens of seedlings detected by our study represent greater genotypic diversity (defined by 99% sequence similarity; Gallery et al. 2007) than was observed by a Panama-based study exploring pathogens of seeds [our study: Fisher's α = 32.29, SD = 5.7 based on 90 fungal isolates (Table 3.6); seed study: Fisher's α = 18.3 based on 141 fungal isolates; Kluger et al. 2008]. It is possible that the greater genotypic richness and diversity of seedling pathogens relative to seed pathogens is related to the number of host species sampled (21 host tree species in our study vs. four host tree species in Kluger et al. 2008).

While the phytopathogens of seedlings in our study represent greater genotypic diversity than was observed for pathogens of seeds in Panama (Kluger et al. 2008), the diversity of fungal species (defined by 95% sequence similarity) observed in our study (Fisher's α = 13.93) is half of the observed diversity of fungal endophyte species in a single tropical forest in Panama [Fisher's α of 30.9 based on 100 endophyte isolates from the leaves of six tree species (16-40 isolates/tree species); Arnold and Lutzoni 2007]. This difference in diversity tentatively suggests that the phytopathogen communities may be less diverse than the endophyte communities. This and other unanswered questions require further investigation.

Richness and diversity of phytopathogen communities in drier vs. wetter forests

The community of phytopathogens in the wetter forest is richer and more diverse than the community in the drier forests (Tables 3.7 and 3.8). Similarly, considering all soil fungi across the same rainfall gradient in Panama, community richness and diversity were greatest in the wettest forest site sampled (McGuire et al. 2012) and, across a rainfall gradient in Hawaii, richness correlates positively with precipitation for fungal endophyte communities (Zimmerman and Vitousek 2012). In Panama, greater richness and diversity of fungal communities in the wetter forests relative to the drier forests may be because the wetter forests support fungi unable to tolerate drying out during the longer dry season (Condit 1998) and frequent dry spells during the wet season (Engelbrecht et al. 2006) that characterize the drier forests.

However, annual precipitation explained a very small proportion of community variation and was not a significant predictor of phytopathogen community structure (adonis: $F_{1,24} = 0.99$, $P = 0.471$, $R^2 = 0.04$). In addition to differing in annual precipitation, the wetter and drier forests host different plant communities (Pyke et al. 2001) and have different soils (Brenes-Arguedas et al. 2008, Condit et al. 2013) and understory light levels (Brenes-Arguedas et al. 2011). The greater diversity of phytopathogens in the wetter versus drier forests could also be the result of a greater diversity of tree species in the wetter forests (average Fisher's α of 80.58 vs. 19.81, respectively, for trees ≥ 10 cm diameter at breast height in 1-ha forest inventory plots; Pyke et al. 2001). Then again, McGuire et al. (2012) showed a positive correlation between soil fungal richness and precipitation, independent of plant diversity, across the

same precipitation gradient. As our study is observational, the spatial patterns identified here are purely correlative and it is not possible to conclude which abiotic and/or biotic factors are shaping the phytopathogen communities. Future work should adopt experimental approaches to tease apart the importance and relative contributions of these biotic and abiotic variables in driving spatial variation in phytopathogen community composition.

Similarity of phytopathogen communities in drier vs. wetter forests

While the community of phytopathogens in the wetter forest is richer and more diverse than that of the drier forests (Tables 3.7 and 3.8), the two communities share 33% of their species (Fig. 3.6, Tables 3.9 and 3.10). Similarly, a single phytopathogen species was isolated from seedlings collected from four forest sites, representing the dry, mid, and wet precipitation categories (see OTU 2 defined by 95% sequence similarity in Table 3.3). These results are surprising for several reasons. First, given the estimated richness of fungi in tropical forests, the likelihood of observing the same species in multiple forests seems low. Given our limited sampling, one might even expect to observe only singletons. Furthermore, the overlap between the phytopathogen communities in the wetter and drier forests (33% of species shared) is greater than that of the tree communities in the wetter and drier forests (forest inventory plots 50 km apart share ca. 1-15% of their tree species; Condit et al. 2002). Second, the wetter forest site is separated from the two drier forest sites by ca. 45 km and evidence suggests that the dispersal ability of plant-associated fungi is geographically limited (Gonthier et al. 2001, Peay et

al. 2012, Higgins et al. 2014). Yet, dispersal ability is not always an important determinant of range size (Lester et al. 2007) and the community similarity and unexpectedly broad spatial distributions observed in this study have also been observed for root-associated fungal species in North America (Queloz et al. 2011). This leads us to ask how they are dispersed.

Although fungal propagules are dispersed passively and most travel very short distances from their source, long-distance dispersal is possible via strong winds, moving water bodies, and the transportation of infected plant tissue (Ristaino and Gumpertz 2000, Davidson et al. 2005, Hyder et al. 2009, Peterson at al. 2014). Furthermore, Martiny et al. (2006) hypothesize that, for microorganisms, the likelihood that at least one propagule will travel a long distance and establish a new population increases with increased propagule production, as is true for passively dispersed macroorganisms (e.g., plant seeds). Conversely, the relatively wide spatial distributions of the phytopathogens may not be due to long-distance dispersal but rather to numerous short-distance dispersals over a long period of time. The forest sites from which symptomatic seedlings were collected are part of an almost continuous band of forest extending between the Caribbean Sea and Pacific Ocean, meaning that habitable areas exist between the sites. Thus, the observed overlap between communities and relatively broad spatial distributions suggest that at least some of the phytopathogen species produce propagules conducive to long-distance dispersal and/or that their propagules have slowly dispersed short distances, leading to relatively wide ranges over time. Phytopathogens with broad geographic distributions may mean that plant hosts cannot escape disease in geographic space, which has important implications for the distance-dependent assumptions of the

Janzen-Connell hypothesis and the enemy release hypothesis (Janzen 1970, Connell 1971, Keane and Crawley 2002).

While a third of the phytopathogens were shared, we did not observe complete overlap between the communities in the drier and wetter forests. Phytopathogens undoubtedly differ in their resource requirements and tolerance of abiotic conditions and, like the plant communities, their spatial distributions across the rainfall gradient may be determined by habitat suitability. If some are host specialized, their spatial distributions may be restricted by host availability (i.e., the plants' distributions). However, for those taxa that were only isolated from either the drier or wetter forests, it is impossible to make conclusions about their habitat affiliations because they are represented by relatively few isolates.

Does phytopathogen community composition contribute to the elevated risk of pathogen-caused damage and mortality in the wetter forests of Panama?

We observed support for our hypothesis that phytopathogens are relatively widespread geographically, with the phytopathogen communities inhabiting the wetter and drier forests showing 33% species overlap. These results tentatively suggests that the elevated risk of pathogen-caused damage and mortality for seedlings in the wetter versus drier forests (Spear et al. 2015) may be unrelated to compositional differences in the phytopathogen communities. Such a conclusion assumes that the most commonly observed phytopathogens are the most ecologically important phytopathogens. However, as hypothesized, we also observed a greater richness and diversity of phytopathogens in

the wetter forest and it is possible that the phytopathogens unique to the wetter forests are responsible for the increased risk of pathogen attack in those forests.

Alternatively, a greater risk of pathogen attack in the wetter forests may result from the interaction between the abiotic environment and disease development. The abiotic conditions in the wetter forests may favor pathogen reproduction, infection, and/or dispersal (Gilbert 2005, Barrett et al. 2009, Hersh et al. 2012, Swinfield et al. 2012) or the abiotic environment may stress seedlings making them more vulnerable to disease (Barrett et al. 2009). To begin to address these unanswered questions, abiotic conditions (e.g., light and water availability) could be artificially manipulated in the forests to "rescue" seedlings from pathogen attack in the wetter forests by mimicking dry-forest conditions and to increase the risk of pathogen attack for seedlings in the drier forests by mimicking wet-forest conditions.

Conclusions and future directions

There is increasing interest in whether and how phytopathogens influence the spatial turnover of plant species and the regional diversity of plant communities (Defossez et al. 2011, Spear et al. 2015). Our study represents an important first step toward identifying the mechanism(s) responsible for higher pathogen pressure in wetter versus drier forests, which is critical for understanding how pathogens influence the spatial turnover of tree species in the forests of Panama (Spear et al. 2015). Additionally, this study provides the first estimation of the taxonomy, diversity, and spatial structure of tropical phytopathogens across a rainfall gradient and at a landscape scale. Future work should involve more intensive sampling in this system, using culture-based and culture-

independent methods in combination, and should employ experimental manipulations to tease apart the importance and relative contributions of the biotic and abiotic variables driving spatial variation in phytopathogen community composition.

Table 3.1. Symptomatic seedlings were collected from lowland, tropical forests that span a rainfall gradient and the Isthmus of Panama and details for the seven forest sites are included herein. The columns include the identification code, approximate location, and annual precipitation (categorized as dry, mid, or wet) of each site, the number of seedlings collected from each site, the number of fungal isolates from those seedlings, the number of fungal species (defined by 95% sequence similarity) observed, and the number of singletons and their contribution to the observed number of species. Forest sites are coded as follows: 1. PNM = Parque Natural Metropolitano, 2. FC = Gunn Hill in Ciudad de Saber (formerly Fort Clayton), 3. SC = Sendero del Charco, 4. CC = Sendero Camino de Cruces, 5. BCI = Barro Colorado Island, Barro Colorado Nature Monument, 6. BV = Buena Vista Peninsula, Barro Colorado Nature Monument, and 7. SRR = Santa Rita Ridge. See Fig. 3.1b for a map of the sites.

	Forest	Approx. location	Annual precipitation (mm)	Precipitation category	No. of seedlings collected	No. of fungal isolates	No. of fungal species	No. of singletons (% of species)
1.	PNM	8°59’36.62”N, 79°32’36.17”W	1,800[A]	dry	29	35	15	6 (40%)
2.	FC	9°0′50″N, 79°35′W	2,010[B]	dry	7	8	5	2 (40%)
3.	SC	9°4'52.93"N, 79°39'57.92"W	N/A	mid	2	2	2	2 (100%)
4.	CC	9°6'6.44"N, 79°36'56.45"W	N/A	mid	1	1	1	1 (100%)
5.	BCI	9° 9'21.90"N, 79°51'5.80"W	2,600[C]	mid	3	3	3	3 (100%)
6.	BV	9°10'46.20"N, 79°49'54.88"W	2,600[C]	mid	5	6	4	2 (50%)
7.	SRR	9°20’03.71” N, 79°46’39.96”W	3,000[A]	wet	28	35	20	12 (60%)

An additional two seedlings were collected from a shadehouse in Gamboa, Panama (GS), resulting in three fungal isolates, representing three additional fungal species.

References for precipitation values: [A]Santiago et al. (2004), [B]Brenes-Arguedas et al. (2013), [C]Windsor (1990), N/A = not available

Table 3.2. Identities of, and collection details for, the 21 tree species, belonging to 11 families, represented by the 75 symptomatic seedlings collected from forests spanning the rainfall gradient. Columns include the full tree species name, family, the number of seedlings collected for each species, the number of fungal isolates from those seedlings, the number of fungal species (defined by 95% sequence similarity) observed, and the number of singletons and their contribution to the observed number of species. Tree species and family names are based on the Tropicos database (tropicos.org) of the Missouri Botanical Garden.

Tree species	Family	No. of seedlings collected	No. of fungal isolates	No. of fungal species	No. of singletons (% of species)
Anacardium excelsum	Anacardiaceae	9	10	9	8 (89%)
Brosimum utile	Moraceae	1	2	2	2 (100%)
Calophyllum longifolium	Clusiaceae	1	1	1	1 (100%)
Cassia moschata	Fabaceae	4	5	4	3 (75%)
Castilla elastica	Moraceae	5	6	4	3 (75%)
Cochlospermum vitifolium	Bixaceae	3	4	3	2 (67%)
Cojoba rufescens	Fabaceae	1	2	2	2 (100%)
Dalbergia retusa	Fabaceae	18	20	10	6 (60%)
Dipteryx oleifera	Fabaceae	1	1	1	1 (100%)
Genipa americana	Rubiaceae	1	1	1	1 (100%)
Hymenaea courbaril	Fabaceae	5	6	5	4 (80%)
Lacmellea panamensis	Apocynaceae	2	2	2	2 (100%)
Nectandra cuspidata	Lauraceae	4	5	5	5 (100%)
Ormosia coccinea	Fabaceae	1	1	1	1 (100%)
Ormosia macrocalyx	Fabaceae	1	1	1	1 (100%)
Protium tenuifolium	Burseraceae	3	4	4	4 (100%)
Randia armata	Rubiaceae	1	1	1	1 (100%)
Swietenia macrophylla	Meliaceae	2	2	2	2 (100%)
Tetragastris panamensis	Burseraceae	1	1	1	1 (100%)
Trichilia tuberculata	Meliaceae	2	2	2	2 (100%)
Virola surinamensis	Myristicaceae	9	13	8	6 (86%)

An additional two seedlings were collected from a shadehouse in Gamboa, Panama (GS), resulting in three fungal isolates, representing three additional fungal species.

Table 3.3. The name, direction (F = forward, R = reverse), binding region, sequence, and reference for the primers used to amplify the internal transcribed spacer (ITS) region of the fungi isolated from seedlings with pathogen-caused damage. A variety of primer pairs (ITS1F/ITS4, ITS1F/LR3, ITS5/ITS4, ITS5/LR3) were used because the amplifications were conducted in different labs, in different years, and, in some cases, because amplification failed when one of the other pairs was used. See Fig. 3.2 for a diagram of the ITS region and the approximate binding positions of the primers.

Name	Dir.	Genic region of rDNA	Sequence (5'→3')	Reference
ITS1-F	F	small subunit (SSU)	CTTGGTCATTTAGAGGAAGTAA	Gardes and Bruns (1993)
ITS5	F	small subunit (SSU)	GGAAGTAAAAGTCGTAACAAGG	White et al. (1990)
ITS4[B]	R	large subunit (LSU)	TCCTCCGCTTATTGATATGC	White et al. (1990)
LR3[C]	R	large subunit (LSU)	GGTCCGTGTTTCAAGAC	Vilgalys and Hester (1990)

Table 3.4. Operational taxonomic unit (OTU) assignments, collection and identification details, and estimated taxonomic placements for 93 fungal isolates. Columns include OTU assignments based on 90-99% sequence similarity, isolate identifier, primer(s) used and sequence length, host tree species, site from which the seedling was collected (superscripts indicate the site's precipitation category: D = dry, M = mid, W = wet, or NA = not applicable), top match and, in parentheses, its accession number and maximum identity value from BLAST searches performed in September of 2014, taxonomic placement at the genus, family, and, in parentheses, order levels, and the letter code identifying the phylogenetic tree for the isolate (see Appendix). For some isolates, two genera are listed because the teleomorph (sexual) and, in parentheses, the anamorph (asexual) stages of the same fungus were assigned different Latin names.

90	95	97	99	Isolate ID. Primer(s) (Seq Length)	Host tree species	Site	Top BLAST match (accession no.)	Max ident.	Genus (= anamorph)	Family (Order)	Phylo. tree
1	1	25	32	2010.155.ITS5 (699 bp)	*Anacardium excelsum*	SRR^{W}	Uncultured *Nectria* (GU055710.1)	93%	unknown	Nectriaceae (Hypocreales)	AC
1	1	1	1	2007.TB227. ITS5/ITS4 (505 bp)	*Hymenaea courbaril*	FC^{D}	Fungal sp. (FJ613095.1)	100%	*Glionectria* (= *Gliocladiopsis*)	Nectriaceae (Hypocreales)	AD
1	1	1	1	2007.TB52.ITS5/ITS4 (517 bp)	*Hymenaea courbaril*	SRR^{W}	Fungal sp. (FJ613095.1)	99%	*Glionectria* (= *Gliocladiopsis*)	Nectriaceae (Hypocreales)	AD
1	1	1	1	2007.TB219. ITS5/LR3 (1093 bp)	*Protium tenuifolium*	SRR^{W}	Uncultured *Nectria* (GU055710.1)	94%	*Glionectria* (= *Gliocladiopsis*)	Nectriaceae (Hypocreales)	AD
1	1	1	1	2007.TB243. ITS5/ITS4 (516 bp)	*Swietenia macrophylla*	FC^{D}	Fungal sp. (FJ613095.1)	100%	*Glionectria* (= *Gliocladiopsis*)	Nectriaceae (Hypocreales)	AD
2	2	2	2	2007.TB282. ITSIF/LR3 (1078 bp)	*Cassia moschata*	BV^{M}	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	16	2007.TB268. ITS5/ITS4 (543 bp)	*Cassia moschata*	FC^{D}	Fungal sp. (KF164316.1)*	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	16	2007.TB69.ITS5/ITS4 (557 bp)	*Cochlospermum vitifolium*	BV^{M}	Fungal sp. (HM211288.1)*	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	2	2012.151S.ITS5 (750 bp)	*Dalbergia retusa*	PNM^{D}	Fungal endophyte (EU687173.1)	100%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E

Table 3.4. (continued)

90	95	97	99	Isolate ID. Primer(s) (Seq Length)	Host tree species	Site	Top BLAST match (accession no.)	Max ident.	Genus (= anamorph)	Family (Order)	Phylo. tree
2	2	2	2	2012.172R.ITS5/LR3 (1000 bp)	*Dalbergia retusa*	PNM[D]	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	2	2012.183S.ITS5 (688 bp)	*Dalbergia retusa*	PNM[D]	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	2	2012.10R.ITS5/LR3 (989 bp)	*Dalbergia retusa*	SRR[W]	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	2	2012.52S.ITS5/LR3 (1028 bp)	*Dalbergia retusa*	SRR[W]	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	2	2012.96L.ITS5/LR3 (932 bp)	*Dalbergia retusa*	SRR[W]	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	2	2010.ES80.ITS5/ITS4 (727 bp)	*Randia armata*	SRR[W]	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	2	2007.TB242. ITS5/LR3 (1148 bp)	*Swietenia macrophylla*	FC[D]	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	2	2010.168aM. ITS1F/LR3 (1081 bp)	*Tetragastris panamensis*	SRR[W]	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
2	2	2	2	2010.160T.ITS1F/LR3 (1074 bp)	*Virola surinamensis*	SRR[W]	Fungal endophyte (EU687173.1)	99%	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)	E
3	3	3	3	2010.143.ITS5/ITS4 (756 bp)	*Anacardium excelsum*	SRR[W]	Fungal endophyte (EU686869.1)	99%	*Diaporthe* (= *Phomopsis*)	Diaporthaceae (Diaporthales)	Q
3	3	3	3	2007.TB67.ITS5/ITS4 (534 bp)	*Cochlospermum vitifolium*	BV[M]	*Diaporthe* sp. (EF423549.2)	100%	*Diaporthe* (= *Phomopsis*)	Diaporthaceae (Diaporthales)	Q
3	3	27	34	2010.156b.ITS1F/LR3 (1054 bp)	*Cojoba rufescens*	GS[NA]	Sordariomycetes sp. (JQ761037.1)	97%	*Diaporthe* (= *Phomopsis*)	Diaporthaceae (Diaporthales)	O
3	3	3	3	2010.107.2l. ITS1F/LR3 (1051 bp)	*Cojoba rufescens*	PNM[D]	Fungal endophyte (EU687127.1)	99%	*Diaporthe* (= *Phomopsis*)	Diaporthaceae (Diaporthales)	Q
3	3	3	3	2012.133L.ITS5/LR3 (899 bp)	*Dalbergia retusa*	PNM[D]	Fungal endophyte (EU687127.1)	99%	*Diaporthe* (= *Phomopsis*)	Diaporthaceae (Diaporthales)	Q

Table 3.4. (continued)

90	95	97	99	Isolate ID. Primer(s) (Seq Length)	Host tree species	Site	Top BLAST match (accession no.)	Max ident.	Genus (= anamorph)	Family (Order)	Phylo. tree
3	3	20	25	2007.TB294. ITS5/ITS4 (533 bp)	*Ormosia coccinea*	FC[D]	*Diaporthe* sp. (FJ799938.1)	99%	*Diaporthe* (= *Phomopsis*)	Diaporthaceae (Diaporthales)	P
4	4	4	4	2007.TB273. ITS5/LR3 (846 bp)	*Cassia moschata*	SRR[W]	Fungal endophyte (EU687174.1)	99%	unknown	unknown	H
4	4	4	4	2010.64a2.ITS1F/LR3 (1052 bp)	*Castilla elastica*	PNM[D]	Sordariomycetes sp. (JQ760669.1)	100%	unknown	unknown	H
4	4	4	4	2010.107.2h. ITS1F/LR3 (1052 bp)	*Cojoba rufescens*	PNM[D]	Fungal endophyte (EU687157.1)	100%	unknown	unknown	H
4	4	4	4	2012.132S.ITS5/LR3 (880 bp)	*Dalbergia retusa*	PNM[D]	Fungal endophyte (EU687157.1)	100%	unknown	unknown	H
4	4	4	4	2012.153S.ITS5/LR3 (981 bp)	*Dalbergia retusa*	PNM[D]	Sordariomycetes sp. (JQ760777.1)	100%	unknown	unknown	H
4	4	4	4	2010.171.ITS5 (630 bp)	*Hymenaea courbaril*	PNM[D]	Fungal endophyte (EU687157.1)	100%	unknown	unknown	H
4	4	4	4	2010.73bL1. ITS1F/LR3 (1061 bp)	*Virola surinamensis*	PNM[D]	Fungal endophyte (EU687157.1)	99%	unknown	unknown	H
1	5	5	5	2010.aAEC. ITS1F/ITS4 (516 bp)	*Anacardium excelsum*	PNM[D]	*Cylindrocladiella* sp. (EU330631.1)*	99%	*Nectricladiella* (= *Cylindrocladiella*)	Nectriaceae (Hypocreales)	Y
1	5	5	5	2011.8ab.ITS5/ITS4 (788 bp)	*Anacardium excelsum*	SC[M]	*Cylindrocladiella* sp. (JX243750.1)*	99%	unknown	Nectriaceae (Hypocreales)	Z
1	5	5	27	2007.TB299. ITS5/ITS4 (465 bp)	*Brosimum utile*	SRR[W]	*Cylindrocladiella pseudoinfestans* CBS (JN099126.1)	98%	*Nectricladiella* (= *Cylindrocladiella*)	Nectriaceae (Hypocreales)	Y

Table 3.4. (continued)

90	95	97	99	Isolate ID. Primer(s) (Seq Length)	Host tree species	Site	Top BLAST match (accession no.)	Max ident.	Genus (= anamorph)	Family (Order)	Phylo. tree
1	5	5	18	2007.TB258. ITS5/ITS4 (487 bp)	*Cassia moschata*	FC[D]	*Cylindrocladiella* sp. (EU330631.1)*	98%	*Nectricladiella* (= *Cylindrocladiella*)	Nectriaceae (Hypocreales)	Y
1	5	5	10	2007.TB290. ITS5/LR3 (1099 bp)	*Cochlospermum vitifolium*	BV[M]	*Cylindrocladiella* sp. (JX243750.1)*	97%	*Neonectria* (= *Cylindrocarpon)*	Nectriaceae (Hypocreales)	AA
1	5	5	5	2007.TB68.ITS5/ITS4 (503 bp)	*Cochlospermum vitifolium*	BV[M]	*Cylindrocladiella* sp. (EU330631.1)*	99%	*Nectricladiella* (= *Cylindrocladiella*)	Nectriaceae (Hypocreales)	Y
1	5	5	10	2010.76.ITS1F/LR3 (1046 bp)	*Hymenaea courbaril*	SRR[W]	*Cylindrocladiella* sp. (JX243750.1)*	97%	*Neonectria* (= *Cylindrocarpon)*	Nectriaceae (Hypocreales)	AA
1	5	5	10	2007.TB283. ITS5/LR3 (1103 bp)	*Lacmellea panamensis*	FC[D]	*Cylindrocladiella* sp. (JX243750.1)*	97%	*Neonectria* (= *Cylindrocarpon*)	Nectriaceae (Hypocreales)	AA
1	5	5	5	2010.124.ITS1F/LR3 (1033 bp)	*Virola surinamensis*	PNM[D]	*Cylindrocladiella* sp. (JX243750.1)*	99%	unknown	Nectriaceae (Hypocreales)	Z
1	5	5	35	2010.157b.ITS1F/LR3 (1035 bp)	*Virola surinamensis*	PNM[D]	*Cylindrocladiella* sp. (JX243750.1)*	97%	*Neonectria* (= *Cylindrocarpon*)	Nectriaceae (Hypocreales)	X
1	5	5	5	2010.162.ITS1F/LR3 (1026 bp)	*Virola surinamensis*	PNM[D]	*Cylindrocladiella* sp. (JX243750.1)*	99%	unknown	Nectriaceae (Hypocreales)	Z
1	5	5	5	2010.163.ITS1F/LR3 (1030 bp)	*Virola surinamensis*	PNM[D]	*Cylindrocladiella* sp. (JX243750.1)*	99%	unknown	Nectriaceae (Hypocreales)	Z
5	6	6	6	2012.32S.ITS5/LR3 (949 bp)	*Dalbergia retusa*	SRR[W]	*Macrophomina phaseolina* NRRL (KM030327.1)*	97%	unknown	Botryosphaeriaceae (Botryosphaeriales)	M

Table 3.4. (continued)

90	95	97	99	Isolate ID. Primer(s) (Seq Length)	Host tree species	Site	Top BLAST match (accession no.)	Max ident.	Genus (= anamorph)	Family (Order)	Phylo. tree
5	6	6	6	2012.74R.ITS5/LR3 (884 bp)	*Dalbergia retusa*	SRR[W]	*Macrophomina phaseolina* NRRL (KM030327.1)*	98%	unknown	Botryosphaeriaceae (Botryosphaeriales)	M
5	6	6	6	2010.137.ITS1F/LR3 (1087 bp)	*Trichilia tuberculata*	GS[NA]	*Botryosphaeria dothidea* (AB454278.1)	97%	unknown	Botryosphaeriaceae (Botryosphaeriales)	M
6	7	7	7	2010.65.2b. ITS1F/LR3 (1064 bp)	*Castilla elastica*	PNM[D]	Fungal sp. (EU563552.1)	100%	*Nectria* (= *Fusarium*)	Nectriaceae (Hypocreales)	AE
6	7	7	7	2010.NtCE.ITS1F (351 bp)	*Castilla elastica*	PNM[D]	*Fusarium solani* (KJ174400.1)*	100%	*Nectria* (= *Fusarium*)	Nectriaceae (Hypocreales)	AE
6	7	7	7	2010.92.2brn. ITS1F/LR3 (1064 bp)	*Castilla elastica*	SRR[W]	Fungal sp. (EU563552.1)	100%	*Nectria* (= *Fusarium*)	Nectriaceae (Hypocreales)	AE
7	8	8	8	2010.bAEC. ITS1F/LR3 (1049 bp)	*Anacardium excelsum*	PNM[D]	Fungal sp. (EU563608.1)	99%	*Bionectria* (= *Clonostachys*)	Bionectriaceae (Hypocreales)	R
7	8	8	12	2010.93FT. ITS1F/LR3 (1061 bp)	*Castilla elastica*	SRR[W]	*Clonostachys rogersoniana* (KC806293.1)*	99%	*Bionectria* (= *Clonostachys*)	Bionectriaceae (Hypocreales)	R
7	8	8	12	2010.169.ITS1F/LR3 (1062 bp)	*Hymenaea courbaril*	PNM[D]	*Clonostachys rogersoniana* (KC806293.1)*	100%	*Bionectria* (= *Clonostachys*)	Bionectriaceae (Hypocreales)	R
7	8	8	8	2010.83a.ITS1F/LR3 (1016 bp)	*Protium tenuifolium*	SRR[W]	*Bionectria* cf. *ochroleuca* CBS (EU552110.1)	99%	*Bionectria* (= *Clonostachys*)	Bionectriaceae (Hypocreales)	R
7	8	8	8	2010.165a.ITS1F/LR3 (1060 bp)	*Virola surinamensis*	PNM[D]	*Bionectria* cf. *ochroleuca* CBS (EU552110.1)	99%	*Bionectria* (= *Clonostachys*)	Bionectriaceae (Hypocreales)	R
7	8	8	8	2010.95.ITS1F/LR3 (1060 bp)	*Virola surinamensis*	PNM[D]	*Bionectria* cf. *ochroleuca* CBS (EU552110.1)	99%	*Bionectria* (= *Clonostachys*)	Bionectriaceae (Hypocreales)	R

Table 3.4. (continued)

90	95	97	99	Isolate ID. Primer(s) (Seq Length)	Host tree species	Site	Top BLAST match (accession no.)	Max ident.	Genus (= anamorph)	Family (Order)	Phylo. tree
7	8	8	8	2010.81.ITS1F/LR3 (1060 bp)	*Virola surinamensis*	SRR[W]	*Bionectria* cf. *ochroleuca* CBS (EU552110.1)	99%	*Bionectria* (= *Clonostachys*)	Bionectriaceae (Hypocreales)	R
8	9	9	9	2012.111S.ITS5 (710 bp)	*Dalbergia retusa*	PNM[D]	Fungal endophyte (EU687143.1)	100%	*Glomerella* (= *Colletotrichum*)	Glomerellaceae (Glomerellales)	T
8	9	9	9	2012.112S.ITS5/LR3 (962 bp)	*Dalbergia retusa*	PNM[D]	Fungal endophyte (EU687143.1)	100%	*Glomerella* (= *Colletotrichum*)	Glomerellaceae (Glomerellales)	T
8	9	9	9	2012.140S.ITS5/LR3 (945 bp)	*Dalbergia retusa*	PNM[D]	Fungal endophyte (EU687103.1)	100%	*Glomerella* (= *Colletotrichum*)	Glomerellaceae (Glomerellales)	T
8	9	9	9	2012.19S.ITS5/LR3 (849 bp)	*Dalbergia retusa*	SRR[W]	Fungal endophyte (EU687143.1)	99%	*Glomerella* (= *Colletotrichum*)	Glomerellaceae (Glomerellales)	T
8	9	9	9	2011.4.ITS5/ITS4 (603 bp)	*Dipteryx oleifera*	BCI[M]	Fungal endophyte (HM537025.1)*	99%	*Glomerella* (= *Colletotrichum*)	Glomerellaceae (Glomerellales)	T
1	10	10	41	2011.1b2.ITS5/LR3 (873 bp)	*Calophyllum longifolium*	BCI[M]	Fungal endophyte (EU686953.1)	99%	*Calonectria* (= *Cylindrocladium*)	Nectriaceae (Hypocreales)	AG
1	10	10	11	2007.TB43.ITS5/ITS4 (516 bp)	*Lacmellea panamensis*	SRR[W]	*Calonectria pini* (GQ280640.1)	100%	*Calonectria* (= *Cylindrocladium*)	Nectriaceae (Hypocreales)	AG
1	10	10	11	2007.TB36.ITS5/ITS4 (508 bp)	*Nectandra cuspidata*	SRR[W]	Calonectria pini (GQ280640.1)	99%	*Calonectria* (= *Cylindrocladium*)	Nectriaceae (Hypocreales)	AG
1	10	10	11	2010.ES81.ITS5/ITS4 (701 bp)	*Trichilia tuberculata*	SRR[W]	*Calonectria hawksworthii* CBS (GQ280580.1)	99%	*Calonectria* (= *Cylindrocladium*)	Nectriaceae (Hypocreales)	AG
1	10	10	42	2011.7b.ITS5/LR3 (791 bp)	*Anacardium excelsum*	SC[M]	Fungal endophyte (EU687024.1)	99%	*Calonectria* (= *Cylindrocladium*)	Nectriaceae (Hypocreales)	AG
9	11	11	13	2012.31S.ITS5/LR3 (900 bp)	*Dalbergia retusa*	SRR[W]	*Trichoderma spirale* NRRL (KM011996.1)*	99%	*Hypocrea* (= *Trichoderma*)	Hypocreaceae (Hypocreales)	U

Table 3.4. (continued)

90	95	97	99	Isolate ID. Primer(s) (Seq Length)	Host tree species	Site	Top BLAST match (accession no.)	Max ident.	Genus (= anamorph)	Family (Order)	Phylo. Tree
9	11	11	13	2010.ES82. ITS1F/LR3 (1109 bp)	*Genipa americana*	SRR[W]	*Trichoderma spirale* NRRL (KM011996.1)*	100%	*Hypocrea* (= *Trichoderma*)	Hypocreaceae (Hypocreales)	U
10	12	12	14	2011.5.ITS5/ITS4 (749 bp)	*Anacardium excelsum*	PNM[D]	Xylariales sp. (AB511813.1)*	97%	unknown	unknown	K
10	12	12	14	2010.161org. ITS1F/LR3 (1086 bp)	*Virola surinamensis*	SRR[W]	Fungal endophyte (EU687114.1)	96%	unknown	unknown	K
8	13	13	15	2011.2b.ITS5/ITS4 (748 bp)	*Anacardium excelsum*	BCI[M]	Fungal endophyte (EU687012.1)	99%	*Glomerella* (= *Colletotrichum*)	Glomerellaceae (Glomerellales)	T
8	13	13	15	2012.32R.ITS5/LR3 (967 bp)	*Dalbergia retusa*	SRR[W]	Fungal sp. (JQ747654.1)	100%	*Glomerella* (= *Colletotrichum*)	Glomerellaceae (Glomerellales)	T
11	14	14	23	2007.TB286. ITS5/LR3 (1036 bp)	*Nectandra cuspidata*	SRR[W]	*Penicillium funiculosum* NRRL (GQ221866.1)	100%	*Talaromyces* (= *Penicillium*)	Trichocomaceae (Eurotiales)	D
11	14	14	24	2007.TB293. ITS5/ITS4 (469 bp)	*Ormosia macrocalyx*	BV[M]	Uncultured fungus (KJ572249.1)*	96%	*Talaromyces* (= *Penicillium*)	Trichocomaceae (Eurotiales)	D
12	15	15	29	2007.TB50. ITS5/ITS4 (527 bp)	*Nectandra cuspidata*	SRR[W]	Leaf litter ascomycete (AF502895.1)*	95%	unknown	unknown	G
12	15	15	20	2007.TB262. ITS5/ITS4 (532 bp)	*Protium tenuifolium*	SRR[W]	Leaf litter ascomycete (AF502895.1)*	94%	unknown	unknown	G
13	16	33	43	2012.117aS.ITS5/LR3 (889 bp)	*Dalbergia retusa*	PNM[D]	*Pestalotiopsis* sp. (KF746154.1)	99%	*Pestalotiopsis*	Amphisphaeriaceae (Xylariales)	L
13	16	23	30	2010.115.ITS1F/LR3 (1041 bp)	*Virola surinamensis*	PNM[D]	*Pestalotiopsis* sp. (KF746122.1)	100%	*Pestalotiopsis*	Amphisphaeriaceae (Xylariales)	L

Table 3.4. (continued)

90	95	97	99	Isolate ID. Primer(s) (Seq Length)	Host tree species	Site	Top BLAST match (accession no.)	Max ident.	Genus (= anamorph)	Family (Order)	Phylo. Tree
4	17	35	45	2012.159L.ITS5/LR3 (954 bp)	*Dalbergia retusa*	PNM[D]	Sordariomycetes sp. (JQ760671.1)	99%	unknown	Xylariaceae (Xylariales)	I
4	17	18	21	2007.TB270. ITS5/ITS4 (549 bp)	*Nectandra cuspidata*	FC[D]	Fungal sp. (FJ613074.1)	99%	unknown	Xylariaceae (Xylariales)	I
13	18	28	36	2010.172.ITS1F/LR3 (1048 bp)	*Trichilia tuberculata*	PNM[D]	Sordariomycetes sp. (JQ761155.1)	99%	*Pestalotiopsis*	Amphisphaeriaceae (Xylariales)	L
13	18	31	39	2010.79.ITS1F/LR3 (1115 bp)	*Virola surinamensis*	PNM[D]	Sordariomycetes sp. (JQ761153.1)	99%	*Pestalotiopsis*	Amphisphaeriaceae (Xylariales)	L
14	19	16	17	2007.TB24.ITS5/ITS4 (514 bp)	*Hymenaea courbaril*	SRR[W]	*Mycosphaerella* sp. (JQ732918.1)*	96%	*Mycosphaerella*	Mycosphaerellaceae (Capnodiales)	A
15	20	17	19	2007.TB261. ITS5/ITS4 (374 bp)	*Protium tenuifolium*	SRR[W]	*Fusarium* sp. (FJ210567.1)	93%	*Gibberella* (= *Fusarium*)	Nectriaceae (Hypocreales)	V
16	21	19	22	2007.TB274. ITS5/ITS4 (542 bp)	*Cassia moschata*	SRR[W]	Sordariomycetes sp. (AB847015.1)	94%	unknown	Chaetosphaeriaceae (Chaetosphaeriales)	F
3	22	21	26	2007.TB298. ITS5/LR3 (1107 bp)	*Brosimum utile*	SRR[W]	*Phomopsis* sp. (KJ412330.1)*	99%	*Diaporthe* (= *Phomopsis*)	Diaporthaceae (Diaporthales)	N
17	23	22	28	2007.TB49. ITS5/ITS4 (522 bp)	*Nectandra cuspidata*	SRR[W]	Dothideomycetes sp. (JQ905752.1)*	97%	unknown	Mycosphaerellaceae (Capnodiales)	B
18	24	24	31	2010.151.ITS1F/LR3 (1044 bp)	*Anacardium excelsum*	PNM[D]	Sordariomycetes sp. (JQ761140.1)	100%	unknown	unknown	J
19	25	26	33	2010.156a.ITS1F (362 bp)	*Cojoba rufescens*	GS	Uncultured fungus (JX174905.1)	99%	*Albonectria*	Nectriaceae (Hypocreales)	AF
6	26	29	37	2010.52.2.1. ITS1F/LR3 (1036 bp)	*Castilla elastica*	PNM[D]	*Fusarium equiseti* (KC311517.1)*	99%	*Fusarium*	Nectriaceae (Hypocreales)	W

Table 3.4. (continued)

90	95	97	99	Isolate ID. Primer(s) (Seq Length)	Host tree species	Site	Top BLAST match (accession no.)	Max ident.	Genus (= anamorph)	Family (Order)	Phylo. Tree
20	27	30	38	2010.73bL2 .ITS1F/LR3 (1096 bp)	*Virola surinamensis*	PNM[D]	*Aspergillus puniceus* NRRL (EF652469.1)	100%	*Emericella* (= *Aspergillus*)	Trichocomaceae (Eurotiales)	C
1	28	32	40	2011.10.ITS5/ITS4 (767 bp)	*Anacardium excelsum*	CC[M]	Uncultured *Fusarium* (FJ179466.1)*	93%	*Leuconectria* (= *Gliocephalotrichum*)	Nectriaceae (Hypocreales)	AB
8	29	34	44	2012.117bS.ITS5/LR3 (938 bp)	*Dalbergia retusa*	PNM[D]	Fungal endophyte (EU686852.1)	99%	*Glomerella* (= *Colletotrichum*)	Glomerellaceae (Glomerellales)	S

Includes consensus sequences and seven forward, unidirectional reads; Of the 10 singleton species, one was classified as such based on a unidirectional read.
*indicate sequences from unpublished studies
Sites are coded as follows: BCI = Isla Barro Colorado, BV = Buena Vista, CC = Sendero Camino de Cruces, FC = FC = Gunn Hill in Ciudad de Saber (old Fort Clayton), GS = Gamboa Shadehouse, PNM = Parque Natural Metropolitano, SC = Sendero Charco, and SRR = Santa Rita Ridge.

Table 3.5. Taxononomic affiliations of the 68 (of 93) fungal isolates that could be classified to the genus-level based on phylogenetic analyses. All of the isolates belong to the phylum (division) Ascomycota and its largest subphylum, Pezizomycotina. While the fungi were isolated from symtomatic tissue and most are likely to be phytopathogens, those in the class Eurotiomycetes (listed in gray) are unlikely to be pathogenic to plants.

Class	Order	Family	Genus (= anamorph)	No. of isolates
Dothideomycetes	Capnodiales	Mycosphaerellaceae	*Mycosphaerella*	1
Eurotiomycetes	Eurotiales	Trichocomaceae	*Emericella* (= *Aspergillus*)	1
			Talaromyces (= *Penicillium*)	2
Sordariomycetes	Diaporthales	Diaporthaceae	*Diaporthe* (= *Phomopsis*)	7
	Glomerellales	Glomerellaceae‡	*Glomerella* (= *Colletotrichum*)	8
	Hypocreales	Bionectriaceae	*Bionectria* (= *Clonostachys*)	7
		Hypocreaceae	*Hypocrea* (= *Trichoderma*)	2
		Nectriaceae	*Albonectria*	1
			Calonectria	5
			Gibberella (= *Fusarium*)	1
			Glionectria (= *Gliocladiopsis*)	4
			Leuconectria (= *Gliocephalotrichum*)	1
			Nectria (= *Fusarium*)	3
			Nectricladiella (= *Cylindrocladiella*)	4
			Neonectria (= *Cylindrocarpon*)	4
	Magnaporthales	Magnaporthaceae‡	*Mycoleptodiscus*	13
	Xylariales	Amphisphaeriaceae	*Pestalotiopsis*	4

‡Uncertain placement in the Sordariomycetes (Kirk et al. 2001).

Table 3.6. Richness and diversity of phytopathogens considering all forests and all seedlings (90 fungal isolates from 75 symptomatic seedlings, representing 21 tree species and 11 families, collected from seven lowland, tropical forests in Panama). For 90, 95, 97, and 99% ITS sequence similarity, the observed richness of operational taxonomic units (OTUs) (S_{obs}), the number of singletons and their contribution to the observed richness, abundance-based estimates of the extrapolated OTU richness [Chao1 (asymmetric, log-linear 95% confidence intervals), Abundance-based Coverage Estimator (ACE)], and diversity [Fisher's α (standard deviation)]. The bias-corrected formula for Chao1 was used when Chao's estimated coefficient of variation (CV) was less than 0.5. For datasets with a CV > 0.5, the classic formula for Chao1 was used because the bias-corrected formula becomes imprecise when CV > 0.5 (Colwell 2013).

Seq. similarity	S_{obs}	No of. singletons (% of S_{obs})	Chao1 (CI)	ACE	Fisher's α (SD)
90%	19	6 (32%)	22.56[C] (19.64, 38.75)	24.92	7.35 (1.25)
95%	28	10 (36%)	32.45[BC] (29.01, 47.43)	37.32	13.93 (2.32)
97%	33	18 (55%)	59.7[C] (41.3, 118.85)	62.56	18.79 (3.16)
99%	43	27 (63%)	103.07[C] (64.04, 214.44)	94.29	32.29 (5.70)

[C] indicates that the classic formula was used.
[BC] indicates that the bias-corrected formula was used.

Table 3.7. Richness and diversity of phytopathogens in the drier (43 isolates from 36 seedlings, representing 13 tree species) versus wetter (35 isolates from 28 seedlings, representing 15 tree species) forests. For 90, 95, 97, and 99% ITS sequence similarity, the observed richness (S_{obs}), the number of singletons and their contribution to the observed richness, abundance-based estimates of the extrapolated species richness [Chao1 (asymmetric, log-linear 95% confidence intervals), Abundance-based Coverage Estimator (ACE)] and diversity [Fisher's α (standard deviation)]. The bias-corrected formula for Chao1 was used when Chao's estimated coefficient of variation (CV) was less than 0.5. For datasets with a CV > 0.5, the classic formula for Chao1 was used because the bias-corrected formula becomes imprecise when CV > 0.5 (Colwell 2013).

	Seq. similarity	S_{obs}	No. of singletons (% of S_{obs})	Chao1 (CI)	ACE	Fisher's α (SD)
Drier	90%	11	3 (27%)	13.93[BC] (11.34, 35.69)	12.73	4.77 (1.15)
	95%	16	5 (31%)	17.62 [BC] (16.22, 27.75)	19.59	9.23 (2.25)
	97%	20	12 (60%)	43.44 [C] (25.58, 118.45)	40.75	14.53 (3.68)
	99%	25	17 (68%)	72.04 [C] (37.38, 203.70)	59.57	24.95 (6.87)
Wetter	90%	16	8 (50%)	22.21[C] (17.32, 45.13)	28.8	11.39 (3.19)
	95%	20	12 (60%)	32.82[BC] (23.33, 69.25)	38.32	19.39 (5.87)
	97%	21	13 (62%)	33.62[BC] (24.44, 67.30)	42.92	22.16 (6.88)
	99%	24	18 (75%)	63.34[C] (35.32, 160.72)	80.05	33.68 (11.51)

[BC] indicates that the bias-corrected formula was used.
[C] indicates that the classic formula was used.

Table 3.8. Richness and diversity of phytopathogens in the drier (38 isolates from 32 seedlings) versus wetter (26 isolates from 21 seedlings) forests, considering only isolates (N = 64) from the nine tree species for which at least one seedling of each species was collected in both the wetter and drier forests. For 90, 95, 97, and 99% ITS sequence similarity, the observed richness of operational taxonomic units (OTUs) (Sobs), the number of singletons and their contribution to the observed richness, abundance-based estimates of the extrapolated OTU richness [Chao1 (asymmetric, log-linear 95% confidence intervals), Abundance-based Coverage Estimator (ACE)], and diversity [Fisher's α (standard deviation)]. The bias-corrected formula for Chao1 was used when Chao's estimated coefficient of variation (CV) was less than 0.5. For datasets with a CV > 0.5, the classic formula for Chao1 was used because the bias-corrected formula becomes imprecise when CV > 0.5 (Colwell 2013).

	Seq. similarity	S_{obs}	Singletons (% of S_{obs})	Chao1 (CI)	ACE	Fisher's α (SD)
Drier	90%	11	4 (36%)	16.84[BC] (11.92, 47.98)	13.76	5.19 (1.33)
	95%	16	7 (44%)	20.08[BC] (16.76, 37.82)	23	10.41 (2.74)
	97%	19	13 (68%)	101.27[C] (32.62, 515.73)	42.27	15.12 (4.16)
	99%	24	18 (75%)	181.73[C] (51.28, 935.86)	69.07	27.97 (8.61)
Wetter	90%	15	10 (67%)	31.02[C] (18.57, 86.79)	37.37	14.78 (5.21)
	95%	18	13 (72%)	36.75[BC] (23, 88.24)	44.56	25.89 (10.35)
	97%	19	15 (79%)	73.08[C] (31.16, 259.48)	63.34	31.79 (13.44)
	99%	20	17 (85%)	158.94[C] (43.87, 828.7)	95.97	39.71 (17.92)

[BC] indicates that the bias-corrected formula was used.
[C] indicates that the classic formula was used.

Table 3.9. Based on 90, 95, 97, and 99% ITS sequence similarity, shared taxa statistics for the phytopathogen communities of drier versus wetter forests. The analyses include 78 fungal isolates from 64 symptomatic seedlings, representing 17 tree species and nine families, collected from three forests in Panama (Table 3.3). The columns detail the observed shared species (Shared S_{obs}), Chao's estimated number of shared species (Shared S_{est}), the classic Jaccard similarity coefficient, and the Chao-Jaccard abundance-based estimated similarity index which is corrected for unseen species (standard deviation).

Sequence similarity	Shared S_{obs}	Shared S_{est}	Jaccard similarity	Chao-Jaccard similarity (SD)
90%	8	12.74	0.42	0.7 (0.14)
95%	9	14.8	0.33	0.55 (0.13)
97%	9	15.21	0.28	0.52 (0.15)
99%	10	31.02	0.26	0.53 (0.18)

Table 3.10. Based on 90, 95, 97, and 99% ITS sequence similarity, shared taxa statistics for the phytopathogen communities of drier versus wetter forests, considering only isolates from the nine tree species for which at least one seedling of each species was collected in both the wetter and drier forests (64 fungal isolates from 53 symptomatic seedlings). The columns detail the observed shared species (Shared Sobs), Chao's estimated number of shared species (Shared Sest), the classic Jaccard similarity coefficient, and the Chao-Jaccard abundance-based estimated similarity index which is corrected for unseen species (standard deviation).

Sequence similarity	Shared S_{obs}	Shared S_{est}	Jaccard similarity	Chao-Jaccard similarity (SD)
90%	8	14.28	0.44	0.77 (0.17)
95%	9	28.17	0.36	0.76 (0.2)
97%	9	29.36	0.31	0.67 (0.22)
99%	10	72.42	0.29	0.97 (0.18)

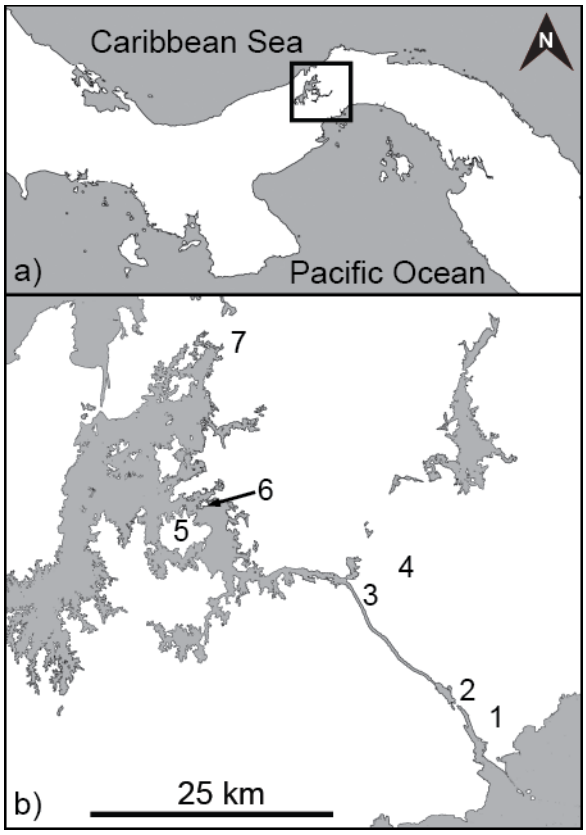
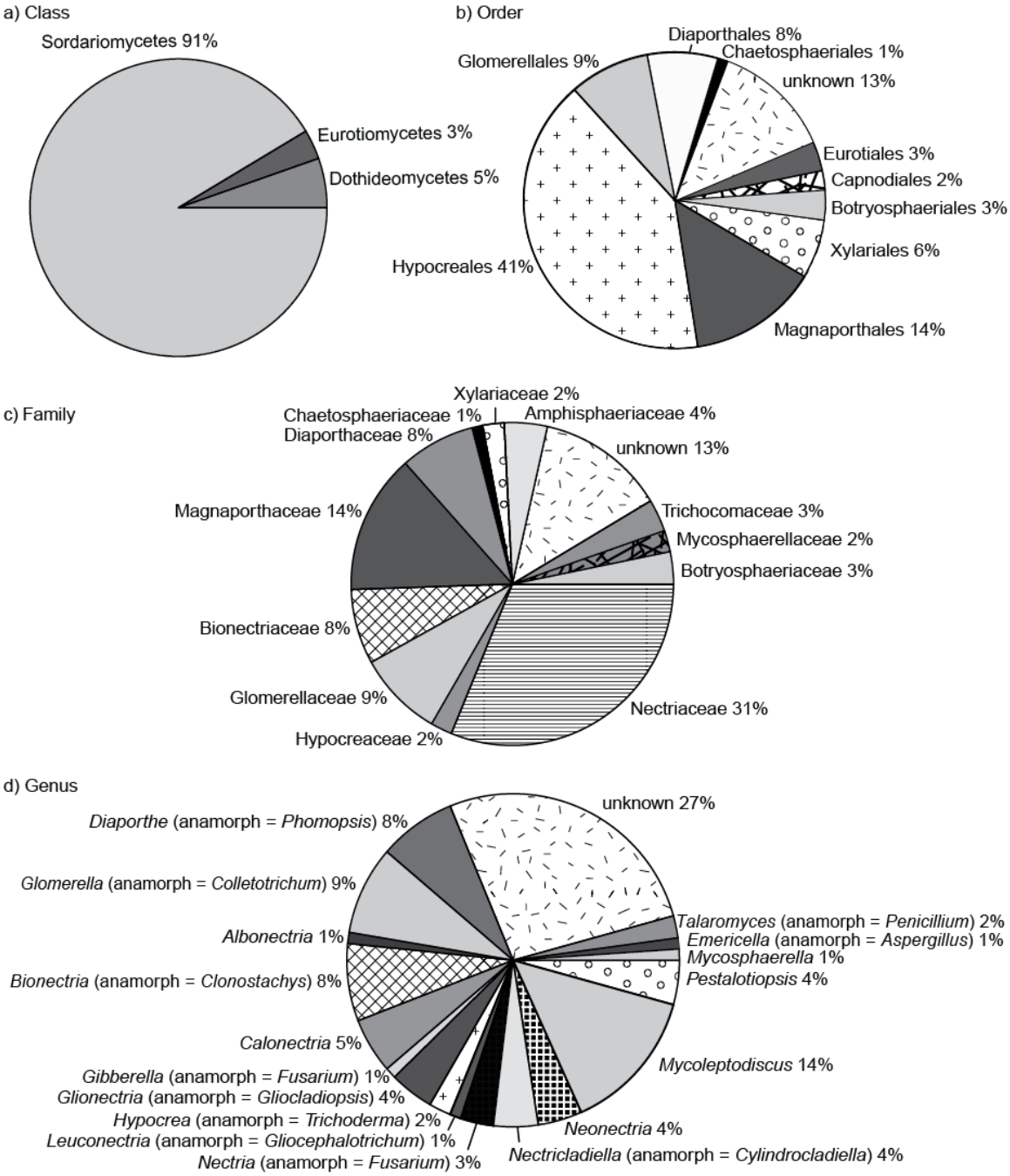


Fig. 3.1. Maps of a) the Republic of Panama and b) the seven forest sites from which symptomatic seedlings were collected (maps modified from Spear et al. 2015). The collection sites span a north to south rainfall gradient and the Isthmus of Panama (see Table 3.1 for site-specific details).

Fig. 3.2. A diagram of the internal transcribed spacer (ITS) region and approximate binding positions for the primers used to amplify the internal transcribed spacer (ITS) region of the fungi isolated from seedlings with pathogen-caused damage. A variety of primer pairs (ITS1F/ITS4, ITS1F/LR3, ITS5/ITS4, ITS5/LR3) were used because the amplifications were conducted in different labs, in different years, and, in some cases, because amplification failed when one of the other pairs was used. See Table 3.3 for additional details about the primers.

Fig. 3.3. Taxonomic summary of fungi isolated from diseased seedlings in Panama, specifying the proportion of all isolates belonging to a specific a) class, b) order, c) family, or d) genus. Thirteen of the 85 isolates classified as Sordariomycetes (panel a) are estimated to be members of the genus *Mycoleptodiscus* (Magnaporthaceae) and eight of the 85 are estimated to be members of the genus *Glomerella* (Table 3.4). Both genera have uncertain placement in the Sordariomycetes (Kirk et al. 2001). An additional two of the 85 isolates classified as Sordariomycetes are not closely related to any reliable, named sequences in GenBank; thus, our assignment was made tentatively (Table 3.4, see tree G in Appendix). For panel d, isolate 2010.52.2.1, which is estimated to be a *Fusarium*, is categorized as unknown because fungi estimated to be closely related represent the genera *Giberella* and *Nectria*.


a) Class
Sordariomycetes 91%
Eurotiomycetes 3%
Dothideomycetes 5%
b) Order
Diaporthales 8%
Chaetosphaeriales 1%
Glomerellales 9%
unknown 13%
Eurotiales 3%
Capnodiales 2%
Botryosphaeriales 3%
Xylariales 6%
Hypocreales 41%
Magnaporthales 14%
c) Family
Xylariaceae 2%
Chaetosphaeriaceae 1%
Amphisphaeriaceae 4%
Diaporthaceae 8%
unknown 13%
Magnaporthaceae 14%
Trichocomaceae 3%
Mycosphaerellaceae 2%
Botryosphaeriaceae 3%
Bionectriaceae 8%
Glomerellaceae 9%
Nectriaceae 31%
Hypocreaceae 2%
d) Genus
unknown 27%
Diaporthe (anamorph = Phomopsis) 8%
Glomerella (anamorph = Colletotrichum) 9%
Talaromyces (anamorph = Penicillium) 2%
Emericella (anamorph = Aspergillus) 1%
Albonectria 1%
Mycosphaerella 1%
Bionectria (anamorph = Clonostachys) 8%
Pestalotiopsis 4%
Calonectria 5%
Mycoleptodiscus 14%
Gibberella (anamorph = Fusarium) 1%
Glionectria (anamorph = Gliocladiopsis) 4%
Hypocrea (anamorph = Trichoderma) 2%
Neonectria 4%
Leuconectria (anamorph = Gliocephalotrichum) 1%
Nectria (anamorph = Fusarium) 3%
Nectricladiella (anamorph = Cylindrocladiella) 4%

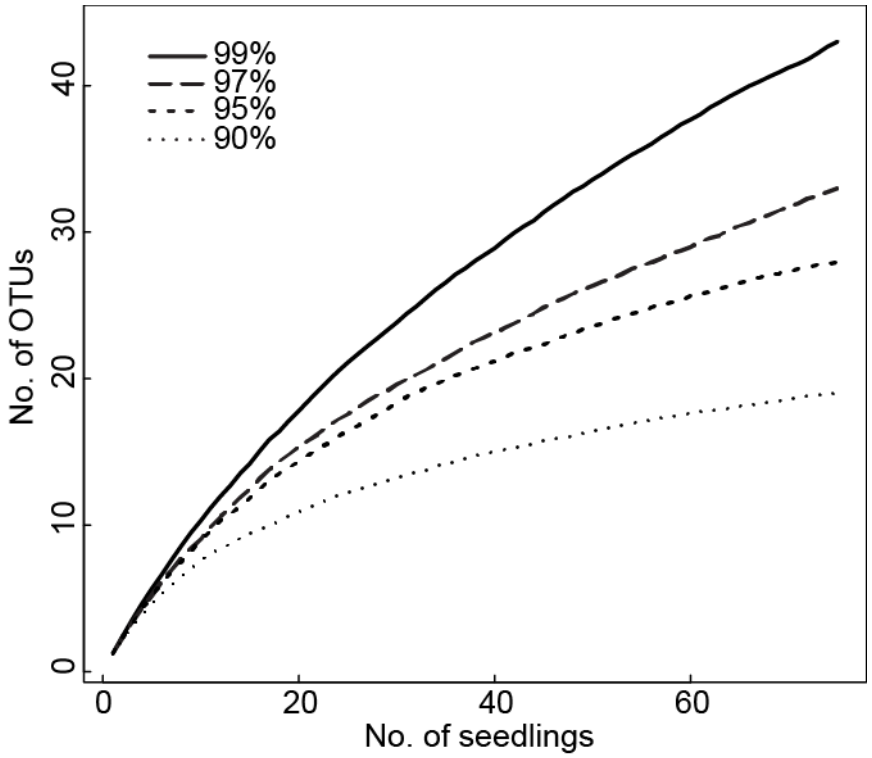


Fig. 3.4. Nonasymptotic accumulation of operational taxonomic units (OTUs) defined by 90-99% ITS sequence similarity for phytopathogens isolated from 75 seedlings with pathogen-caused damage collected from tropical forests in Panama. The curves represent the mean OTU accumulation of 100 randomizations of seedling order derived from the observed richness.

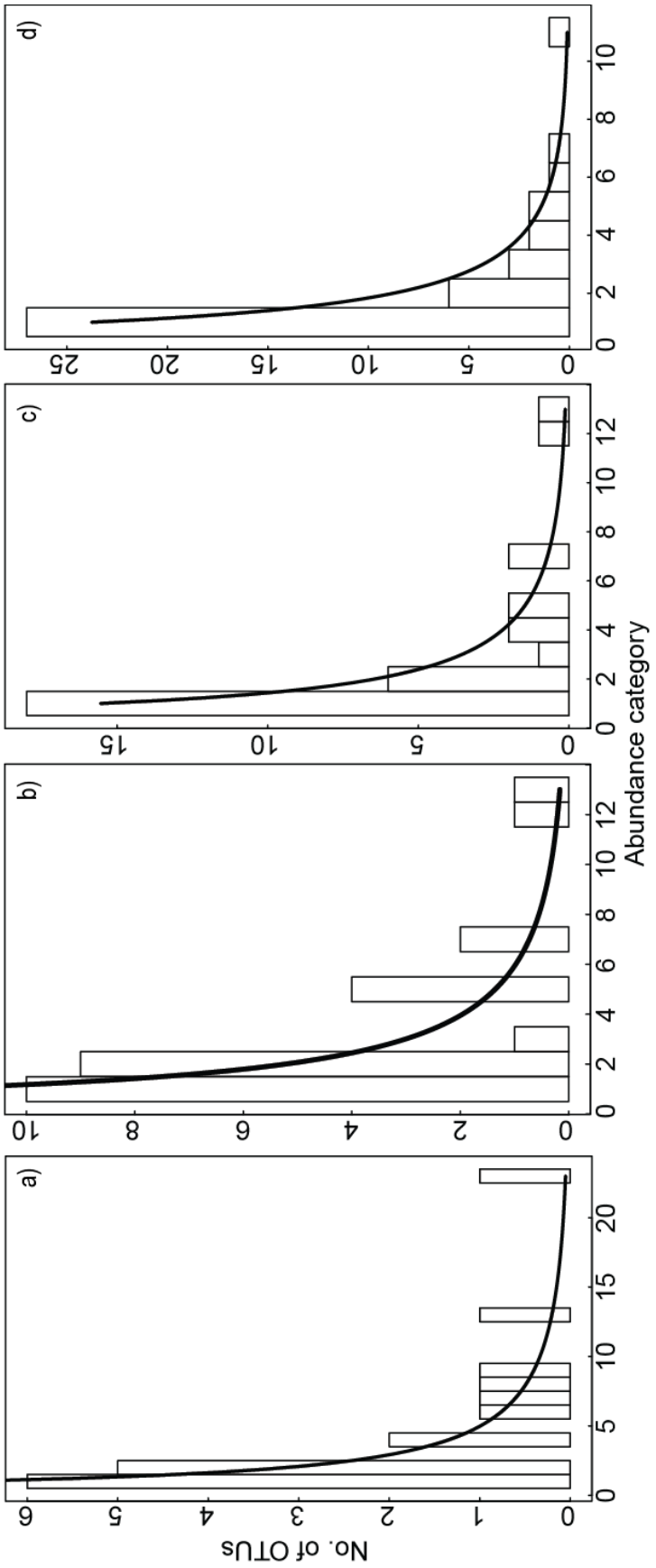


Fig. 3.5. Relative abundance distributions based on Fisher's log-series for operational taxonomic units (OTUs) defined by 90, 95, 97, and 99% ITS sequence similarity (panels a-d, respectively) for phytopathogens isolated from seedlings with pathogen-caused damage collected from tropical forests in Panama (90 isolates from 75 seedlings). Most of the OTUs are rare and relatively few are abundant. The curves represent the expected number of OTUs based on observed abundance (bars).

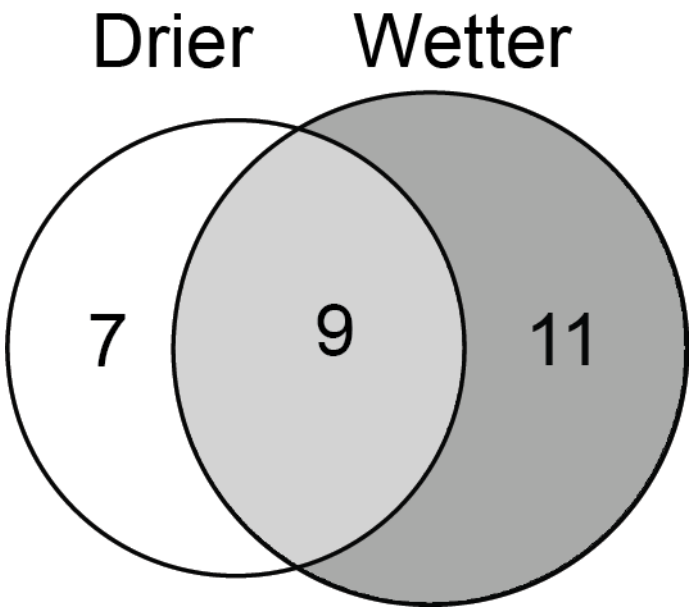


Fig. 3.6. Community similarity (overlap) for phytopathogen species isolated from seedlings with pathogen-caused damage in the drier versus wetter forests. Of the 27 species of phytopathogens observed in the drier and wetter forests, seven species were unique to the drier forests (white circle), 11 species were unique to the wetter forest (dark grey circle), and nine species were observed in both the drier and wetter forests (light grey intersection) (classic Jaccard similarity coefficient = 0.33; Chao-Jaccard abundance-based similarity index = 0.55; see Table 3.9 for shared taxa statistics based on 90-99% ITS sequence similarity).

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CHAPTER 4

HOST GENERALISM EXHIBITED BY FUNGI THAT ARE PATHOGENIC TO SEEDLINGS IN THE TROPICAL FORESTS OF PANAMA

Abstract

For the past 40 years, host-specialized pests of plants, including phytopathogens, have been credited with contributing to the maintenance of tropical forest diversity under the Janzen-Connell hypothesis. Yet, the relative rarity of tree species in diverse tropical forests and the passive dispersal of phytopathogens should favor phytopathogens with relatively wide host ranges. We surveyed the host associations of potential phytopathogens and used shadehouse-based inoculations to experimentally assess (i) the pathogenicity of fungi isolated from symptomatic seedlings, (ii) the host ranges of the pathogenic isolates, and (iii) differences among tree species in vulnerability. We identified 11 pathogenic isolates belonging to the genera *Mycoleptodiscus*, *Bionectria*, *Calonectria*, and *Pestalotiopsis*. The majority of the pathogenic isolates were multihost and we observed no phylogenetic signal to their host range. The tree species tested were differentially susceptible to disease, with some species seemingly resistant to all fungal isolates tested and other species susceptible to multiple isolates. Furthermore, the

outcome of infection differed among tree species susceptible to a given pathogenic, multihost isolate, ranging from tissue damage to death. Our results add to the growing body of evidence that plant-associated fungi in the tropics are able to infect a wide range of species. However, we also show that tree species are differentially vulnerable to these generalist pathogens, which suggests that generalist pathogens can contribute to the maintenance of local forest diversity via host-specific impacts rather than the host specificity originally envisioned under the Janzen-Connell hypothesis. Additionally, generalist pathogens may contribute to the spatial turnover of plant species on a regional scale by excluding disease-sensitive tree species from disease-prone habitats.

Introduction

The regulation of the relative abundances of plant species by host-specific natural enemies is a commonly cited mechanism for the maintenance of local plant diversity. Under the Janzen-Connell hypothesis, conspecific seeds and seedlings at high densities near conspecific adult trees suffer disproportionately higher mortality relative to heterospecific seeds and seedlings due to an accumulation of host-specialized enemies, such as insects and phytopathogens (Janzen 1970, Connell 1971; see also Gillett 1962). Thus, host-specific natural enemies facilitate the establishment of heterospecific seedlings in areas from which conspecifics are excluded and prevent any single species from becoming competitively dominant.

Much of the support for the Janzen-Connell hypothesis has been based on spatial and temporal patterns of conspecific negative density-dependent mortality (Harms et al. 2000, Ahumada et al. 2004, Comita et al. 2010), but pattern-based studies cannot identify

the mechanism(s) driving the observed patterns (Comita et al. 2014). Experimental studies suggest that phytopathogens may contribute to the observed Janzen-Connell effects (Augspurger and Kelly 1984, Bell et al. 2006, Mangan et al. 2010, Bagchi et al. 2014). However, the phytopathogens are rarely identified and most studies fail to experimentally address a crucial assumption of the Janzen-Connell hypothesis, that the natural enemies exhibit high host specificity with regard to locally available hosts (but see Packer and Clay 2000, Liu et al. 2012).

While it has been widely assumed that the phytopathogens generating the observed Janzen-Connell effects are host specific, the relative rarity of tree species in diverse tropical forests and the passive dispersal of plant-associated fungi may actually select for phytopathogens with intermediate to broad host ranges (May 1991, Coley and Barone 1996). Indeed, mounting evidence from tropical and temperate forests suggests that multihost phytopathogens are prevalent (Augspurger and Wilkinson 2007, Gallery et al. 2007, Gilbert and Webb 2007, Kluger et al. 2008, Hersh et al. 2012). Nonetheless, multihost phytopathogens could influence plant community composition if host species vary in their vulnerability to infection by a shared phytopathogen. Specifically, multihost phytopathogens can promote coexistence and local diversity if the host plant species are competing for resources and the more vulnerable plant species is also the superior competitor (Mordecai 2011). These seemingly conflicting ideas about if and how phytopathogens contribute to the maintenance of plant community diversity highlight a growing interest in the identities and host specificities of phytopathogens in natural systems.

Here we (i) identify some of the fungal pathogens killing tree seedlings in the tropical forests of Panama, (ii) describe their host ranges, and (iii) explore interspecific variability in tree species vulnerability to pathogens. To accomplish this, we first isolated 93 potentially pathogenic fungal isolates from symptomatic seedlings that were collected in Panama. We then inoculated seedlings of 36 native tree species with 34 of the fungal isolates in shadehouse-based experiments. We had three hypotheses. First, in the diverse tropical forests of Panama, phytopathogens will tend to have broad host ranges because a given host species is relatively rare and the phytopathogens are passively dispersed. Pathogens are often classified as specialists or generalists based on the number of different host species they attack and the phylogenetic relatedness among the hosts. Herein, we refer to phytopathogens that are able to cause disease in plant species in multiple families as generalists. Second, under the assumption that closely related plant species will tend to share similar defense traits inherited from a common ancestor, multihost phytopathogens are more likely to attack two closely related tree species than two distantly related tree species. Third, tree species will differ in their vulnerability to phytopathogen attack because the cost-to-benefit ratio of defenses against and tolerance of attack differs among species (Strauss and Agrawal 1999, Endara and Coley 2010). We focus on the seedling stage because seedling survival directly shapes plant communities (Engelbrecht et al. 2007, Comita et al. 2010, Mangan et al. 2010, Baldeck et al. 2014, Green et al. 2014). We investigated fungi that attack seedlings because they are an important cause of seedling mortality in the tropics (Augspurger 1983, Gilbert 2005, Alvarez-Loayza and Terborgh 2011) and because they can promote diversity in seedling communities (Bagchi et al. 2014).

Methods

We conducted inoculation experiments to evaluate: (i) the pathogenicity of 34 fungal isolates isolated from symptomatic seedlings, (ii) the host ranges of the phytopathogens attacking seedlings, and (iii) if and how tree specifies differ in their vulnerability to phytopathogens. In addition to describing the host ranges of the phytopathogens based on experimental inoculations, we used the host associations and isolation frequencies of 93 fungal isolates, isolated from seedlings of 21 tree species collected from forests and a shadehouse in Panama, to describe the host ranges of potential phytopathogens.

Fungal isolates evaluated

Ninety-three potentially pathogenic fungal isolates were isolated from 77 seedlings with pathogen-caused damage (see the Methods in Ch. 3 for additional details). The seedlings represented 21 tree species (Table 4.1) and were collected from seven forests and a shadehouse in Panama. The fungal isolates were assembled into species-level operational taxonomic units (OTUs) according to 95% sequence similarity and, based on Arnold and Lutzoni (2007) and U'Ren et al. (2009), we consider 95% sequence similarity to be a proxy for species and refer to the OTUs as "species" hereafter. The taxonomic placement of each fungal isolate was estimated via phylogenetic analyses based on the internal transcribed spacer (ITS) region of rDNA (see the Methods in Ch. 3). The 93 fungal isolates represent 29 fungal species. All 93 fungal isolates belong to the subphylum Pezizomycotina (phylum Ascomycota) and most belong to the class Sordariomycetes (see the Results in Ch. 3).

We experimentally assessed the pathogenicity and host specificities of a subset of the fungal isolates (34 of the 93, representing 18 fungal species; Table 4.2). Selected to represent a variety of host tree species, the 34 fungal isolates tested were isolated from seedlings of 12 tree species (Tables 4.1 and 4.2). Thirty-two of the 34 fungal isolates tested were isolated from seedlings collected from five forest sites in Panama and two of the isolates tested were isolated from seedlings collected from a shadehouse in Gamboa, Panama (Table 4.2). The majority of the 34 isolates were isolated from seedlings with pathogen-caused stem damage (70.6%), followed by pathogen-caused root (17.6%) and leaf (11.8%) damage.

Experimental assessments of pathogenicity were necessary because, while we isolated the fungi directly from diseased seedlings, fungi isolated from symptomatic plant tissue are not necessarily the causative pathogens and taxonomic affiliations are not indicative of pathogenicity (Delaye et al. 2013).

Tree species evaluated

The symptomatic seedlings from which the 93 potentially pathogenic fungal isolates were isolated represent 21 tree species and 11 families (original hosts; Table 4.1). Via inoculation experiments, 34 of the 93 fungal isolates were tested against seedlings of 36 tree species, representing 20 families spread across the phylogeny (targets; Table 4.1).

Seeds of the 36 tree species tested in the inoculation experiments (described below) were collected in the forests bordering the Panama Canal during the rainy seasons (May-Nov) of 2011 and 2012. Due to time constraints, we preferentially collected tree species without seed dormancy. Seeds were surface-sterilized using sequential washes of

ethanol and bleach (95% ethanol for 10 seconds, 10% commercial bleach for 2 minutes, 70% ethanol for 2 minutes). Surface-sterilized seeds were planted in seedling flats containing autoclave-sterilized commercial soil (autoclaved twice for 1 hour at 121º C) to generate seedlings for use in the inoculation experiments.

Shadehouse conditions

Inoculation experiments were conducted in Smithsonian Tropical Research Institute (STRI) shadehouses in Gamboa, Panama (elev = 36.9 m, 9º7'10"N, 79º42'5"W) from June through December of 2011 and from June through August of 2012. In 2011, two shadehouses were used. The shadehouses were covered with shadecloth and plastic to (i) mimic the conditions of the forest understory and to (ii) exclude rainwater in order to minimize outside contamination and splashing between pots. Similar to the forest understory, the photosynthetically active radiation reaching the seedlings was, on average, 1.5% and 1.8% of full sunlight in the two shadehouses (LI-250 light meter and a one-meter LI-191 line quantum sensor, LI-COR, Lincoln, NE, USA; Table 4.3). Light availability was somewhat variable within one of the shadehouses. To control for this, seedlings were rotated weekly among the tables within a shadehouse. The average air temperatures (26.1ºC and 25.6ºC) within the shadehouses were similar to ambient air temperatures (Table 4.3). The relative humidities (85.1% - 90.5%) within the shadehouses were similar to the relative humidity of the forest understory during the wet season (90% to 95% at midday; Windsor 1990; Table 4.3).

All seedlings were hand-watered every three days. The soil was not allowed to dry out with the intention of mimicking natural wet-season conditions in the forests. To

prevent contamination via splashing between pots when the seedlings were watered, the seedlings were grouped by the isolate with which they were inoculated and inoculum-free (control) seedlings were kept separate from the inoculated seedlings within a shadehouse. A given treatment group included seedlings of multiple tree species and within that group the seedlings were haphazardly arranged on the shadehouse table.

Inoculation experiments

To inoculate the seedlings with the fungal isolates, we used a modified version of the oat kernel inoculation technique, which is commonly used in phytopathology and was employed by Augspurger and Wilkinson (2007). Under sterile conditions in the lab, autoclave-sterilized rice grains (substituted for oat kernels) were inoculated with one of the 34 fungal isolates selected for screening. Inoculated rice grains were incubated at room temperature until the rice grains were visibly colonized by mycelia. The colonized and inoculum-free, autoclave-sterilized rice grains were transferred to the individual seedling pots with flame-sterilized tweezers. This passive inoculation technique simulates infested plant material in the soil.

Recently emerged seedlings were transplanted to individual pots containing autoclave-sterilized commercial soil with rice colonized by one of the 34 fungal isolates or autoclave-sterilized commercial soil with inoculum-free, autoclave-sterilized rice (control/sham treatment). While the process of autoclave-sterilization has the potential to alter soil structure and nutrient availability, this effect was common among all of the treatment groups. In 2011, seedlings (N = 2,688) were planted in Ray Leach "Cone-tainers"™ (volume = 164 cm^3; Stuewe & Sons, Inc., Tangent, OR, USA) and in

germination trays with individual cones (cone volume = 175 cm^3; Totrotrac, Panamá City, Panamá). In 2012, seedlings (N = 1,090) were only planted in "Cone-tainers"™. For all inoculation experiments, the seedlings in a treatment group and its paired control group were planted in the same type of pot.

Since fruiting and germination times differ among species, it was necessary to stagger the initiations of the inoculation experiments. Each host tree species-by-fungal isolate combination had a paired control group with seedlings of the same tree species that were transplanted on the same date, planted in the same type of pot, kept in the same shadehouse and, thus, subjected to the same conditions. Depending on seed availability, four to 20 seedlings per species were included in each treatment (nine to 40 seedlings, in total, for paired inoculated and inoculum-free/control groups). For the initial inoculation experiments, the seedlings (N = 892) were transplanted to individual pots and then the rice grains were added to the soil adjacent to the seedling. In subsequent inoculations, the rice grains were added to the soil in the individual pots and then the seedlings (N = 2,886) were planted with their roots in contact with the inoculum. Since all seeds of a given tree species did not germinate simultaneously, it was necessary to use seedlings at different developmental stages (radicle/no stem, stem/no leaves, leaves) to achieve the minimum number of replicates. Initially in 2011, seedlings (N = 892) were randomly assigned to a treatment without consideration of their developmental stage. However, for the remainder of the experiments in 2011 and all of the experiments in 2012, we ensured that either (i) all seedlings in the inoculated and inoculum-free/control groups were the same developmental stage or that (ii) seedlings of different developmental stages were evenly distributed between the inoculated and control groups to ensure that the treatment effects

were independent of the potential effects of seedling developmental stage. In all cases, we documented the developmental stage of the seedlings at the time of inoculation.

The tree species-by-fungal isolate combinations were not fully reciprocal (332 of the 1,224 possible combinations were made; 3,778 seedlings in total). A given fungal isolate was tested against two to 29 tree species and 79% of the 34 fungal isolates evaluated were tested against seedlings of ≥5 tree species. A given tree species was tested against one to 31 fungal isolates and 67% of the 36 tree species evaluated were tested against ≥5 fungal isolates.

All inoculation experiments were run for at least four weeks and some were run for up to 22 weeks; however, to standardize the time of exposure across treatments, our analyses only include observations made within the first five weeks. Based on our first year of inoculation experiments (2011), the majority of pathogen-caused seedling deaths occurred within the first five weeks following inoculation (Fig. 4.1).

Traditionally, pathogenicity is assessed by inoculating healthy individuals of the species from which the potential pathogen was isolated to generate the symptoms originally observed (partial fulfillment of Koch's postulates; Agrios 2005). Whenever possible, a given fungal isolate was tested against seedlings of the same tree species from which it was originally isolated, hereafter referred to as "conspecific seedlings", as specified by Koch's postulates. However, we were unable to test four of the 34 fungal isolates against conspecific seedlings because seeds of the original host tree species were unavailable or were available in insufficient quantities. Thus, for proof of pathogenicity, those four fungal isolates were tested against seedlings of tree species different from the tree species from which they were originally isolated, hereafter referred to as

"heterospecific seedlings". The alternative tree species against which they were tested included tree species with previously observed disease susceptibility, such as *Luehea seemannii* (Augspurger and Wilkinson 2007) and *Dalbergia retusa* (E. Spear, personal observation). To fulfill Koch's postulates, it is necessary to re-isolate the organism being evaluated for pathogenicity from inoculated individuals exhibiting the symptoms originally observed. However, when disease symptoms appeared, we did not attempt to re-isolate the fungal isolate because a main objective of our study was to determine if and how tree species differ in their vulnerability to disease, including if phytopathogen attack does or does not lead to seedling mortality for different tree species. We did not attempt to re-isolate the fungal isolate being evaluated post-seedling mortality because necrotic tissue is often colonized by saprophytes, making it challenging to re-isolate the specific isolate being tested, and because were limited by time and resources.

Documenting symptoms of disease development

Disease symptoms were documented every three days and were categorized as: (i) seedling mortality; (ii) stem damage, including necrotic lesions on the stem, collapse of the stem at the soil line, and stem dieback from the tip; (iii) wilted tissue; and (iv) stunted seedling growth. A seedling was classified as morbid or being in a diseased state if it had any of the four aforementioned disease symptoms. Wilted tissue was considered a symptom of disease because the soil was not allowed to dry out and phytopathogens attacking the roots can interfere with water uptake via blockage of the xylem vessels (Yadeta and Thomma 2013). Similarly, stunted growth can be indicative of phytopathogen-caused problems with the root system (e.g., rot), xylem obstruction,

and/or stunting may occur because energy is allocated to defenses and/or tissue replacement rather than growth. While we were particularly interested in documenting pathogen-caused seedling mortality because of its clear fitness cost and because seedling mortality directly shapes plant communities, pathogen-caused tissue damage and stunted growth are important consequences because they have the potential to negatively affect a seedling's competitive ability. Furthermore, documenting the occurrence of a variety of symptoms makes it possible to (i) track if a specific symptom of pathogen attack (e.g., wilt) leads to death for certain host tree species-by-phytopathogen combinations and to (ii) describe if and how tree species differ in their impact from infection by a shared pathogen.

While we noted both foliar and root damage, we did not analyze those data. Root damage could only be observed if seedlings survived to the end of the experiment and were harvested. The foliar damage (e.g., spots of necrotic tissue and defoliation) data were not included in our analyses because we were inoculating the soil and because similar foliar damage appeared for seedlings inoculated with different isolates and seedlings in the control groups at roughly the same time, suggesting that the biotic agent causing the foliar damage may have blown into the screened-in shadehouses.

Statistical analyses

Bias-reduced generalized linear models ('brglm' function; Kosmidis 2013), assuming binomial error distributions and probit link functions, were used to compare the proportion of seedlings of a given tree species that experienced a specific disease symptom or general morbidity when inoculated with one of the 34 fungal isolates versus

seedlings of the same species subjected to the sham (control) treatment. The response variables were incidence (presence/absence) of (i) seedling mortality, (ii) stem damage, (iii) wilted tissue, (iv) stunted growth, and (v) general morbidity. As mentioned above, for the initial experiments conducted in 2011, seedlings (N = 892) were randomly assigned to a treatment without consideration of their developmental stage. Thus, to verify that treatment effects were independent of seedling developmental stage for those inoculation experiments, our initial models included treatment (inoculated vs. paired, inoculum-free control) and seedling developmental stage (radicle/no stem, stem/no leaves, leaves) as predictor variables for each tree species-by-fungal isolate combination. As seedling developmental stage was not a significant predictor of disease development in any of the experiments, only treatment was included as a predictor variable in all final models. Generalized linear models with the Firth bias-correction were used because we observed quasi-complete separation of variables for some fungal isolate (inoculated)-by-control (inoculum-free) comparisons and because we had small datasets. Each tree species-by-fungal isolate combination was considered individually for each response variable. The likelihood of disease development was considered significantly different at $P < 0.10$ because the threshold for treatment effects that could be detected was limited by the study's short duration and small sample sizes. A fungal isolate was considered pathogenic if it generated significant disease symptoms in at least one tree species, which was not necessarily the original host tree species.

We explored the correlations between the frequency with which a given fungal species was isolated and (i) the number of tree species from which it was isolated and (ii) the number of tree species it successfully attacked using the Spearman's rank method. For

both analyses, the alternative hypothesis of interest was that the two values under consideration were positively associated.

All analyses were performed in R v. 3.1.1 (R Core Team 2014).

Evaluating the presence of a phylogenetic signal to host range

To determine if there is a phylogenetic signal to the host ranges of multihost pathogens, we analyzed the probability of successful pathogen attack as a function of the pairwise phylogenetic distances between the tree species from which a fungal isolate was isolated (original host) and the tree species it was tested against (target) during the inoculation experiments. We defined successful attack as instances in which a significantly greater proportion of the seedlings inoculated with a given fungal isolate suffered any one of the five pathogen symptoms (described above) than the seedlings in their paired control group. Modeled after Gilbert and Webb (2007), we fit a logistic regression model that included every original host tree species-by-target tree species combination ('glm' function in R, assuming a binomial error distribution). The response variable was successful pathogen attack (1), unsuccessful pathogen attack (0), or comparison not made (NA) and the continuous predictor variable was the log-transformed phylogenetic distance (estimated time of independent evolution) between the two tree species plus one, including inoculations of conspecific seedlings.

To estimate phylogenetic distances between tree species, we created a hypothesis for the phylogenetic relationships among our tree species based on a recently developed angiosperm supertree (R2G220140601 tree and ages file; G. S. Gilbert, unpublished manuscript) using the desktop version of Phylocom (Webb et al. 2008).

Results

Identities of phytopathogenic fungi

Based on general morbidity (i.e., the presence of any of the four specific disease symptoms), pathogenicity was observed for 19 of the 332 tree species-by-fungal isolate combinations (Fig. 4.2, Table 4.4). For 13 of these combinations, infection led to seedling mortality (Fig. 4.3, Table 4.4). When specific symptoms of pathogen attack were each considered in isolation, three additional tree species-by-fungal isolate combinations that were not significant for morbidity indicate pathogenicity (e.g., both the control *Castilla elastica* seedlings and those inoculated with isolate 2010.52.2.1 had stem damage; however, the inoculated seedlings were significantly more likely to suffer from wilted tissue; Figs. 4.4-4.6, Table 4.4).

It should be noted that for two of the 332 tree species-by-fungal isolate combinations, the inoculated seedlings experienced significantly fewer symptoms of pathogen attack than the seedlings in their paired control group (Figs. 4.2 and 4.3, Table 4.4). One of those two combinations included a fungal isolate that was pathogenic to three of the other tree species against which it was tested.

In total, 11 of the 34 isolates tested were pathogenic to at least one of the tree species tested (Table 4.4). For 37% of the 30 combinations in which the fungal isolate was tested against conspecific seedlings, a greater proportion of the inoculated seedlings suffered morbidity than the inoculum-free, control seedlings but not significantly so (Fig. 4.2). For one of the 30 combinations, a significantly greater proportion of the seedlings inoculated with the fungal isolate 2010.52.2.1 suffered wilted tissue, a symptom of disease, than the inoculum-free, control seedlings (in accordance with Koch's postulates;

Fig. 4.5, Table 4.4). Thus, for 10 of the 11 fungal isolates that we classified as phytopathogens, that classification was based on their ability to generate disease in heterospecific seedlings.

For seven of the 11 pathogenic isolates, infection resulted in seedling mortality for at least one of the tree species against which it was tested (Fig. 4.3, Table 4.4). Six of the pathogenic isolates caused stem damage (Fig. 4.4), four caused wilted tissue (Fig. 4.5), and three caused stunted growth (Fig. 4.6, Table 4.4). Multiple disease symptoms were often observed for a single tree species-by-phytopathogen combination (e.g., *Annona glabra* seedlings suffered stem damage, wilt, and mortality when inoculated with isolate 2010.160T; Table 4.4).

The 11 pathogenic isolates were isolated from eight tree species, representing eight families (Tables 4.1 and 4.4). Based on 95% sequence similarity for the internal transcribed spacer (ITS) region, the 11 pathogenic isolates are members of six species of fungi (Tables 4.2 and 4.4; see the Methods in Ch. 3 for additional details about species assignments). The majority of the 11 pathogenic isolates are in the order Hypocreales (64%) and in the families Nectriaceae (36%) and Bionectriaceae (27%) based on phylogenetic analyses to estimate taxonomic placement (see the Methods and Results in Ch. 3 for additional details). The genera represented by the isolates with confirmed pathogenicity include *Mycoleptodiscus*, *Bionectria*, *Calonectria*, and *Pestalotiopsis*. Interestingly, based on ITS region sequence similarity, some of the pathogenic isolates are closely related to fungi isolated from apparently healthy leaves of tropical grasses in Panama (Higgins et al. 2011; see the Results in Ch. 3).

Fungal phytopathogens attacking seedlings are multihost and have wide host ranges

The 11 isolates with confirmed pathogenicity generated significant disease symptoms in 5-31% of the tree species against which they were tested (median = 8%). Furthermore, based on general morbidity, those 11 pathogenic isolates generated some evidence of pathogen attack (i.e., proportion of inoculated seedlings with symptoms - proportion of control seedlings with symptoms > 0, but nonsignificant) in 25-58% of the tree species against which they were tested (median = 43%). Five of the 11 pathogenic isolates successfully attacked more than one tree species. When considering the pathogenic isolates according to their species membership, four of the six pathogenic species caused significant disease symptoms for seedlings of four to five tree species, belonging to three to five families (Table 4.4).

Considering the host associations of all 93 fungal isolates isolated from symptomatic seedlings of 21 tree species, a given fungal species was isolated from one to seven tree species (Fig. 4.7). Similarly, multiple fungal species were isolated from a given tree species (Fig. 4.7). At the high end, the 20 fungal isolates from symptomatic seedlings of *Dalbergia retusa* represent 10 of the 29 fungal species that were observed. The number of host tree species from which a given fungal species was isolated increased with the frequency with which the fungal species was isolated (Spearman's rank correlation rho = 0.925, $P < 0.001$; Fig. 4.8 black circles). Seventeen of the 18 nonsingleton fungal species were isolated from more than one host tree species (Fig. 4.7). Finally, there was a weak, positive association between the number of tree species that a pathogenic species was capable of successfully attacking and the frequency with which

that fungal species was isolated (one-tailed Spearman's rank correlation rho = 0.424, P = 0.201; Figs. 4.7 and 4.8 gray circles).

There is no phylogenetic signal to the host ranges of multihost phytopathogens attacking seedlings

Based on all 332 tree species-by-fungal isolate combinations experimentally tested, we detected no phylogenetic signal to the host ranges of the 11 pathogenic isolates. In other words, phylogenetic relatedness of host trees did not influence the odds of successful pathogen attack [logit(successful/unsuccessful) = -3.35 + 0.141 x log(phylogenetic distance +1); GLM: Est = 0.141, SE = 0.182, Z = 0.773, P = 0.439].

Tree species differ in their vulnerability to pathogens

Twelve of the 36 tree species tested were susceptible to at least one of the 34 fungal isolates tested and six were susceptible to two to four of the isolates (Table 4.4). The 12 susceptible tree species suffered significant pathogen symptoms from five to 67% of the fungal isolates against which they were tested and some evidence of pathogen attack from 33 to 100% of the isolates against which they were tested. None of the tree species was susceptible to all of the fungal isolates against which they were tested (Fig. 4.2). While seedlings of *Castilla elastica* and *Dalbergia retusa* were more vulnerable to pathogen attack than the majority of tree species tested, *C. elastica* and *D. retusa* seedlings only exhibited evidence of morbidity from 36% of the 25 and 39% of the 23 isolates with which they were inoculated, respectively (Fig. 4.2). Most of the tree species tested were seemingly resistant to or tolerant of all fungal isolates tested (Fig. 4.2). Eight

of the tree species tested did not exhibit evidence of morbidity for any of the isolates against which they were tested.

In addition to differences in vulnerability to attack among the 36 tree species tested, the 12 susceptible tree species differed in their tolerance of attack. *D. retusa* seedlings were susceptible to three pathogenic isolates, representing two fungal species, and, for all three pathogenic isolates, infection led to seedling death (Fig. 4.3, Table 4.4). However, for most of the other susceptible tree species, pathogen attack did not consistently lead to death. For those tree species, the impact from infection differed depending on the pathogenic isolate with which it was inoculated. For example, infection led to death for only two of the four isolates that were pathogenic to *C. elastica* seedlings (Fig. 4.3, Table 4.4). Similarly, *Genipa americana* seedlings inoculated with *Pestalotiopsis* sp. (isolate 2010.172) suffered significantly more mortality than the seedlings in their paired control group (Fig. 4.3, Table 4.4), while *G. americana* seedlings inoculated with *Mycoleptodiscus* sp. (isolate 2010.160T) suffered significantly more stem damage, but not mortality, than the seedlings in their paired control group, during the five-week study period (Figs. 4.3 and 4.4, Table 4.4).

Finally, for those tree species that were susceptible to the same multihost pathogen, the impact from attack differed among species. For example, attack by *Pestalotiopsis* sp. (isolate 2010.172) resulted in mortality for seedlings of *Brosimum utile* and *G. americana*, in stem damage for seedlings of *Cochlospermum vitifolium*, and in stunted growth for seedlings of *C. elastica* (Table 4.4).

Discussion

Identities of phytopathogenic fungi

One-third of the 34 fungal isolates tested were pathogenic to seedlings of at least one tree species during the inoculation experiments. Sixty-four percent of the pathogenic isolates (seven of 11) caused lethal infections in at least one tree species. This reinforces the importance of phytopathogens as a source of seedling mortality, their role as a selective pressure, and their potential to directly shape plant community composition.

The pathogenic isolates belong to genera with species known to be phytopathogens (Kirk et al. 2001) and there is overlap with the genera of fungi believed to be pathogens of seedlings in a temperate forest (specifically, *Bionectria*; Hersh et al. 2012) and in a topical forest in Panama (*Pestalotiopsis;* Davidson 2000), foliar pathogens in Panama (*Pestalotiopsis* and *Calonectria;* Gilbert and Webb 2007, Schweizer et al. 2013), and seed-infecting fungi in Panama *(Bionectria* and *Pestalotiopsis*; Kluger et al. 2008). The pathogenic isolates are also closely related to fungi isolated during a Panama-based study of endophytes in tropical grasses (Higgins et al. 2011). It is not surprising that the pathogenic isolates in our study are closely related to fungi isolated from apparently healthy plant tissue because many fungi can colonize the living tissue of plants asymptomatically and then become antagonistic pathogens under different environmental conditions, when a host's health is compromised, or in a different host species (Delaye et al. 2013, Stergiopoulos and Gordon 2014).

We classified 23 of the 34 fungal isolates as nonpathogenic based on the inoculation experiments. In some cases, a fungal isolate may have failed to generate significant symptoms of disease during the inoculation experiments because it did not

cause the disease symptoms exhibited by the seedling from which it was originally isolated. Necrotic tissue is often colonized by saprophytes and, while we isolated from the advancing margin of disease, some of the fungi that we isolated in culture may be secondary invaders rather than the causative pathogen. However, it is likely that we misclassified some pathogenic isolates as nonpathogenic and that a greater proportion of the 34 fungal isolates, for which pathogenicity was experimentally assessed, are, in fact, pathogenic.

First, we may have misclassified some pathogenic isolates as nonpathogenic because we may not have observed all infections. Some of the tree species-by-fungal isolate combinations may have resulted in latent infections, lacking visible symptoms.

Second, our experimental set-up was intended to mimic the conditions of the forest understory and for one crucial variable, light availability, we closely replicated natural levels (ca. 1.5% of full sun; Table 4.3; Brenes-Arguedas et al. 2011). Nevertheless, the tree species-by-fungal isolate combinations were tested under artificial conditions and it is possible that we failed to replicate the conditions that facilitate disease development in the natural environment. Disease development is not only contingent on the presence of a susceptible host species and a competent pathogen, but also on environmental conditions that compromise the host's defenses and favor the pathogen's growth and colonization of plant tissue (Barrett et al. 2009, Hersh et al. 2012). Future assessments of pathogenicity could incorporate conditions that reduce host vigor (e.g., short-term drought events, defoliation, very low light, or nutrient limitations; Desprez-Loustau et al. 2006) to assist or expedite disease development.

Third, the fungal isolates classified as nonpathogenic may not have been solely responsible for the disease symptoms exhibited by the seedling from which it was originally isolated. Infections by multiple organisms are common (Barrett et al. 2009, Hersh et al. 2012, E. Spear, personal observation) and are particularly likely if the phytopathogens attacking seedlings have broad host ranges. Thus, we may not have observed evidence of pathogenicity for isolates capable of causing disease via the synergistic effect of co-infection because we considered each fungal isolate singly and apart from the context of its natural microbial community. In a study of temperate seedling pathogens, two of the five tree species studied were only negatively impacted when infected by more than one fungus (Hersh et al. 2012). Furthermore, unequal effects on seedling survival among plant species have been observed for different combinations of co-infection, suggesting an additional mechanism by which generalist phytopathogens may influence plant community diversity (Hersh et al. 2012, Benítez et al. 2013). Thus, an important next step is to experimentally evaluate disease incidence and severity when a given host species is infected by different combinations of potentially pathogenic fungal isolates.

As mentioned in the Methods, pathogenicity is traditionally determined by inoculating healthy individuals of the original host species to generate the symptoms originally observed and then re-isolating the putative phytopathogen (Koch's postulates; Agrios 2005). While we were able to test 30 of the 34 isolates against seedlings of the original host species, only one of those isolates (2010.52.2.1) generated disease symptoms in conspecific seedlings (*Castilla elastica*) in accordance with Koch's postulates. Failure to generate disease symptoms when conspecific seedlings were

inoculated may have resulted from any of the reasons listed above. However, some of the fungal isolates successfully attacked at least one of the tree species tested (proof of pathogenicity) even though they failed to generate disease symptoms in seedlings of the original host species, which suggests that our shadehouse-based experiments may not have replicated the abiotic or biotic conditions that originally led to the development of disease for the original tree species-phytopathogen combination.

Fungal phytopathogens attacking seedlings are multihost and have wide host ranges

Together, the host associations of the 93 fungal isolates isolated from symptomatic seedlings and the results of the inoculation experiments provide strong support for our hypothesis that the phytopathogens attacking seedlings in tropical forests tend to be generalists. All but one of the nonsingleton fungal species were isolated from more than one host tree species (Fig. 4.7) and, in general, a given fungal species was isolated from heterofamilial tree species (except fungal species nine, which was only isolated from species of Fabaceae; Fig. 4.7). Focusing on experimental assessments of host specificity, five of the 11 pathogenic isolates generated significant disease symptoms in seedlings of two to four tree species. Furthermore, among the six pathogenic isolates that successfully attacked only a single tree species, five were originally isolated from an alternate host species (Table 4.4), supporting their classification as multihost pathogens. The majority of pathogenic isolates were capable of successfully attacking heterofamilial tree species. Even more striking is the observation that our pathogenic isolates are closely related to fungi isolated as endophytes from grasses within 40 km of our study areas

(Higgins et al. 2011, see the Results in Ch. 3). This reinforces our observation that fungi with the capacity to act as plant pathogens have relatively wide host ranges.

Only one of the 11 pathogenic isolates appears to be specialized. Isolate 2010.52.2.1 (*Fusarium* sp.) was originally isolated from a *Castilla elastica* (Moraceae) seedling, only produced significant disease symptoms in *C. elastica* seedlings, and failed to produce disease symptoms in a confamilial species, *Brosimum utile* (Figs. 4.2-4.6, Table 4.4). Still, we cannot definitively classify isolate 2010.52.2.1 as a specialist because *Copaifera aromatica* (Fabaceae) seedlings inoculated with isolate 2010.52.2.1 exhibited some evidence of pathogen attack (prop. of inoculated seedlings with symptoms – prop. of control seedlings with symptoms > 0, but nonsignificant; Figs. 4.2, 4.4, and 4.6). Nor can we rule out the possibility that isolate 2010.52.2.1 infected an additional tree species against which it was tested but the infection was asymptomatic and went unobserved.

Consistent with our results, host generalism has been observed for pathogenic fungi attacking temperate seedlings (Hersh et al. 2012), for pathogenic oomycetes attacking seedlings in the forests of Barro Colorado Island, Panama (Augspurger and Wilkinson 2007), and for fungal pathogens attacking leaves in Panama (Gilbert and Webb 2007, Schweizer et al. 2013). Furthermore, host generalism has been observed for plant-associated fungi in different functional guilds, including fungal endophytes of tropical grasses (Higgins et al. 2011), seed-infecting fungi (Kluger et al. 2008), and wood-decaying fungi (Ferrer and Gilbert 2003). Thus, running contrary to the explicit assumption of host specificity made by the Janzen-Connell hypothesis, this study adds to growing evidence that host generalism may be the rule rather than the exception for at

least some guilds of plant-associated fungi. Yet, none of the pathogenic species successfully attacked all of the tree species against which they were tested. Differential vulnerability to pathogens among tree species suggests that multihost phytopathogens can contribute to variability in seedling survival among tree species and, thus, influence plant community diversity.

The weak positive relationship between the number of tree species that a pathogenic fungal species successfully attacked and the frequency with which that species was isolated suggests that generalist phytopathogens are relatively abundant members of the phytopathogen communities (Fig. 4.8 gray circles). This pattern is concordant with fungi residing within dead or dying seedlings in a temperate forest (Hersh et al. 2012), foliar fungal pathogens attacking native and nonnative clovers in California (Parker and Gilbert 2007), and seed-infecting fungi in Panama (Gallery et al. 2007). Conversely, the rare fungal species (singletons) in our study may be relatively specialized. In support of this hypothesis, fungal species 26 was rare and successfully attacked only one of the 18 tree species against which it was tested. Alternatively, the fungal species rarely observed may be equally generalized, but our limited sampling failed to capture their actual host range. The latter seems likely given that all but one of the fungal species isolated more than once were isolated from more than one tree species (Fig. 4.7).

While there is compelling evidence that the phytopathogens attacking seedlings are multihost and some appear to have relatively broad host ranges, we cannot accurately describe the breadth of their host ranges because a given phytopathogen was tested against, at most, 29 tree species (isolate 2010.ES81; Fig. 4.2), a small fraction of the

hundreds of tree species that are present in the local forests (Pyke et al. 2001). However, we speculate that the host ranges of the multihost pathogens are broader than the ranges observed by our survey-based and experimental approaches because the number of host species from which a given fungal species was isolated failed to level out (Fig. 4.8 black circles). This suggests that, if the sampling effort were expanded, a given fungal species would be detected in additional host tree species.

The presence of a particular fungal species in the seedlings of multiple host tree species does not mean that those hosts are equally likely to be infected by that fungal species. Even host-generalized pathogens may be more likely to infect certain hosts over others as a result of each host's relative abundance, defense traits, and/or occurrence in a habitat with environmental conditions amenable to pathogen attack (Ferrer and Gilbert 2003). Our experiments were not designed to detect how the likelihood of infection differs among host tree species that share a multihost pathogen and this remains a compelling question.

There is no phylogenetic signal to the host ranges of multihost phytopathogens attacking seedlings

Contrary to our second hypothesis that the likelihood of any two tree species sharing a pathogen increases with increasing phylogenetic relatedness, we observed phylogenetically dispersed host ranges and no evidence for a phylogenetic signal to the host ranges of multihost pathogens. The absence of a significant phylogenetic signal is consistent with one Panama-based study of foliar pathogens (Schweizer et al. 2013) but conflicts with two other studies that observed a continuous decline in the likelihood of

sharing a fungal pathogen with increasing distance between host species (Gilbert and Webb 2007, Gilbert et al. 2012). Our study did not include as many congeneric or confamilial host pairs as Gilbert and Webb (2007) or Gilbert et al. (2012); hence, our conclusions about phylogenetic signal are tentative.

The lack of a phylogenetic signal to the host ranges of the phytopathogens in our study suggests that the plant traits that influence how seedlings and pathogens interact may evolve rapidly. The same argument was made for highly divergent antiherbivore defenses among species of *Inga*, a genus of tropical trees (Kursar et al. 2009). Parallel evolution of similar defenses in unrelated lineages would explain why the phytopathogens in this and other studies can infect many distantly related tree species. Additional studies are needed to test the hypothesis that the defenses of host species that are vulnerable to the same phytopathogen(s) are more similar than expected by chance.

Tree species differ in their vulnerability to pathogens

In support of our third hypothesis and consistent with previous studies of seedling and foliar pathogens, the 36 tree species tested differed in their vulnerability to pathogen attack (Fig. 4.2; Augspurger 1984, Augspurger and Kelly 1984, Augspurger and Wilkinson 2007, Gilbert and Webb 2007). Twelve of the 36 tree species tested were successfully attacked by at least one of the pathogenic isolates tested and six were susceptible to multiple pathogenic isolates (Table 4.4). In contrast, eight of the tree species tested did not exhibit evidence of morbidity for any of the fungal isolates against which they were tested (Fig. 4.2).

In addition to differences among tree species in vulnerability to pathogen attack, those host tree species sharing a multihost phytopathogen were differentially impacted by infection (Table 4.4). For example, infection by isolate 2010.160T, a multihost phytopathogen, led to death for most but not all of its hosts (Table 4.4). Host-specific impacts of infection have also been observed for pathogenic oomycetes of tropical seedlings (Davidson 2000, Davidson et al. 2000, Augspurger and Wilkinson 2007) and for fungal pathogens of temperate seedlings (Hersh et al. 2012).

While pathogen-caused seedling mortality that unevenly affects different plant species can directly influence plant community composition, nonlethal infections can also be a structuring force. Stem damage, wilted tissue, and stunted growth could predispose seedlings to death under stressful abiotic conditions such as seasonal drought. Additionally, nonlethal infections have the potential to compromise a seedling's competitive ability and alter competitive interactions among co-occurring species (e.g., Ditommaso and Watson 1995). If the plant species that is most severely impacted is the superior competitor, the differential impacts among plant species could enhance plant community diversity.

Most of the tree species tested were seemingly resistant to or suffered minimal impact from all of the pathogenic isolates (Fig. 4.2). However, as described above, the absence of obvious symptoms does not necessarily equate with a lack of infection. The susceptible and seemingly nonsusceptible tree species may actually differ in their tolerance of infection rather than their susceptibility to infection. In which case, the tolerant tree species that are not killed or severely impacted by infection may act as reservoir hosts that facilitate the persistence of a phytopathogen within the community, as

is the case for *Phytophthora ramorum*, the oomycete responsible for Sudden Oak Death (Haas et al. 2011).

As we posited in the Introduction, tree species may be differentially vulnerable to disease because the cost-to-benefit ratio of defenses against phytopathogens differs among species (Strauss and Agrawal 1999, Endara and Coley 2010). If a trade-off between defenses and competitive ability exists, selection should favor competitive ability over defenses in environments with relatively low risk of phytopathogen attack. Forthcoming work by the authors will investigate the links between vulnerability to pathogen attack and the habitat associations and functional traits of tree species (e.g., shade-tolerance, drought-tolerance, seed size, tissue toughness, growth rate, and lifespan).

Conclusions, potential consequences for plant community composition, and future directions

Here we identified some of the fungal phytopathogens attacking seedlings in the tropical forests of Panama and we showed that (i) the phytopathogens are capable of attacking multiple host species, (ii) there is no phylogenetic signal to the host ranges of the generalist phytopathogens, and (iii) tree species differ in their vulnerability to phytopathogens. To date, only two other studies have both identified and examined the host specificities of the phytopathogens contributing to seedling mortality in the tropics (Augspurger and Wilkinson 2007, Schweizer et al. 2013).

The maintenance of local plant community diversity by natural enemies has traditionally been ascribed to specialist enemies (Janzen 1970, Connell 1971). However,

variablity among tree species in their vulnerability to pathogens observed by this and other studies (Augspurger and Wilkinson 2007, Hersh et al. 2012, Spear et al. 2015) suggests that generalized phytopathogens have the potential to influence plant community diversity by unevenly affecting seedling recruitment. Host-generalized phytopathogens could enhance plant community diversity if the plant species that are superior competitors are less resistant to or tolerant of attack by the generalist phytopathogens. Conversely, generalist phytopathogens could reduce plant community diversity if the plant species that are superior competitors are more resistant to or tolerant of attack, by reinforcing competitive dominance (Viola et al. 2010). Therefore, to determine if the host-generalized phytopathogens commonly attacking seedlings in the tropical forests of Panama are contributing to the maintenance of local diversity, it is necessary to determine whether or not a competition-defense tradeoff exists among the host plants. Future work should include competition experiments between coexisting species. Information about competitive ability and disease sensitivity should then be related to the observed relative abundances and spatial distributions of plant species.

Interspecific variation in host vulnerability to generalist pathogens may also contribute to the maintenance of regional forest diversity by excluding disease-sensitive tree species from disease-prone habitats (i.e., restricting the ranges of host species). In a Panama-based reciprocal transplant experiment, we showed that there is an elevated risk of pathogen-caused damage and mortality for seedlings in the wetter versus drier forests and a greater impact from pathogen attack for seedlings of tree species typical of the drier versus wetter forests (Spear et al. 2015). Thus, phytopathogens contribute to the exclusion of dry-forest tree species from the wetter forests. As suggested by models of

feedback between plants and the soil biota, plants influence the community of organisms in their vicinity and host-specific pathogens should accumulate in the vicinity of their hosts (Bever et al. 2012). Under the same logic, the absence of dry-forest tree species in the wetter forests suggests that any phytopathogens specific to them should also be absent. Hence, we hypothesize that relatively generalized phytopathogens, like those documented in this study, must contribute to the exclusion of the disease-sensitive, dry-forest species from the wetter forest. While the maintenance of local diversity has commonly been attributed to specialized phytopathogens, our work suggests that the phytopathogens attacking seedlings may be more generalized in their host range and have an underappreciated impact on regional diversity.

Table 4.1. Tree species from which the fungi were isolated (original host) and/or for which vulnerability to phytopathogen attack was assessed (target) via inoculation experiments. Family and species names are based on the Tropicos database of the Missouri Botanical Garden.

Species	Code	Family	Original host (OH) and/or target (T) tree species
Anacardium excelsum	AE	Anacardiaceae	OH, T
Annona glabra	AG	Annonaceae	T
Brosimum utile	BU	Moraceae	OH, T
Calophyllum longifolium	CL	Calophyllaceae	OH, T
Cassia moschata	C2	Fabaceae	OH
Castilla elastica	CE	Moraceae	OH, T
Coccoloba manzinellensis	C1	Polygonaceae	T
Cochlospermum vitifolium	CV	Bixaceae	OH, T
Cojoba rufescens	CR	Fabaceae	OH, T
Copaifera aromatica	CA	Fabaceae	T
Dalbergia retusa	DR	Fabaceae	OH, T
Desmopsis panamensis	DP	Annonaceae	T
Dipteryx oleifera	DO	Fabaceae	OH
Eugenia nesiotica	EN	Myrtaceae	T
Garcinia intermedia	GI	Clusiaceae	T
Genipa americana	GA	Rubiaceae	OH, T
Guapira standleyana	GS	Nyctaginaceae	T
Hymenaea courbaril	HC	Fabaceae	OH, T
Inga goldmanii	IG	Fabaceae	T
Inga sapindoides	IS	Fabaceae	T
Jacaranda copaia	JC	Bignoniaceae	T
Lacistema aggregatum	LA	Lacistemataceae	T
Lacmellea panamensis	L2	Apocynaceae	OH, T
Licania platypus	L1	Chrysobalanaceae	T
Luehea seemannii	LS	Malvaceae	T
Nectandra cuspidata	NC	Lauraceae	OH
Ormosia coccinea	OM	Fabaceae	OH
Ormosia macrocalyx	OC	Fabaceae	OH, T
Pachira quinata	PQ	Malvaceae	T
Posoqueria latifolia	P1	Rubiaceae	T
Protium tenuifolium	PT	Burseraceae	OH
Psychotria limonensis	P2	Rubiaceae	T
Psychotria marginata	PM	Rubiaceae	T
Quararibea asterolepis	QA	Malvaceae	T
Randia armata	RA	Rubiaceae	OH
Siparuna pauciflora	SP	Siparunaceae	T
Swartzia simplex	SS	Fabaceae	T
Swietenia macrophylla	SM	Meliaceae	OH
Symphonia globulifera	SG	Clusiaceae	T
Tetragastris panamensis	T1	Burseraceae	OH, T
Tocoyena pittieri	T2	Rubiaceae	T
Trichilia tuberculata	TT	Meliaceae	OH, T
Virola surinamensis	VS	Myristicaceae	OH, T

Table 4.2. The 34 (of 93) fungal isolates for which pathogenicity and host range were experimentally evaluated via seedling inoculation experiments. Columns include the fungal isolate identifier; the host tree species and seedling tissue from which it was isolated; a code for the site from which the symptomatic seedling was collected; the fungal species to which the isolate belongs; and estimated taxonomic placement at the genus, family, and, in parentheses, order levels based on phylogenetic analyses. For some isolates, two genera are listed because the teleomorph (sexual) and anamorph (asexual) stages of the same fungus were assigned different Latin names.

Isolate ID	Host species	Seedling part	Coll. site	Fugal species	Genus	Family (Order)
2010.155	*Anacardium excelsum*	stem	SRR	1	unknown	Nectriaceae (Hypocreales)
2010.160T‡	*Virola surinamensis*	stem	SRR	2	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)
2010.168aM	*Tetragastris panamensis*	stem	SRR	2	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)
2010.ES80‡	*Randia armata*	stem	SRR	2	*Mycoleptodiscus*	Magnaporthaceae (Magnaporthales)
2010.143	*Anacardium excelsum*	leaf	SRR	3	*Diaporthe* (anamorph = *Phomopsis*)	Diaporthaceae (Diaporthales)
2010.107.2l	*Cojoba rufescens*	leaf	PNM	3	*Diaporthe* (anamorph = *Phomopsis*)	Diaporthaceae (Diaporthales)
2010.107.2h	*Cojoba rufescens*	leaf	PNM	4	unknown	unknown
2010.171‡	*Hymenaea courbaril*	stem	PNM	4	unknown	unknown
2010.64a2	*Castilla elastica*	stem	PNM	4	unknown	unknown
2010.73bl1	*Virola surinamensis*	stem	PNM	4	unknown	unknown
2010.76	*Hymenaea courbaril*	stem	SRR	5	*Neonectria*	Nectriaceae (Hypocreales)
2010.157b	*Virola surinamensis*	stem	PNM	5	*Neonectria*	Nectriaceae (Hypocreales)
2010.124	*Virola surinamensis*	stem	PNM	5	unknown	Nectriaceae (Hypocreales)

Table 4.2. (continued)

Isolate ID	Host species	Seedling part	Coll. site	Fugal species	Genus	Family (Order)
2011.8ab	*Anacardium excelsum*	root	SC	5	unknown	Nectriaceae (Hypocreales)
2010.137	*Trichilia tuberculata*	stem	GS	6	unknown	Botryosphaeriaceae (Botryosphaeriales)
2010.65.2b	*Castilla elastica*	root	PNM	7	*Nectria* (anamorph = *Fusarium*)	Nectriaceae (Hypocreales)
2010.92.2brn	*Castilla elastica*	stem	SRR	7	*Nectria* (anamorph = *Fusarium*)	Nectriaceae (Hypocreales)
2010.81‡	*Virola surinamensis*	root	SRR	8	*Bionectria* (anamorph = *Clonostachys*)	Bionectriaceae (Hypocreales)
2010.83a‡	*Protium tenuifolium*	leaf	SRR	8	*Bionectria* (anamorph = *Clonostachys*)	Bionectriaceae (Hypocreales)
2010.93FT	*Castilla elastica*	stem	SRR	8	*Bionectria* (anamorph = *Clonostachys*)	Bionectriaceae (Hypocreales)
2010.95‡	*Virola surinamensis*	stem	PNM	8	*Bionectria* (anamorph = *Clonostachys*)	Bionectriaceae (Hypocreales)
2010.bAEC	*Anacardium excelsum*	stem	PNM	8	*Bionectria* (anamorph = *Clonostachys*)	Bionectriaceae (Hypocreales)
2011.4	*Dipteryx oleifera*	stem	BCI	9	*Glomerella* (anamorph = *Colletotrichum*)	Glomerellaceae (Glomerellales)
2010.ES81‡	*Trichilia tuberculata*	root	SRR	10	*Calonectria*	Nectriaceae (Hypocreales)
2011.1b2‡	*Calophyllum longifolium*	stem	BCI	10	*Calonectria*	Nectriaceae (Hypocreales)
2011.7b‡	*Anacardium excelsum*	stem	SC	10	*Calonectria*	Nectriaceae (Hypocreales)
2010.ES82	*Genipa americana*	root	SRR	11	*Hypocrea* (anamorph = *Trichoderma*)	Hypocreaceae (Hypocreales)
2011.5	*Anacardium excelsum*	stem	PNM	12	unknown	unknown (Xylariales *inc. sed.*)

Table 4.2. (continued)

Isolate ID	Host species	Seedling part	Coll. site	Fugal species	Genus	Family (Order)
2011.2b	*Anacardium excelsum*	stem	BCI	13	*Glomerella* (anamorph = *Colletotrichum*)	Glomerellaceae (Glomerellales)
2010.172‡	*Trichilia tuberculata*	root	PNM	18	*Pestalotiopsis*	Amphisphaeriaceae (Xylariales)
2010.151	*Anacardium excelsum*	stem	PNM	24	unknown	unknown
2010.156a	*Cojoba rufescens*	stem	GS	25	*Albonectria*	Nectriaceae (Hypocreales)
2010.52.2.1‡	*Castilla elastica*	stem	PNM	26	unknown (anamorph = *Fusarium*)	Nectriaceae (Hypocreales)
2011.10	*Anacardium excelsum*	stem	CC	28	*Leuconectria* (anamorph = *Gliocephalotrichum*)	Nectriaceae (Hypocreales)

‡ Isolates that exhibited pathogenicity during the inoculation experiments.
inc. sed. = *incertae sedis* or uncertain placement.
Seedling collection sites are coded as follows: SRR = Santa Rita Ridge, PNM = Parque Natural Metropolitano, BCI = Barro Colorado Island, Barro Colorado Nature Monument, SC = Sendero del Charco, CC = Sendero Camino de Cruces, GS = shadehouse in Gamboa

Table 4.3. Average light levels, air temperatures, and relative humidities of the two shadehouses used for the inoculation experiments. The photosynthetically active radiation (PAR) reaching the seedlings was measured during a uniformly overcast day in 2011 (Oct. 12). Measurements were taken inside and directly outside of the shadehouses with a LI-250 light meter and a one-meter LI-191 line quantum sensor (LI-COR, Lincoln, NE, USA). In each shadehouse, we measured air temperature and relative humidity (CS500 probe, Campbell Scientific, Inc., Logan, UT, USA) every 10 minutes and hourly mean temperature and minimum and maximum relative humidity (RH) were recorded on a CR200 datalogger (Campbell Scientific). Measurements were taken from November 3-5 and November 5-11, 2011 for shadehouses 2 and 1, respectively. We obtained ambient air temperature data for the same time period (Nov 3-11, 2011) from the Office of Bioinformatics at the Smithsonian Tropical Research Institute. Ambient air temperature was measured at a nearby site in Gamboa (elev = 31.4 m, 9º6'44"N, 79º41'38"W) by the Meteorological and Hydrological Branch of the Panama Canal Authority. Corresponding ambient RH data were unavailable. Both shadehouses were used for the inoculation experiments in 2011 and only shadehouse 1 was used in for the inoculation experiments in 2012.

Shadehouse	PAR (% of full sunlight)	Mean shadehouse temperature	Mean ambient temperature	Mean min. RH	Mean max. RH
1	1.5%	26.1ºC	25.6ºC	85.1%	88.1%
2	1.8%	25.6ºC	25.4ºC	87.6%	90.5%

Table 4.4. The 22 fungal isolate-by-tree species combinations for which pathogenicity was observed during the inoculation experiments. Metrics for pathogenicity included the incidence of mortality, stem damage, stunted growth, wilt tissue, and general morbidity. The 11 fungal isolates with confirmed pathogenicity are listed according to the fungal species to which they belong. Listed for each isolate are the identifier code, the estimated taxonomic placement in parentheses, a code for the tree species from which it was originally isolated (see Table 4.1 for full names), the seedling tissue from which it was isolated, and a code for the site from which the symptomatic seedling was collected. For each isolate, the vulnerable tree species are listed in alphabetical order followed by the experiment sample size, the indicative disease metric(s), the proportion of seedlings that suffered that specific disease metric in the inoculated group minus the proportion in the paired control group, and the results of the bias-reduced generalized linear model used to assess significance. The likelihood of disease development was considered significantly different at $P < 0.10$. For two of the fungal isolate-by-tree species combinations, the inoculated seedlings experienced significantly fewer symptoms of pathogen attack than the seedlings in their paired control group (listed in grey).

Fungal species	Fungal isolate	Vuln. species	*N*	Metric	Inoc-cntrl	Est.	SE	Z	*P*	
2	2010.160T (*Mycoleptodiscus* sp.), VS, stem, SRR	AG	24	mortality	0.58	2.033	0.788	2.579	0.010	***
				stem	0.33	1.433	0.792	1.809	0.07	*
				wilt	0.75	2.462	0.8	3.078	0.002	***
				morbidity	0.75	2.462	0.800	3.078	0.002	***
		DR	20	mortality	0.8	2.526	0.848	2.977	0.003	***
				stem	0.6	1.530	0.624	2.45	0.014	**
				wilt	0.5	1.36	0.643	2.117	0.034	**
				morbidity	0.7	1.891	0.669	2.826	0.005	***
		GA	20	stem	0.4	1.254	0.654	1.917	0.055	*
				morbidity	0.5	1.360	0.643	2.117	0.034	**
		P2	20	mortality	0.4	1.527	0.828	1.844	0.065	*
				morbidity	0.4	1.527	0.828	1.844	0.065	*
	2010.ES80 (*Mycoleptodiscus* sp.), RA, stem, SRR	LS	20	mortality	0.9	2.887	0.882	3.274	0.001	****
				stem	0.4	1.923	0.867	2.218	0.027	**
				wilt	0.6	2.737	0.929	2.947	0.003	***
				morbidity	0.9	2.887	0.882	3.274	0.001	***
4	2010.171 (Sordariomycetes sp.), HC, stem, PNM	CA	10	morbidity	0.6	1.685	1.017	1.657	0.098	*

Table 4.4. (continued)

Fungal species	Fungal isolate	Vuln. species	*N*	Metric	Inoc-cntrl	Est.	SE	Z	*P*	
	2010.81 (*Bionectria* sp.), VS, root, SRR	LS	20	mortality	0.4	1.126	0.640	1.759	0.079	*
				morbidity	0.4	1.126	0.640	1.759	0.079	*
8		TT	17	mortality	0.44	1.540	0.867	1.776	0.076	*
				morbidity	0.56	1.796	0.867	2.072	0.038	**
	2010.83a (*Bionectria* sp.), PT, leaf, SRR	CA	10	stem	0.6	1.685	1.017	1.657	0.098	*
				morbidity	0.6	1.685	1.017	1.657	0.098	*
	2010.95 (*Bionectria* sp.), VS, stem, PNM	CL	10	morbidity	0.6	1.685	1.017	1.657	0.098	*
	2011.1b2 (*Calonectria* sp.), CL, stem, BCI	CE	30	mortality	0.33	1.518	0.749	2.026	0.043	**
				stem	0.4	1.107	0.51	2.17	0.03	**
				stunt	0.47	1.425	0.56	2.547	0.011	**
				morbidity	0.6	1.577	0.513	3.072	0.002	***
		CV	20	mortality	0.4	1.126	0.640	1.759	0.079	*
				morbidity	0.4	1.126	0.640	1.759	0.079	*
		DR	30	mortality	0.4	1.107	0.51	2.17	0.03	**
				stem	0.33	0.868	0.487	1.784	0.074	*
				wilt	0.27	1.338	0.754	1.774	0.076	*
10				morbidity	0.47	1.197	0.493	2.427	0.015	**
		GS		mortality	-0.4	-1.126	0.640	-1.759	0.079	*
				morbidity	-0.4	-0.999	0.596	-1.677	0.094	*
	2011.7b (*Calonectria* sp.), AE, stem, SC	CE	19	mortality	0.89	2.827	0.890	3.177	0.002	***
				stunt	0.79	2.192	0.721	3.041	0.002	***
				morbidity	0.9	2.843	0.895	3.178	0.001	***
	2010.ES81 (*Calonectria* sp.), TT, root, SRR	DR	10	mortality	0.8	2.169	1.044	2.076	0.038	**
				morbidity	0.6	1.685	1.017	1.657	0.098	*
		GS	20	stem	0.7	2.244	0.834	2.69	0.007	***
				morbidity	0.6	1.609	0.651	2.471	0.014	**
		LS	10	morbidity	0.4	1.685	1.017	1.657	0.098	*
26	2010.52.2.1 (*Fusarium* sp.), CE, stem, PNM	CE	10	wilt	0.6	1.685	1.017	1.657	0.098	*

Table 4.4. (continued)

Fungal species	Fungal isolate	Vuln. species	*N*	Metric	Inoc-cntrl	Est.	SE	Z	*P*	
	2010.172 (*Pestalotiopsis* sp.), TT, root, PNM	BU	20	mortality	0.4	1.527	0.828	1.844	0.065	*
				morbidity	0.4	1.527	0.828	1.844	0.065	*
18		CE	10	stunt	0.6	1.685	1.017	1.657	0.098	*
		CV	20	stem	0.4	0.999	0.596	1.677	0.094	*
		GA	20	mortality	0.4	1.126	0.640	1.759	0.079	*
				morbidity	0.4	1.126	0.640	1.759	0.079	*
28	2011.10 (*Leuconectria* sp.), AE, stem, CC	HC	10	mortality	-0.6	-1.685	1.017	-1.657	0.098	*

Significance is indicated by asterisks (*$P \leq 0.10$, ** $P \leq 0.05$, ***$P \leq 0.01$, ****$P \leq 0.001$).
Seedling collection sites are coded as follows: SRR = Santa Rita Ridge, PNM = Parque Natural Metropolitano, BCI = Barro Colorado Island, Barro Colorado Nature Monument, SC = Sendero del Charco, CC = Sendero Camino de Cruces.

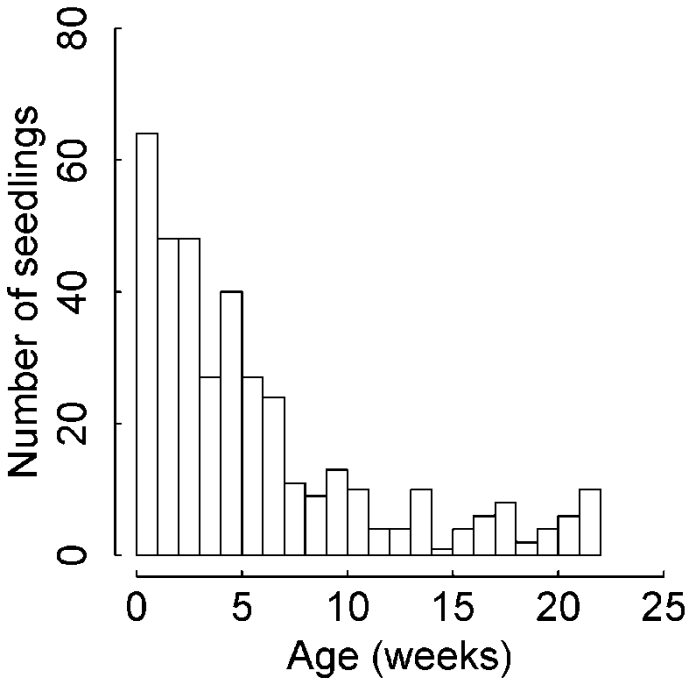
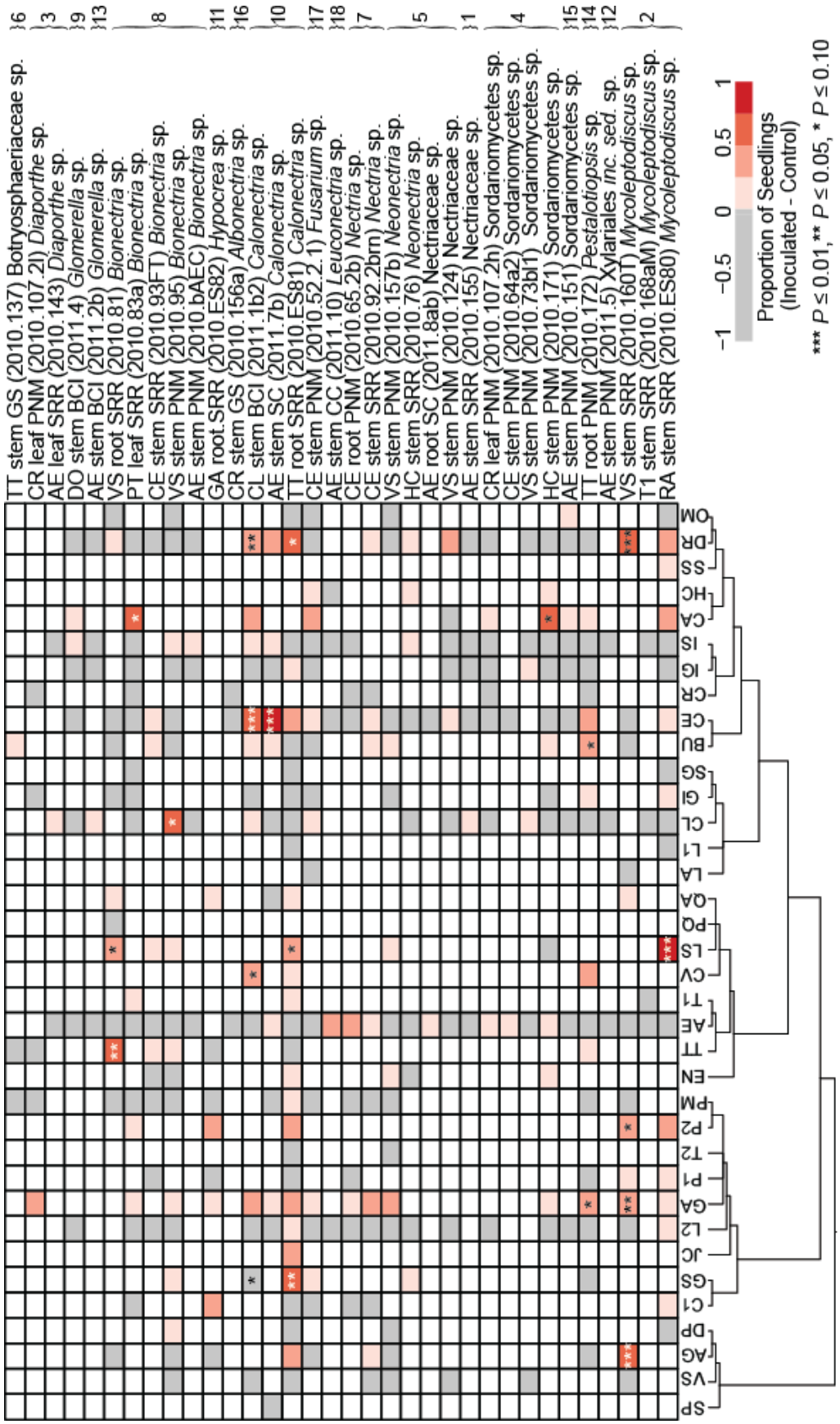


Fig. 4.1. Frequency distribution of age (in weeks) for the seedlings that suffered pathogen-caused death during the 2011 inoculation experiments (N = 382). The median age was five weeks.

Fig. 4.2. Matrix depicting the pathogenicity of the 34 fungal isolates tested and the vulnerability of the 36 tree species tested based on morbidity. Listed along the vertical axis, the fungal isolates tested are arranged according to their class, order, and family (when known) and are grouped according to the fungal species to which they belong as specified by the numbered brackets (Table 4.2). The following is listed for each fungal isolate: a code for the original host tree species (Table 4.1), the seedling tissue from which it was isolated, a code for the site from which the symptomatic seedling was collected, the isolate's identifier (in parentheses), and its estimated taxonomic placement. Seedling collection sites are coded as follows: GS = shadehouse in Gamboa, PNM = Parque Natural Metropolitano, SRR = Santa Rita Ridge, BCI = Barro Colorado Island, SC = Sendero del Charco, and CC = Sendero Camino de Cruces. Along the horizontal axis, the tree species tested (targets; Table 4.1) are arranged based on their evolutionary relationships and the topology of the cladogram is based on a recently developed supertree (R2G2 tree and ages file; G. S. Gilbert, unpublished manuscript). Tree species-by-fungal isolate combinations for which evidence of pathogenicity was observed (proportions > 0) are depicted in shades of red, with the relative extent of disease indicated by color saturation (from pale pink for minimal to red for substantial). Combinations for which there was no evidence of pathogenicity (proportions ≤ 0) are depicted in gray. White cells indicate that the combination was not tested. Significant differences ($P < 0.10$) in the likelihood of disease development between an inoculated group and its inoculum-free, paired control are identified with asterisks. For 19 of the 332 tree species-by-fungal isolate combinations tested, the inoculated seedlings suffered significant morbidity. For one combination, a significantly greater proportion of the seedlings in the control group had symptoms of disease than the seedlings inoculated with the fungal isolate being evaluated (*Guapira standleyana* and isolate 2011.1b2; Table 4.4).


TT stem GS (2010.137) Botryosphaeriaceae sp.
CR leaf PNM (2010.107.2l) Diaporthe sp.
AE leaf SRR (2010.143) Diaporthe sp.
DO stem BCI (2011.4) Glomerella sp.
AE stem BCI (2011.2b) Glomerella sp.
VS root SRR (2010.81) Bionectria sp.
PT leaf SRR (2010.83a) Bionectria sp.
CE stem SRR (2010.93FT) Bionectria sp.
VS stem PNM (2010.95) Bionectria sp.
AE stem PNM (2010.bAEC) Bionectria sp.
GA root.SRR (2010.ES82) Hypocrea sp.
CR stem GS (2010.156a) Albonectria sp.
CL stem BCI (2011.1b2) Calonectria sp.
AE stem SC (2011.7b) Calonectria sp.
TT root SRR (2010.ES81) Calonectria sp.
CE stem PNM (2010.52.2.1) Fusarium sp.
AE stem CC (2011.10) Leuconectria sp.
CE root PNM (2010.65.2b) Nectria sp.
CE stem SRR (2010.92.2brn) Nectria sp.
VS stem PNM (2010.157b) Neonectria sp.
HC stem SRR (2010.76) Neonectria sp.
AE root SC (2011.8ab) Nectriaceae sp.
VS stem PNM (2010.124) Nectriaceae sp.
AE stem SRR (2010.155) Nectriaceae sp.
CR leaf PNM (2010.107.2h) Sordariomycetes sp.
CE stem PNM (2010.64a2) Sordariomycetes sp.
VS stem PNM (2010.73b1) Sordariomycetes sp.
HC stem PNM (2010.171) Sordariomycetes sp.
AE stem PNM (2010.151) Sordariomycetes sp.
TT root PNM (2010.172) Pestalotiopsis sp.
AE stem PNM (2011.5) Xylariales inc. sed. sp.
VS stem SRR (2010.160T) Mycoleptodiscus sp.
T1 stem SRR (2010.168aM) Mycoleptodiscus sp.
RA stem SRR (2010.ES80) Mycoleptodiscus sp.
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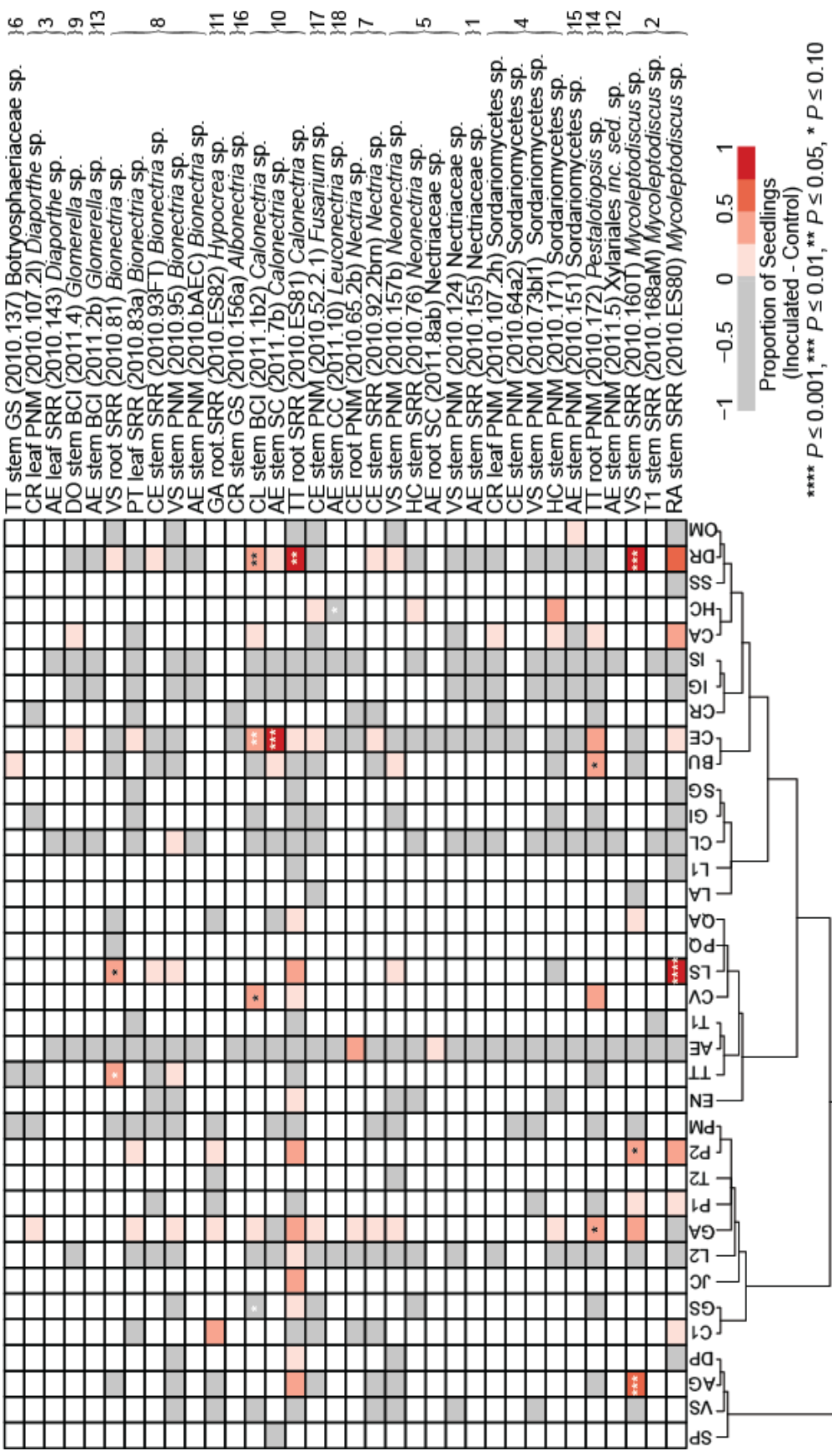
Proportion of Seedlings
(Inoculated - Control)
*** P ≤ 0.01, ** P ≤ 0.05, * P ≤ 0.10

Fig. 4.3. Matrix depicting the pathogenicity of the 34 fungal isolates tested and the vulnerability of the 36 tree species tested based on seedling mortality. Listed along the vertical axis, the fungal isolates tested are arranged according to their class, order, and family (when known) and are grouped according to the fungal species to which they belong as specified by the numbered brackets (Table 4.2). The following is listed for each fungal isolate: a code for the original host tree species (Table 4.1), the seedling tissue from which it was isolated, a code for the site from which the symptomatic seedling was collected, the isolate's identifier (in parentheses), and its estimated taxonomic placement. Seedling collection sites are coded as follows: GS = shadehouse in Gamboa, PNM = Parque Natural Metropolitano, SRR = Santa Rita Ridge, BCI = Barro Colorado Island, SC = Sendero del Charco, and CC = Sendero Camino de Cruces. Along the horizontal axis, the tree species tested (targets; Table 4.1) are arranged based on their evolutionary relationships and the topology of the cladogram is based on a recently developed supertree (R2G220140601 tree and ages file; G. S. Gilbert, unpublished manuscript). Tree species-by-fungal isolate combinations for which evidence of pathogenicity was observed (proportions > 0) are depicted in shades of red, with the relative extent of disease indicated by color saturation (from pale pink for minimal to red for substantial). Combinations for which there was no evidence of pathogenicity (proportions ≤ 0) are depicted in gray. White cells indicate that the combination was not tested. Significant differences ($P < 0.10$) in the likelihood of disease development between an inoculated group and its inoculum-free, paired control are identified with asterisks. For 13 of the 332 tree species-by-fungal isolate combinations tested, the inoculated seedlings suffered significant mortality. For two combinations, a significantly greater proportion of the control seedlings suffered pathogen-caused mortality than the inoculated seedlings (*Guapira standleyana* and isolate 2011.1b2 and *Hymenaea courbaril* and isolate 2011.10; Table 4.4).


TT stem GS (2010.137) Botryosphaeriaceae sp.
CR leaf PNM (2010.107.2l) Diaporthe sp.
AE leaf SRR (2010.143) Diaporthe sp.
DO stem BCI (2011.4) Glomerella sp.
AE stem BCI (2011.2b) Glomerella sp.
VS root SRR (2010.81) Bionectria sp.
PT leaf SRR (2010.83a) Bionectria sp.
CE stem SRR (2010.93FT) Bionectria sp.
VS stem PNM (2010.95) Bionectria sp.
AE stem PNM (2010.bAEC) Bionectria sp.
GA root.SRR (2010.ES82) Hypocrea sp.
CR stem GS (2010.156a) Albonectria sp.
CL stem BCI (2011.1b2) Calonectria sp.
AE stem SC (2011.7b) Calonectria sp.
TT root SRR (2010.ES81) Calonectria sp.
CE stem PNM (2010.52.2.1) Fusarium sp.
AE stem CC (2011.10) Leuconectria sp.
CE root PNM (2010.65.2b) Nectria sp.
CE stem SRR (2010.92.2brn) Nectria sp.
VS stem PNM (2010.157b) Neonectria sp.
HC stem SRR (2010.76) Neonectria sp.
AE root SC (2011.8ab) Nectriaceae sp.
VS stem PNM (2010.124) Nectriaceae sp.
AE stem SRR (2010.155) Nectriaceae sp.
CR leaf PNM (2010.107.2h) Sordariomycetes sp.
CE stem PNM (2010.64a2) Sordariomycetes sp.
VS stem PNM (2010.73bl1) Sordariomycetes sp.
HC stem PNM (2010.171) Sordariomycetes sp.
AE stem PNM (2010.151) Sordariomycetes sp.
TT root PNM (2010.172) Pestalotiopsis sp.
AE stem PNM (2011.5) Xylariales inc. sed. sp.
VS stem SRR (2010.160T) Mycoleptodiscus sp.
T1 stem SRR (2010.168aM) Mycoleptodiscus sp.
RA stem SRR (2010.ES80) Mycoleptodiscus sp.
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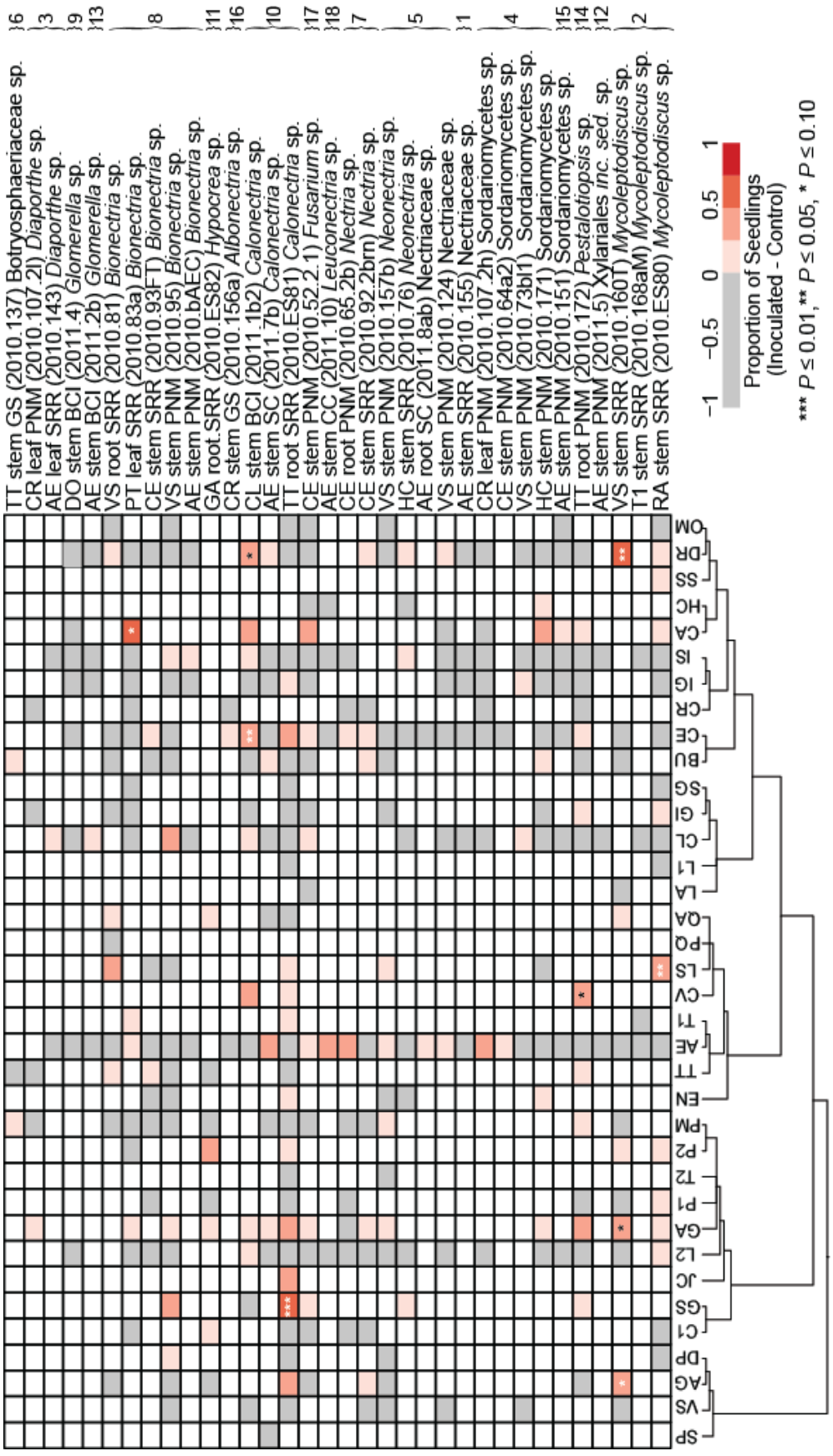
Proportion of Seedlings
(Inoculated - Control)
**** P ≤ 0.001, *** P ≤ 0.01, ** P ≤ 0.05, * P ≤ 0.10

Fig. 4.4. Matrix depicting the pathogenicity of the 34 fungal isolates tested and the vulnerability of the 36 tree species tested based on stem damage. Listed along the vertical axis, the fungal isolates tested are arranged according to their class, order, and family (when known) and are grouped according to the fungal species to which they belong as specified by the numbered brackets (Table 4.2). The following is listed for each fungal isolate: a code for the original host tree species (Table 4.1), the seedling tissue from which it was isolated, a code for the site from which the symptomatic seedling was collected, the isolate's identifier (in parentheses), and its estimated taxonomic placement. Seedling collection sites are coded as follows: GS = shadehouse in Gamboa, PNM = Parque Natural Metropolitano, SRR = Santa Rita Ridge, BCI = Barro Colorado Island, SC = Sendero del Charco, and CC = Sendero Camino de Cruces. Along the horizontal axis, the tree species tested (targets; Table 4.1) are arranged based on their evolutionary relationships and the topology of the cladogram is based on a recently developed supertree (R2G2 tree and ages file; G. S. Gilbert, unpublished manuscript). Tree species-by-fungal isolate combinations for which evidence of pathogenicity was observed (proportions > 0) are depicted in shades of red, with the relative extent of disease indicated by color saturation (from pale pink for minimal to red for substantial). Combinations for which there was no evidence of pathogenicity (proportions ≤ 0) are depicted in gray. White cells indicate that the combination was not tested. Significant differences ($P < 0.10$) in the likelihood of disease development between an inoculated group and its inoculum-free, paired control are identified with asterisks. For nine of the 332 tree species-by-fungal isolate combinations tested, the inoculated seedlings suffered significant pathogen-caused stem damage (Table 4.4).


TT stem GS (2010.137) Botryosphaeriaceae sp.
CR leaf PNM (2010.107.2l) Diaporthe sp.
AE leaf SRR (2010.143) Diaporthe sp.
DO stem BCI (2011.4) Glomerella sp.
AE stem BCI (2011.2b) Glomerella sp.
VS root SRR (2010.81) Bionectria sp.
PT leaf SRR (2010.83a) Bionectria sp.
CE stem SRR (2010.93FT) Bionectria sp.
VS stem PNM (2010.95) Bionectria sp.
AE stem PNM (2010.bAEC) Bionectria sp.
GA root.SRR (2010.ES82) Hypocrea sp.
CR stem GS (2010.156a) Albonectria sp.
CL stem BCI (2011.1b2) Calonectria sp.
AE stem SC (2011.7b) Calonectria sp.
TT root SRR (2010.ES81) Calonectria sp.
CE stem PNM (2010.52.2.1) Fusarium sp.
AE stem CC (2011.10) Leuconectria sp.
CE root PNM (2010.65.2b) Nectria sp.
CE stem SRR (2010.92.2brn) Nectria sp.
VS stem PNM (2010.157b) Neonectria sp.
HC stem SRR (2010.76) Neonectria sp.
AE root SC (2011.8ab) Nectriaceae sp.
VS stem PNM (2010.124) Nectriaceae sp.
AE stem SRR (2010.155) Nectriaceae sp.
CR leaf PNM (2010.107.2h) Sordariomycetes sp.
CE stem PNM (2010.64a2) Sordariomycetes sp.
VS stem PNM (2010.73bl1) Sordariomycetes sp.
HC stem PNM (2010.171) Sordariomycetes sp.
AE stem PNM (2010.151) Sordariomycetes sp.
TT root PNM (2010.172) Pestalotiopsis sp.
AE stem PNM (2011.5) Xylariales inc. sed. sp.
VS stem SRR (2010.160T) Mycoleptodiscus sp.
T1 stem SRR (2010.168aM) Mycoleptodiscus sp.
RA stem SRR (2010.ES80) Mycoleptodiscus sp.
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Proportion of Seedlings
(Inoculated - Control)
*** P ≤ 0.01, ** P ≤ 0.05, * P ≤ 0.10

Fig. 4.5. Matrix depicting the pathogenicity of the 34 fungal isolates tested and the vulnerability of the 36 tree species tested based on wilted tissue. Listed along the vertical axis, the fungal isolates tested are arranged according to their class, order, and family (when known) and are grouped according to the fungal species to which they belong as specified by the numbered brackets (Table 4.2). The following is listed for each fungal isolate: a code for the original host tree species (Table 4.1), the seedling tissue from which it was isolated, a code for the site from which the symptomatic seedling was collected, the isolate's identifier (in parentheses), and its estimated taxonomic placement. Seedling collection sites are coded as follows: GS = shadehouse in Gamboa, PNM = Parque Natural Metropolitano, SRR = Santa Rita Ridge, BCI = Barro Colorado Island, SC = Sendero del Charco, and CC = Sendero Camino de Cruces. Along the horizontal axis, the tree species tested (targets; Table 4.1) are arranged based on their evolutionary relationships and the topology of the cladogram is based on a recently developed supertree (R2G2 tree and ages file; G. S. Gilbert, unpublished manuscript). Tree species-by-fungal isolate combinations for which evidence of pathogenicity was observed (proportions > 0) are depicted in shades of red, with the relative extent of disease indicated by color saturation (from pale pink for minimal to red for substantial). Combinations for which there was no evidence of pathogenicity (proportions ≤ 0) are depicted in gray. White cells indicate that the combination was not tested. Significant differences ($P < 0.10$) in the likelihood of disease development between an inoculated group and its inoculum-free, paired control are identified with asterisks. For five of the 332 tree species-by-fungal isolate combinations tested, the inoculated seedlings suffered significant pathogen-caused wilt (Table 4.4).

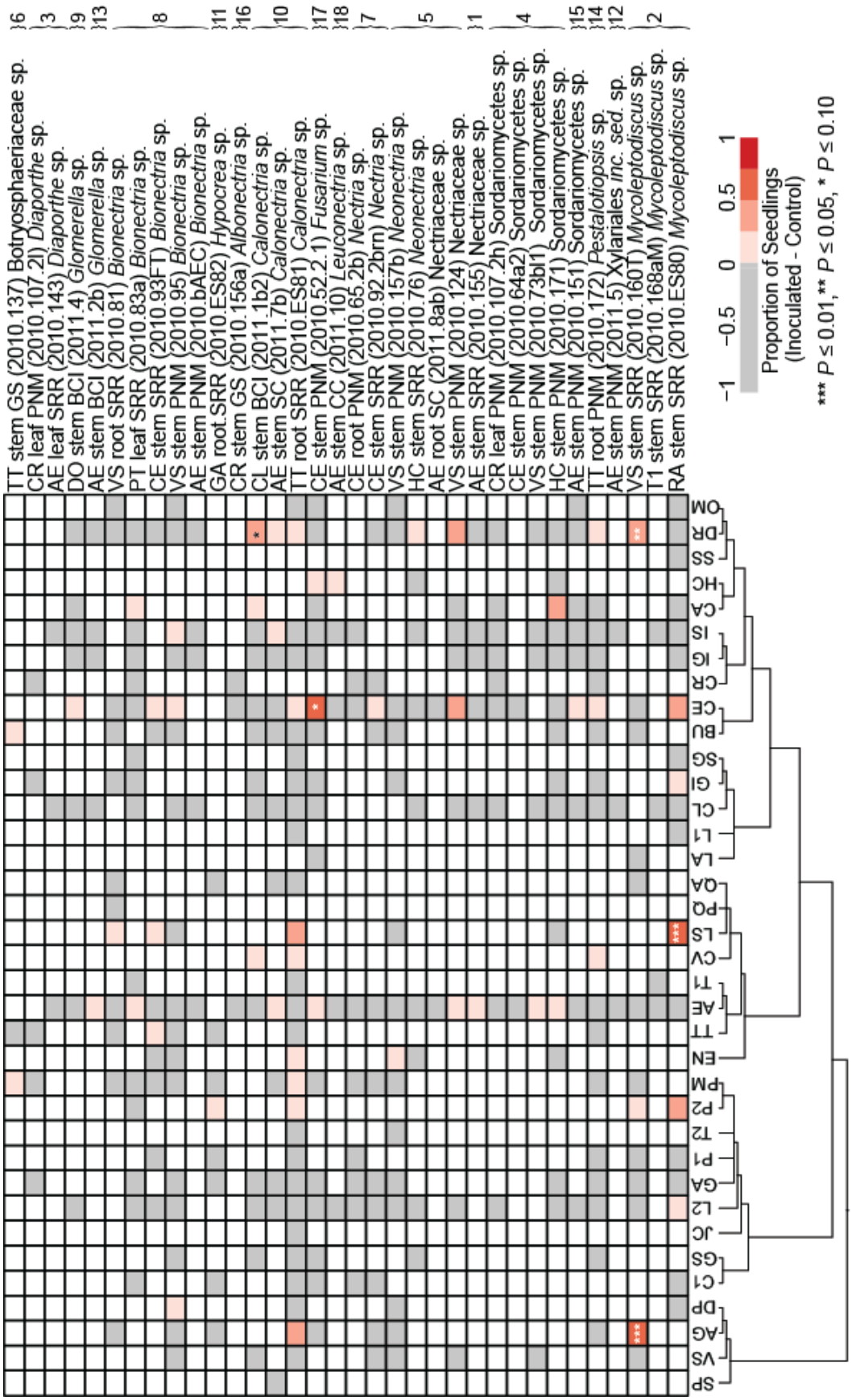
TT stem GS (2010.137) Botryosphaeriaceae sp.
CR leaf PNM (2010.107.2l) Diaporthe sp.
AE leaf SRR (2010.143) Diaporthe sp.
DO stem BCI (2011.4) Glomerella sp.
AE stem BCI (2011.2b) Glomerella sp.
VS root SRR (2010.81) Bionectria sp.
PT leaf SRR (2010.83a) Bionectria sp.
CE stem SRR (2010.93FT) Bionectria sp.
VS stem PNM (2010.95) Bionectria sp.
AE stem PNM (2010.bAEC) Bionectria sp.
GA root.SRR (2010.ES82) Hypocrea sp.
CR stem GS (2010.156a) Albonectria sp.
CL stem BCI (2011.1b2) Calonectria sp.
AE stem SC (2011.7b) Calonectria sp.
TT root SRR (2010.ES81) Calonectria sp.
CE stem PNM (2010.52.2.1) Fusarium sp.
AE stem CC (2011.10) Leuconectria sp.
CE root PNM (2010.65.2b) Nectria sp.
CE stem SRR (2010.92.2brn) Nectria sp.
VS stem PNM (2010.157b) Neonectria sp.
HC stem SRR (2010.76) Neonectria sp.
AE root SC (2011.8ab) Nectriaceae sp.
VS stem PNM (2010.124) Nectriaceae sp.
AE stem SRR (2010.155) Nectriaceae sp.
CR leaf PNM (2010.107.2h) Sordariomycetes sp.
CE stem PNM (2010.64a2) Sordariomycetes sp.
VS stem PNM (2010.73bl1) Sordariomycetes sp.
HC stem PNM (2010.171) Sordariomycetes sp.
AE stem PNM (2010.151) Sordariomycetes sp.
TT root PNM (2010.172) Pestalotiopsis sp.
AE stem PNM (2011.5) Xylariales inc. sed. sp.
VS stem SRR (2010.160T) Mycoleptodiscus sp.
T1 stem SRR (2010.168aM) Mycoleptodiscus sp.
RA stem SRR (2010.ES80) Mycoleptodiscus sp.
16
3
9
13
8
11
16
10
17
18
7
5
1
4
15
14
12
2
SP
VS
AG
DP
C1
GS
JC
L2
GA
P1
T2
P2
PM
EN
TT
AE
T1
CV
LS
PQ
QA
LA
L1
CL
GI
SG
BU
CE
CR
IG
IS
CA
HC
SS
DR
OM
−1
−0.5
0
0.5
1
Proportion of Seedlings
(Inoculated - Control)
*** P ≤ 0.01, ** P ≤ 0.05, * P ≤ 0.10

Fig. 4.6. Matrix depicting the pathogenicity of the 34 fungal isolates tested and the vulnerability of the 36 tree species tested based on stunted growth. Listed along the vertical axis, the fungal isolates tested are arranged according to their class, order, and family (when known) and are grouped according to the fungal species to which they belong as specified by the numbered brackets (Table 4.2). The following is listed for each fungal isolate: a code for the original host tree species (Table 4.1), the seedling tissue from which it was isolated, a code for the site from which the symptomatic seedling was collected, the isolate's identifier (in parentheses), and its estimated taxonomic placement. Seedling collection sites are coded as follows: GS = shadehouse in Gamboa, PNM = Parque Natural Metropolitano, SRR = Santa Rita Ridge, BCI = Barro Colorado Island, SC = Sendero del Charco, and CC = Sendero Camino de Cruces. Along the horizontal axis, the tree species tested (targets; Table 4.1) are arranged based on their evolutionary relationships and the topology of the cladogram is based on a recently developed supertree (R2G2 tree and ages file; G. S. Gilbert, unpublished manuscript). Tree species-by-fungal isolate combinations for which evidence of pathogenicity was observed (proportions > 0) are depicted in shades of red, with the relative extent of disease indicated by color saturation (from pale pink for minimal to red for substantial). Combinations for which there was no evidence of pathogenicity (proportions ≤ 0) are depicted in gray. White cells indicate that the combination was not tested. Significant differences ($P < 0.10$) in the likelihood of disease development between an inoculated group and its inoculum-free, paired control are identified with asterisks. For three of the 332 tree species-by-fungal isolate combinations tested, all involving the tree species *Castilla elastica*, the inoculated seedlings suffered significant pathogen-caused stunted growth (Table 4.4).

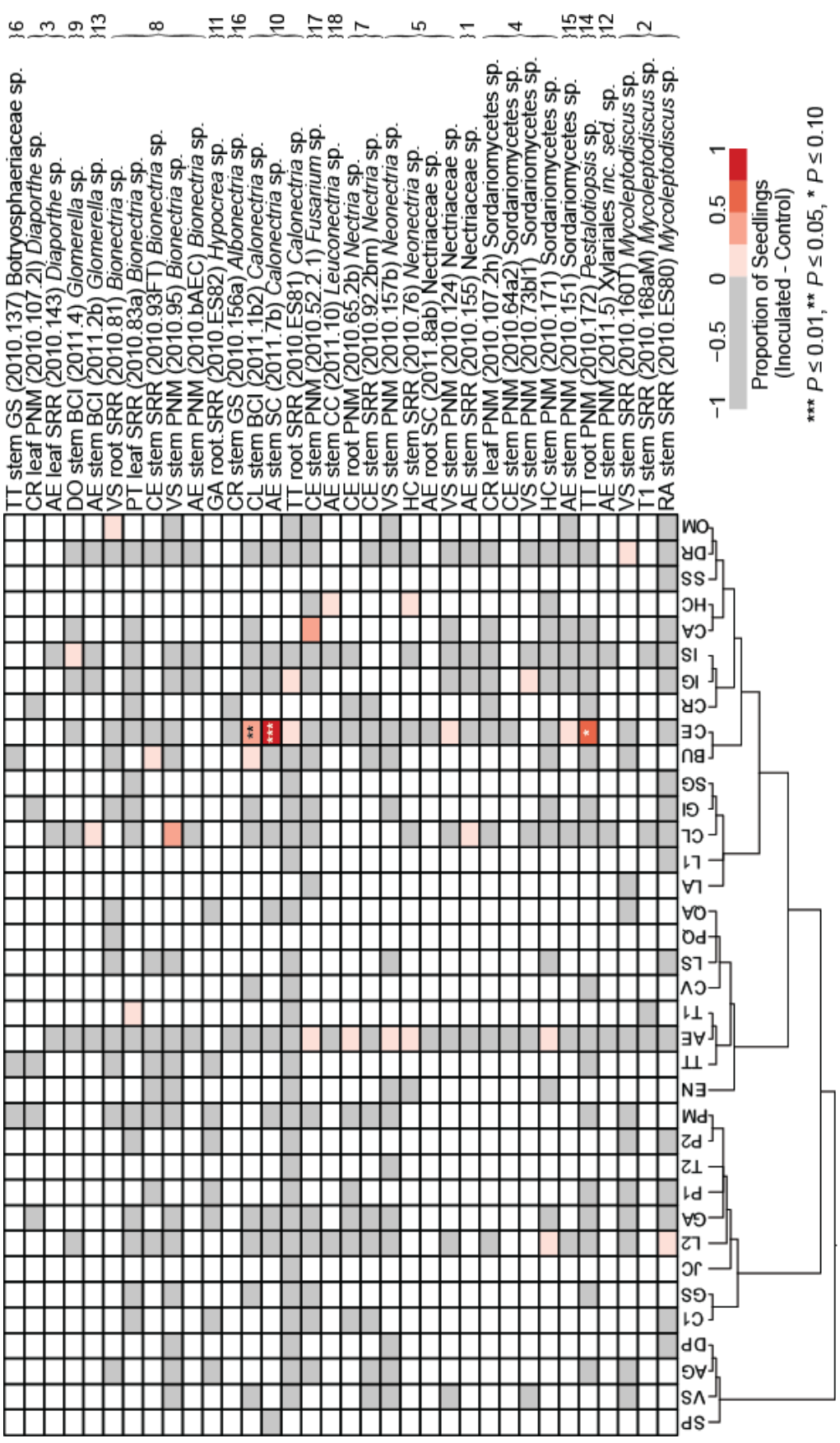
TT stem GS (2010.137) Botryosphaeriaceae sp.
CR leaf PNM (2010.107.2i) Diaporthe sp.
AE leaf SRR (2010.143) Diaporthe sp.
DO stem BCI (2011.4) Glomerella sp.
AE stem BCI (2011.2b) Glomerella sp.
VS root SRR (2010.81) Bionectria sp.
PT leaf SRR (2010.83a) Bionectria sp.
CE stem SRR (2010.93FT) Bionectria sp.
VS stem PNM (2010.95) Bionectria sp.
AE stem PNM (2010.bAEC) Bionectria sp.
GA root.SRR (2010.ES82) Hypocrea sp.
CR stem GS (2010.156a) Albonectria sp.
CL stem BCI (2011.1b2) Calonectria sp.
AE stem SC (2011.7b) Calonectria sp.
TT root SRR (2010.ES81) Calonectria sp.
CE stem PNM (2010.52.2.1) Fusarium sp.
AE stem CC (2011.10) Leuconectria sp.
CE root PNM (2010.65.2b) Nectria sp.
CE stem SRR (2010.92.2brn) Nectria sp.
VS stem PNM (2010.157b) Neonectria sp.
HC stem SRR (2010.76) Neonectria sp.
AE root SC (2011.8ab) Nectriaceae sp.
VS stem PNM (2010.124) Nectriaceae sp.
AE stem SRR (2010.155) Nectriaceae sp.
CR leaf PNM (2010.107.2h) Sordariomycetes sp.
CE stem PNM (2010.64a2) Sordariomycetes sp.
VS stem PNM (2010.73b1) Sordariomycetes sp.
HC stem PNM (2010.171) Sordariomycetes sp.
AE stem PNM (2010.151) Sordariomycetes sp.
TT root PNM (2010.172) Pestalotiopsis sp.
AE stem PNM (2011.5) Xylariales inc. sed. sp.
VS stem SRR (2010.160T) Mycoleptodiscus sp.
T1 stem SRR (2010.168aM) Mycoleptodiscus sp.
RA stem SRR (2010.ES80) Mycoleptodiscus sp.
6
3
9
13
8
11
16
10
17
18
7
5
1
4
15
14
12
2
SP VS AG DP C1 GS JC L2 GA P1 T2 P2 PM EN T1 AE TT CV LS PQ QA LA L1 CL GI SG BU CE CR IG IS CA HC SS DR OM
**
***
*
−1 −0.5 0 0.5 1
Proportion of Seedlings (Inoculated - Control)
*** P ≤ 0.01, ** P ≤ 0.05, * P ≤ 0.10

Fig. 4.7. Matrix depicting the number of times a fungal species was isolated from symptomatic seedlings of a given tree species. Along the vertical axis, the 21 host tree species are arranged based on their evolutionary relationships and the topology of the cladogram is based on a recently developed supertree (R2G220140601 tree and ages file; G. S. Gilbert, unpublished manuscript). Along the horizontal axis, the 29 fungal species are arranged according to their class (Sordariomycetes, Eurotiomycetes, and Dothideomycetes), order, family, and, when known, genus (see Tables 4.1 and 4.2 for additional taxonomic details for the trees and fungi, respectively). The estimated taxonomic placement of each species is listed (*inc. sed.* = *incertae sedis*) followed by the species identification number in parentheses. The fungal "species" with members that exhibited pathogenicity during the inoculation experiments are identified with a double dagger (‡) symbol. The values inside the cells indicate the number of times that particular fungal species was isolated from a seedling of that particular tree species. Empty cells indicate that that particular fungal species was not isolated from that particular tree species. The counts include all 93 fungal isolates from symptomatic seedlings and the ecological role of each isolate has not been determined.

NC
VS
L2
GA
RA
CV
SM
TT
AE
PT
T1
CL
BU
CE
C2
CR
HC
DO
DR
OC
OM
Chaetosphaeriaceae sp. (21)
Diaporthe sp. (22)
Glomerella sp. (3)
Glomerella sp. (9)
Bionectria sp. (13)
Hypocrea sp. (29)
Albonectria sp. (8)+
Calonectria sp. (11)
Fusarium sp. (25)
Gibberella sp. (10)+
Leuconectria sp. (26)+
Nectria sp. (20)
Nectriaceae sp. (7)
Pestalotiopsis sp. (28)
Pestalotiopsis sp. (1)
Xylariaceae sp. (5)
Sordariomycetes (inc. sed.) sp. (16)
Sordariomycetes sp. (18)+
Sordariomycetes sp. (17)
Myconeptodiscus sp. (12)
Emericella sp. (4)+
Talaromyces sp. (24)
Boltryosphaeria sp. (15)
Myconeptodiscus sp. (2)+
Myrmecridium sp. (27)
Myrmecridium sp. (14)
Myrmecridium sp. (19)
Myrmecridium sp. (6)
Myrmecridium sp. (23)

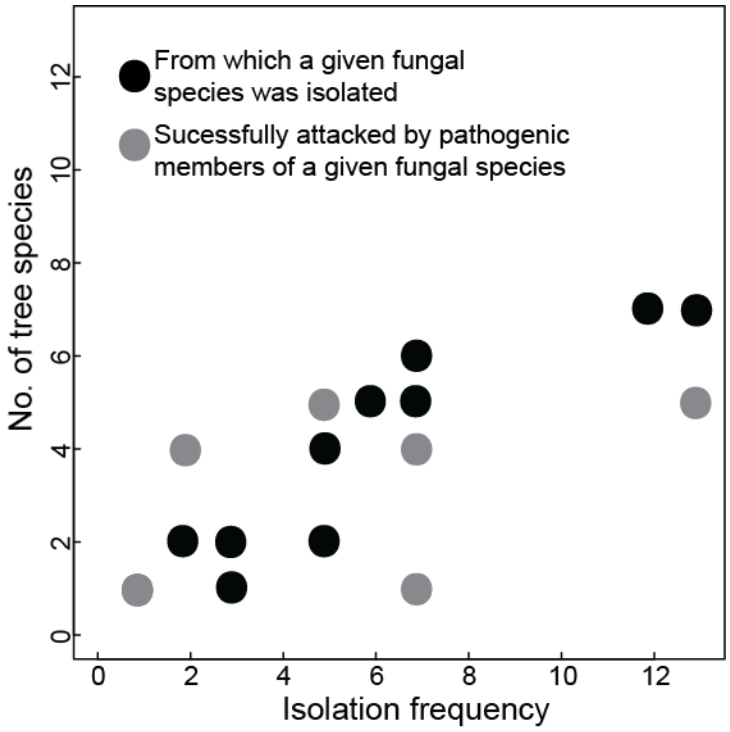

Fig. 4.8. The relationship between the frequency with which a given fungal species was isolated and (i) the number of tree species from which a given fungal species was isolated (black circles) and (ii) the number of tree species successfully attacked by a given fungal species (gray circles). Fungal species that were isolated from multiple tree species were isolated more frequently (Spearman's rank correlation rho = 0.925, $P < 0.001$), suggesting that host-generalized fungi may be more common. Similarly, fungal species with pathogenic members capable of generating disease in more than one tree species tended to be isolated more frequently (one-tailed Spearman's rank correlation rho = 0.424, $P = 0.201$).

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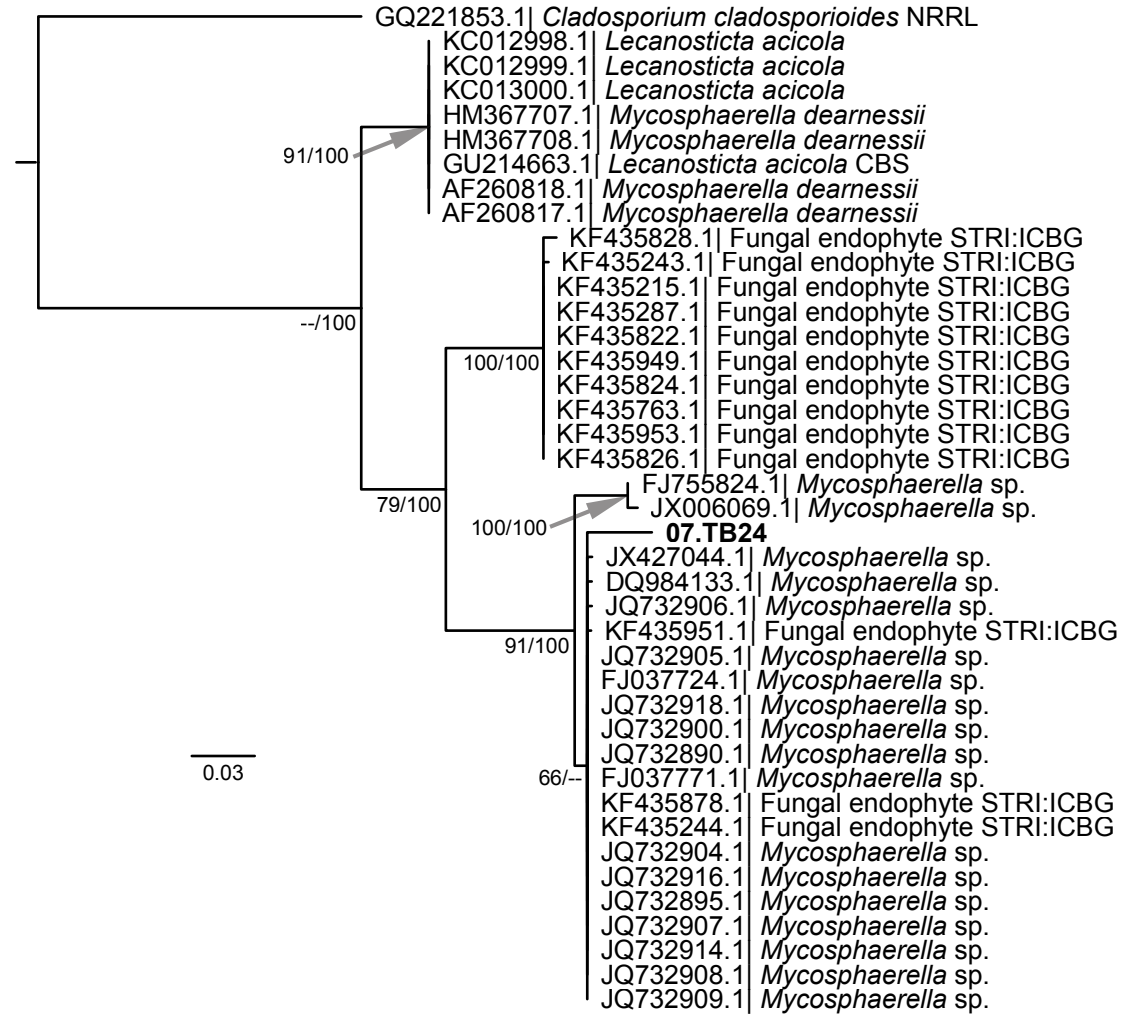
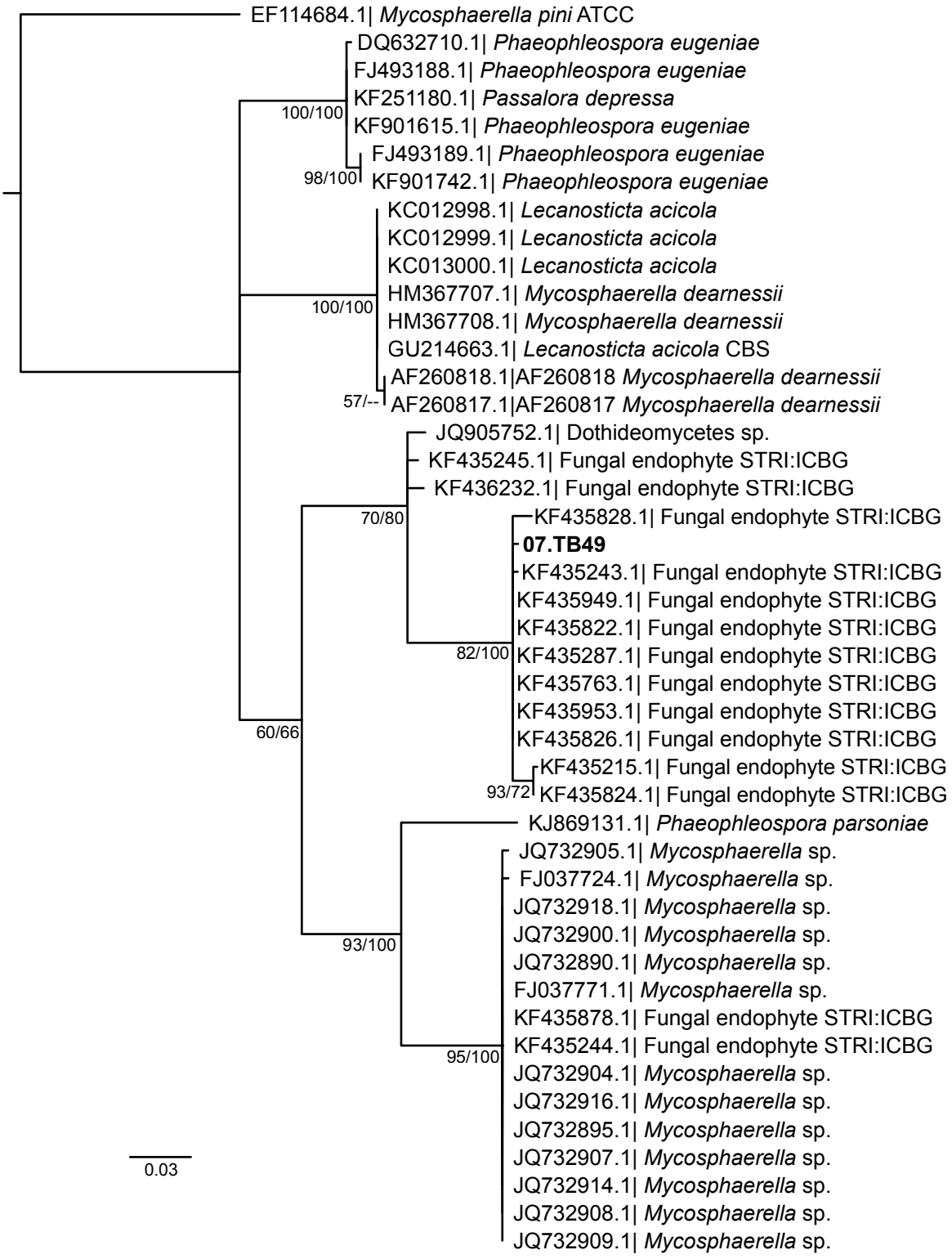
APPENDIX

PHYLOGENETIC TREES GENERATED TO ESTIMATE TAXONOMIC PLACEMENT FOR FUNGI ISOLATED FROM SEEDLINGS WITH PATHOGEN-CAUSED DAMAGE

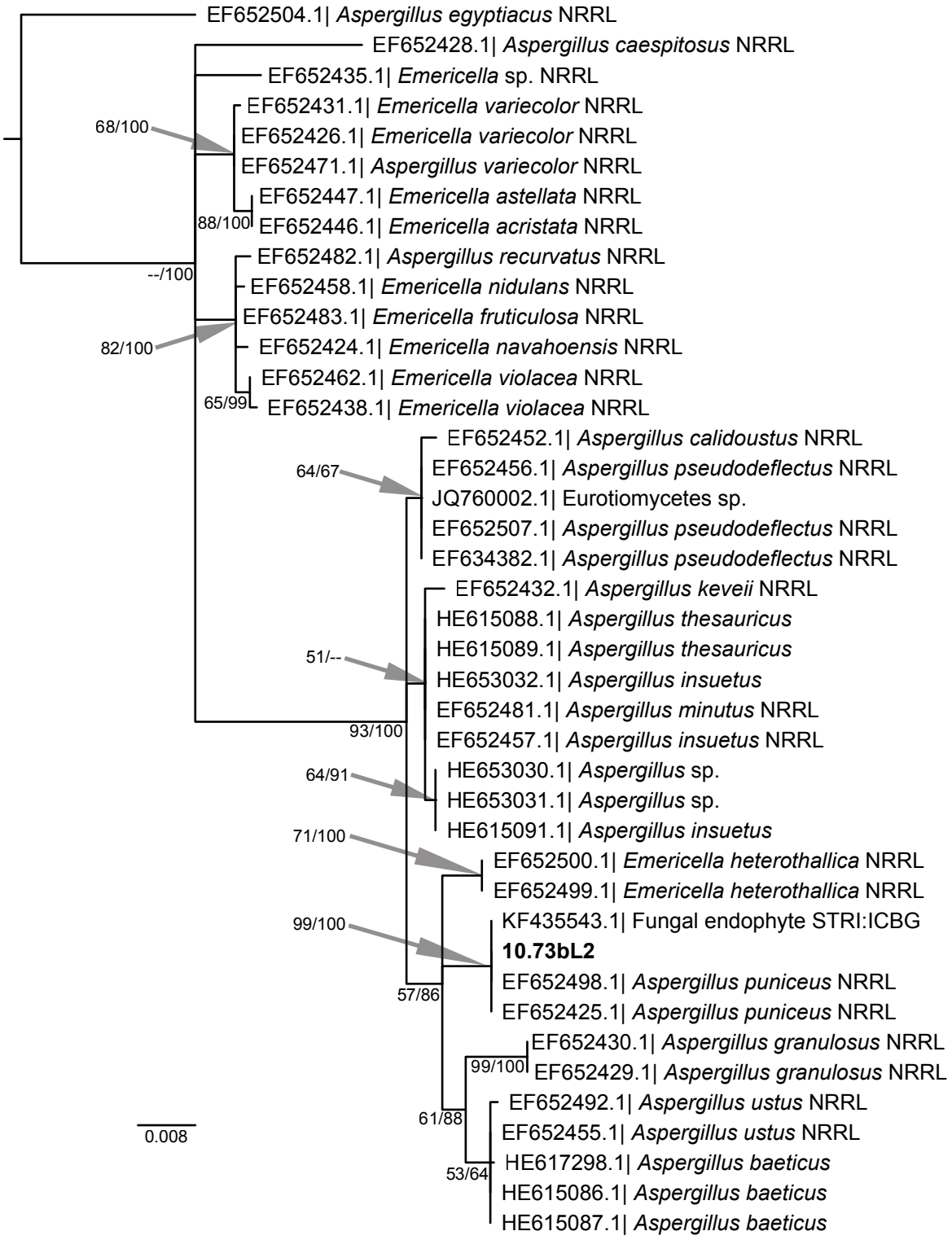
Appendix. Thirty-three majority rule consensus trees (A-AG), based on maximum likelihood (ML) analyses of ITS rDNA data, generated to estimate taxonomic placement for fungi isolated from seedlings with pathogen-caused damage that were collected from forests spanning a rainfall gradient (90 isolates from 75 seedlings) and from a shadehouse (3 isolates from 2 seedlings) in Panama. The fungal isolates are indicated by bolded codes. For each tree, support for each clade is presented as ML bootstrap values (≥50%, before the slash) and Bayesian posterior probabilities (≥50%, after the slash). Outgroups were selected by reviewing published literature. The taxonomic conclusions based on these trees are summarized in Fig. 3.3 and reported in Table 3.4. Accession numbers are listed for sequences obtained from GenBank. In some cases, the culture collection is indicated (e.g., NRRL, CBS, and STRI:ICBG). The scale is in substitutions per site.


GQ221853.1| Cladosporium cladosporioides NRRL
KC012998.1| Lecanosticta acicola
KC012999.1| Lecanosticta acicola
KC013000.1| Lecanosticta acicola
HM367707.1| Mycosphaerella dearnessii
HM367708.1| Mycosphaerella dearnessii
GU214663.1| Lecanosticta acicola CBS
AF260818.1| Mycosphaerella dearnessii
AF260817.1| Mycosphaerella dearnessii
KF435828.1| Fungal endophyte STRI:ICBG
KF435243.1| Fungal endophyte STRI:ICBG
KF435215.1| Fungal endophyte STRI:ICBG
KF435287.1| Fungal endophyte STRI:ICBG
KF435822.1| Fungal endophyte STRI:ICBG
KF435949.1| Fungal endophyte STRI:ICBG
KF435824.1| Fungal endophyte STRI:ICBG
KF435763.1| Fungal endophyte STRI:ICBG
KF435953.1| Fungal endophyte STRI:ICBG
KF435826.1| Fungal endophyte STRI:ICBG
FJ755824.1| Mycosphaerella sp.
JX006069.1| Mycosphaerella sp.
07.TB24
JX427044.1| Mycosphaerella sp.
DQ984133.1| Mycosphaerella sp.
JQ732906.1| Mycosphaerella sp.
KF435951.1| Fungal endophyte STRI:ICBG
JQ732905.1| Mycosphaerella sp.
FJ037724.1| Mycosphaerella sp.
JQ732918.1| Mycosphaerella sp.
JQ732900.1| Mycosphaerella sp.
JQ732890.1| Mycosphaerella sp.
FJ037771.1| Mycosphaerella sp.
KF435878.1| Fungal endophyte STRI:ICBG
KF435244.1| Fungal endophyte STRI:ICBG
JQ732904.1| Mycosphaerella sp.
JQ732916.1| Mycosphaerella sp.
JQ732895.1| Mycosphaerella sp.
JQ732907.1| Mycosphaerella sp.
JQ732914.1| Mycosphaerella sp.
JQ732908.1| Mycosphaerella sp.
JQ732909.1| Mycosphaerella sp.
91/100
--/100
100/100
79/100
100/100
91/100
66/--
0.03

A


EF114684.1| *Mycosphaerella pini* ATCC
DQ632710.1| *Phaeophleospora eugeniae*
FJ493188.1| *Phaeophleospora eugeniae*
KF251180.1| *Passalora depressa*
100/100
KF901615.1| *Phaeophleospora eugeniae*
FJ493189.1| *Phaeophleospora eugeniae*
98/100
KF901742.1| *Phaeophleospora eugeniae*
KC012998.1| *Lecanosticta acicola*
KC012999.1| *Lecanosticta acicola*
KC013000.1| *Lecanosticta acicola*
HM367707.1| *Mycosphaerella dearnessii*
100/100
HM367708.1| *Mycosphaerella dearnessii*
GU214663.1| *Lecanosticta acicola* CBS
AF260818.1|AF260818 *Mycosphaerella dearnessii*
57/--
AF260817.1|AF260817 *Mycosphaerella dearnessii*
JQ905752.1| Dothideomycetes sp.
KF435245.1| Fungal endophyte STRI:ICBG
KF436232.1| Fungal endophyte STRI:ICBG
70/80
KF435828.1| Fungal endophyte STRI:ICBG
07.TB49
KF435243.1| Fungal endophyte STRI:ICBG
KF435949.1| Fungal endophyte STRI:ICBG
KF435822.1| Fungal endophyte STRI:ICBG
82/100
KF435287.1| Fungal endophyte STRI:ICBG
KF435763.1| Fungal endophyte STRI:ICBG
KF435953.1| Fungal endophyte STRI:ICBG
60/66
KF435826.1| Fungal endophyte STRI:ICBG
KF435215.1| Fungal endophyte STRI:ICBG
93/72
KF435824.1| Fungal endophyte STRI:ICBG
KJ869131.1| *Phaeophleospora parsoniae*
JQ732905.1| *Mycosphaerella* sp.
FJ037724.1| *Mycosphaerella* sp.
JQ732918.1| *Mycosphaerella* sp.
JQ732900.1| *Mycosphaerella* sp.
93/100
JQ732890.1| *Mycosphaerella* sp.
FJ037771.1| *Mycosphaerella* sp.
KF435878.1| Fungal endophyte STRI:ICBG
KF435244.1| Fungal endophyte STRI:ICBG
95/100
JQ732904.1| *Mycosphaerella* sp.
JQ732916.1| *Mycosphaerella* sp.
JQ732895.1| *Mycosphaerella* sp.
JQ732907.1| *Mycosphaerella* sp.
JQ732914.1| *Mycosphaerella* sp.
JQ732908.1| *Mycosphaerella* sp.
JQ732909.1| *Mycosphaerella* sp.
0.03

B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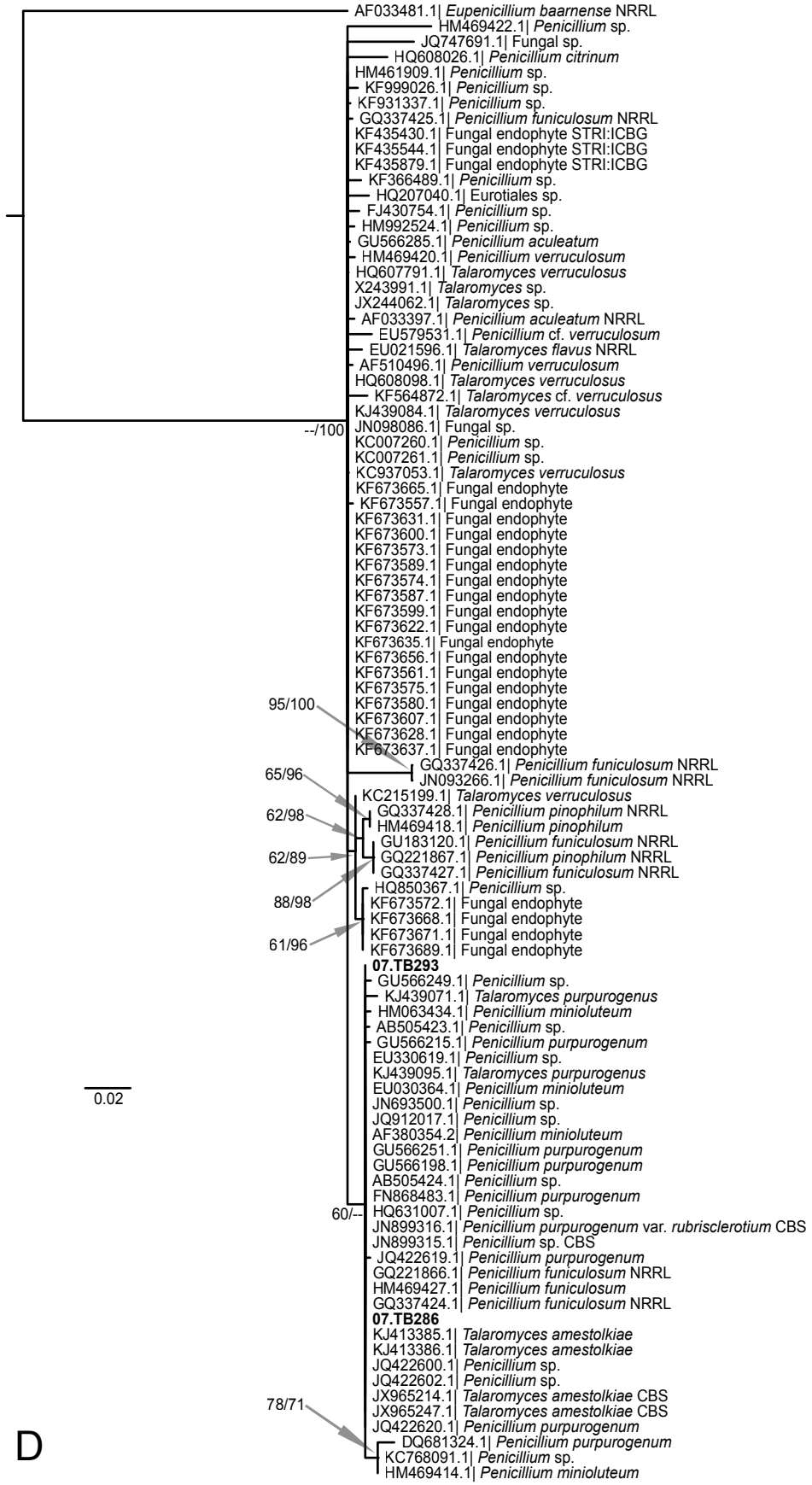
EF652428.1| Aspergillus caespitosus NRRL
EF652435.1| Emericella sp. NRRL
EF652431.1| Emericella variecolor NRRL
EF652426.1| Emericella variecolor NRRL
EF652471.1| Aspergillus variecolor NRRL
EF652447.1| Emericella astellata NRRL
EF652446.1| Emericella acristata NRRL
EF652482.1| Aspergillus recurvatus NRRL
EF652458.1| Emericella nidulans NRRL
EF652483.1| Emericella fruticulosa NRRL
EF652424.1| Emericella navahoensis NRRL
EF652462.1| Emericella violacea NRRL
EF652438.1| Emericella violacea NRRL
EF652452.1| Aspergillus calidoustus NRRL
EF652456.1| Aspergillus pseudodeflectus NRRL
JQ760002.1| Eurotiomycetes sp.
EF652507.1| Aspergillus pseudodeflectus NRRL
EF634382.1| Aspergillus pseudodeflectus NRRL
EF652432.1| Aspergillus keveii NRRL
HE615088.1| Aspergillus thesauricus
HE615089.1| Aspergillus thesauricus
HE653032.1| Aspergillus insuetus
EF652481.1| Aspergillus minutus NRRL
EF652457.1| Aspergillus insuetus NRRL
HE653030.1| Aspergillus sp.
HE653031.1| Aspergillus sp.
HE615091.1| Aspergillus insuetus
EF652500.1| Emericella heterothallica NRRL
EF652499.1| Emericella heterothallica NRRL
KF435543.1| Fungal endophyte STRI:ICBG
10.73bL2
EF652498.1| Aspergillus puniceus NRRL
EF652425.1| Aspergillus puniceus NRRL
EF652430.1| Aspergillus granulosus NRRL
EF652429.1| Aspergillus granulosus NRRL
EF652492.1| Aspergillus ustus NRRL
EF652455.1| Aspergillus ustus NRRL
HE617298.1| Aspergillus baeticus
HE615086.1| Aspergillus baeticus
HE615087.1| Aspergillus baeticus
68/100
88/100
--/100
82/100
65/99
64/67
51/--
93/100
64/91
71/100
99/100
57/86
99/100
61/88
53/64
0.008

C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HM461909.1| Penicillium sp.
KF999026.1| Penicillium sp.
KF931337.1| Penicillium sp.
GQ337425.1| Penicillium funiculosum NRRL
KF435430.1| Fungal endophyte STRI:ICBG
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KF366489.1| Penicillium sp.
HQ207040.1| Eurotiales sp.
FJ430754.1| Penicillium sp.
HM992524.1| Penicillium sp.
GU566285.1| Penicillium aculeatum
HM469420.1| Penicillium verruculosum
HQ607791.1| Talaromyces verruculosus
X243991.1| Talaromyces sp.
JX244062.1| Talaromyces sp.
AF033397.1| Penicillium aculeatum NRRL
EU579531.1| Penicillium cf. verruculosum
EU021596.1| Talaromyces flavus NRRL
AF510496.1| Penicillium verruculosum
HQ608098.1| Talaromyces verruculosus
KF564872.1| Talaromyces cf. verruculosus
KJ439084.1| Talaromyces verruculosus
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JN098086.1| Fungal sp.
KC007260.1| Penicillium sp.
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KC937053.1| Talaromyces verruculosus
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KF673573.1| Fungal endophyte
KF673589.1| Fungal endophyte
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KF673587.1| Fungal endophyte
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KF673635.1| Fungal endophyte
KF673656.1| Fungal endophyte
KF673561.1| Fungal endophyte
KF673575.1| Fungal endophyte
95/100
KF673580.1| Fungal endophyte
KF673607.1| Fungal endophyte
KF673628.1| Fungal endophyte
KF673637.1| Fungal endophyte
GQ337426.1| Penicillium funiculosum NRRL
JN093266.1| Penicillium funiculosum NRRL
65/96
KC215199.1| Talaromyces verruculosus
62/98
GQ337428.1| Penicillium pinophilum NRRL
HM469418.1| Penicillium pinophilum
GU183120.1| Penicillium funiculosum NRRL
62/89
GQ221867.1| Penicillium pinophilum NRRL
GQ337427.1| Penicillium funiculosum NRRL
HQ850367.1| Penicillium sp.
88/98
KF673572.1| Fungal endophyte
KF673668.1| Fungal endophyte
KF673671.1| Fungal endophyte
61/96
KF673689.1| Fungal endophyte
07.TB293
GU566249.1| Penicillium sp.
KJ439071.1| Talaromyces purpurogenus
HM063434.1| Penicillium minioluteum
AB505423.1| Penicillium sp.
GU566215.1| Penicillium purpurogenum
EU330619.1| Penicillium sp.
KJ439095.1| Talaromyces purpurogenus
EU030364.1| Penicillium minioluteum
JN693500.1| Penicillium sp.
JQ912017.1| Penicillium sp.
AF380354.2| Penicillium minioluteum
GU566251.1| Penicillium purpurogenum
GU566198.1| Penicillium purpurogenum
AB505424.1| Penicillium sp.
FN868483.1| Penicillium purpurogenum
60/--
HQ631007.1| Penicillium sp.
JN899316.1| Penicillium purpurogenum var. rubrisclerotium CBS
JN899315.1| Penicillium sp. CBS
JQ422619.1| Penicillium purpurogenum
GQ221866.1| Penicillium funiculosum NRRL
HM469427.1| Penicillium funiculosum
GQ337424.1| Penicillium funiculosum NRRL
07.TB286
KJ413385.1| Talaromyces amestolkiae
KJ413386.1| Talaromyces amestolkiae
JQ422600.1| Penicillium sp.
JQ422602.1| Penicillium sp.
JX965214.1| Talaromyces amestolkiae CBS
78/71
JX965247.1| Talaromyces amestolkiae CBS
JQ422620.1| Penicillium purpurogenum
DQ681324.1| Penicillium purpurogenum
KC768091.1| Penicillium sp.
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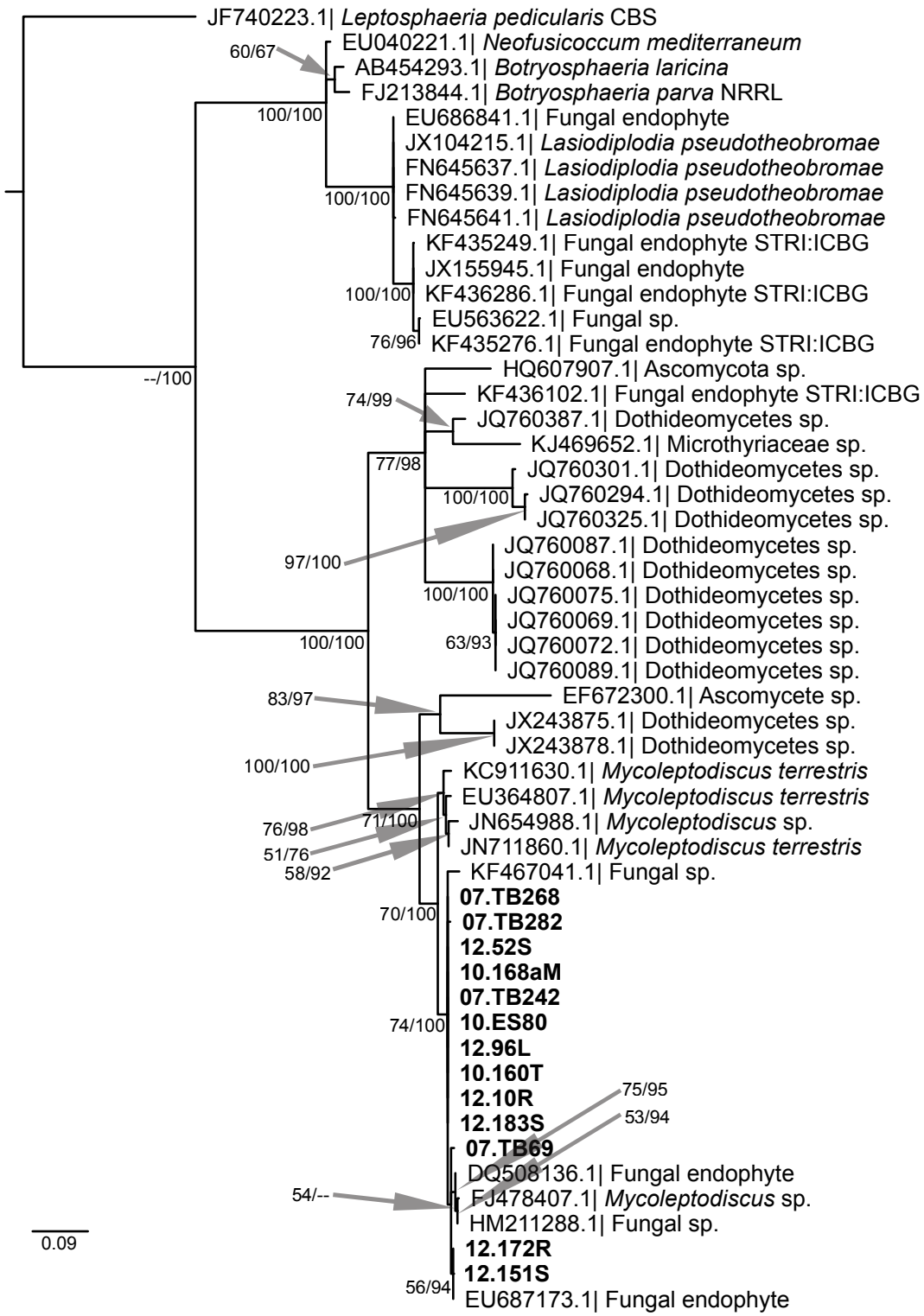
0.02
D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EU040221.1| Neofusicoccum mediterraneum
AB454293.1| Botryosphaeria laricina
FJ213844.1| Botryosphaeria parva NRRL
EU686841.1| Fungal endophyte
JX104215.1| Lasiodiplodia pseudotheobromae
FN645637.1| Lasiodiplodia pseudotheobromae
FN645639.1| Lasiodiplodia pseudotheobromae
FN645641.1| Lasiodiplodia pseudotheobromae
KF435249.1| Fungal endophyte STRI:ICBG
JX155945.1| Fungal endophyte
KF436286.1| Fungal endophyte STRI:ICBG
EU563622.1| Fungal sp.
KF435276.1| Fungal endophyte STRI:ICBG
HQ607907.1| Ascomycota sp.
KF436102.1| Fungal endophyte STRI:ICBG
JQ760387.1| Dothideomycetes sp.
KJ469652.1| Microthyriaceae sp.
JQ760301.1| Dothideomycetes sp.
JQ760294.1| Dothideomycetes sp.
JQ760325.1| Dothideomycetes sp.
JQ760087.1| Dothideomycetes sp.
JQ760068.1| Dothideomycetes sp.
JQ760075.1| Dothideomycetes sp.
JQ760069.1| Dothideomycetes sp.
JQ760072.1| Dothideomycetes sp.
JQ760089.1| Dothideomycetes sp.
EF672300.1| Ascomycete sp.
JX243875.1| Dothideomycetes sp.
JX243878.1| Dothideomycetes sp.
KC911630.1| Mycoleptodiscus terrestris
EU364807.1| Mycoleptodiscus terrestris
JN654988.1| Mycoleptodiscus sp.
JN711860.1| Mycoleptodiscus terrestris
KF467041.1| Fungal sp.
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07.TB282
12.52S
10.168aM
07.TB242
10.ES80
12.96L
10.160T
12.10R
12.183S
07.TB69
DQ508136.1| Fungal endophyte
FJ478407.1| Mycoleptodiscus sp.
HM211288.1| Fungal sp.
12.172R
12.151S
EU687173.1| Fungal endophyte
60/67
100/100
100/100
100/100
76/96
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74/99
77/98
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97/100
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63/93
83/97
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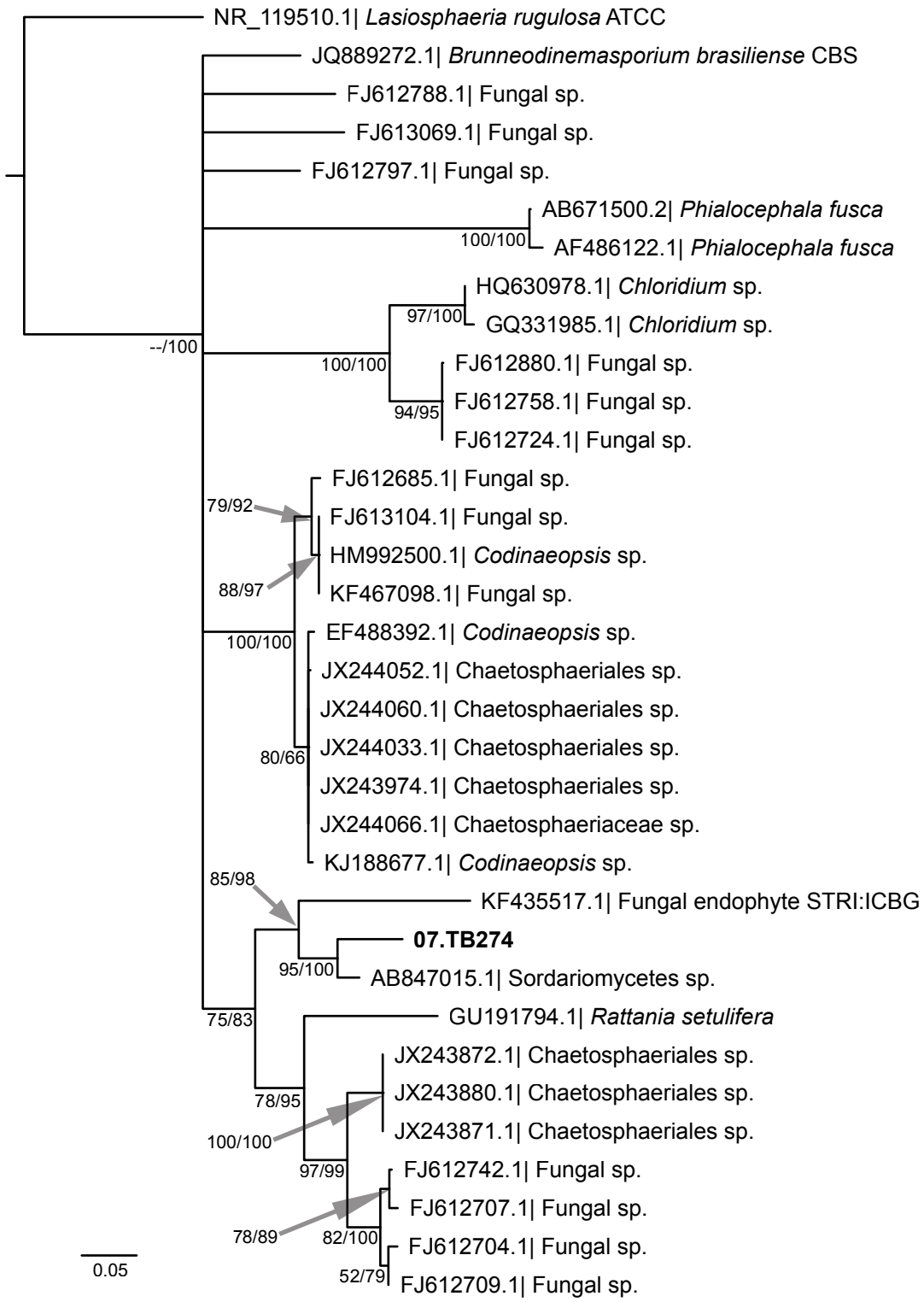
75/95
53/94
54/--
56/94
0.09

E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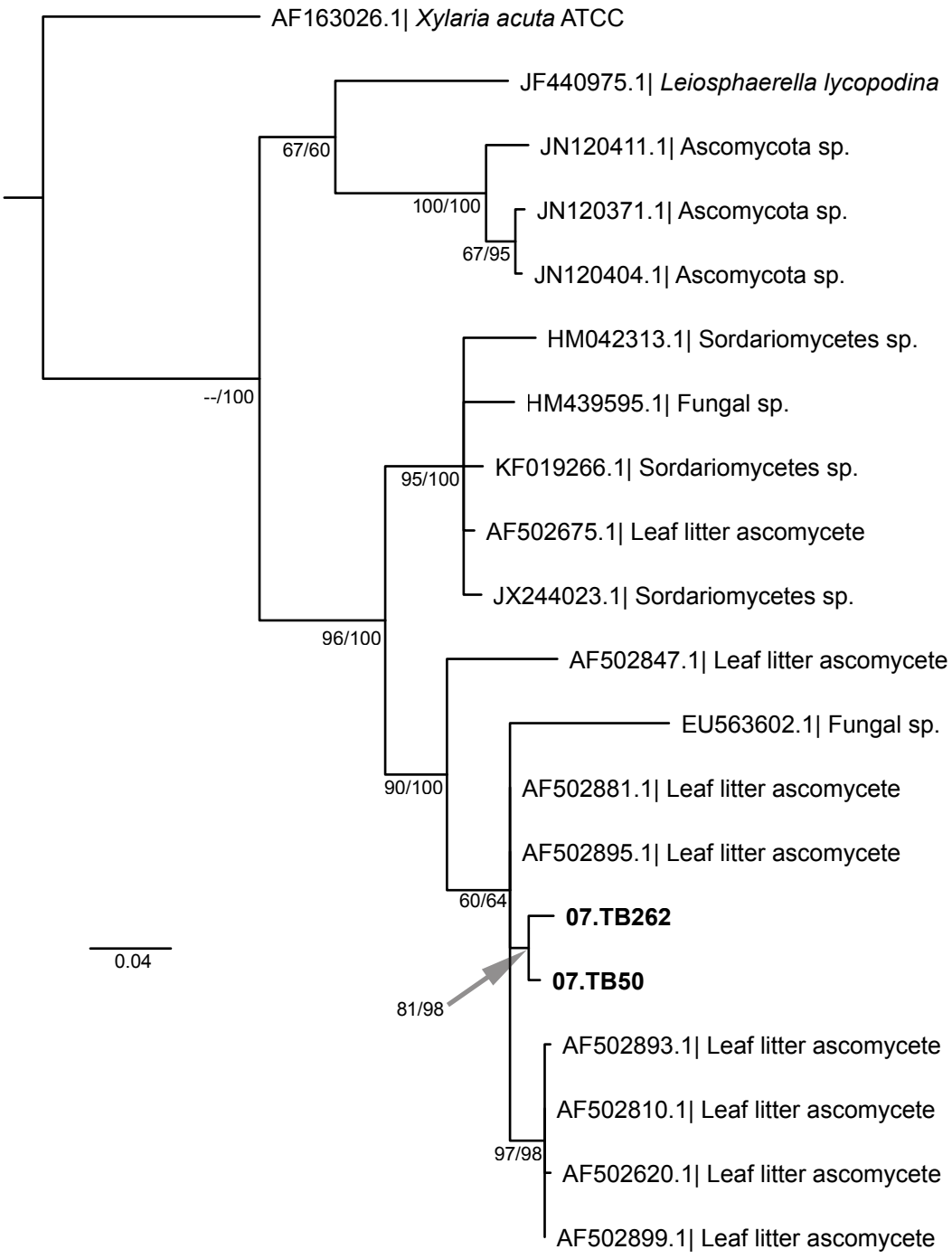
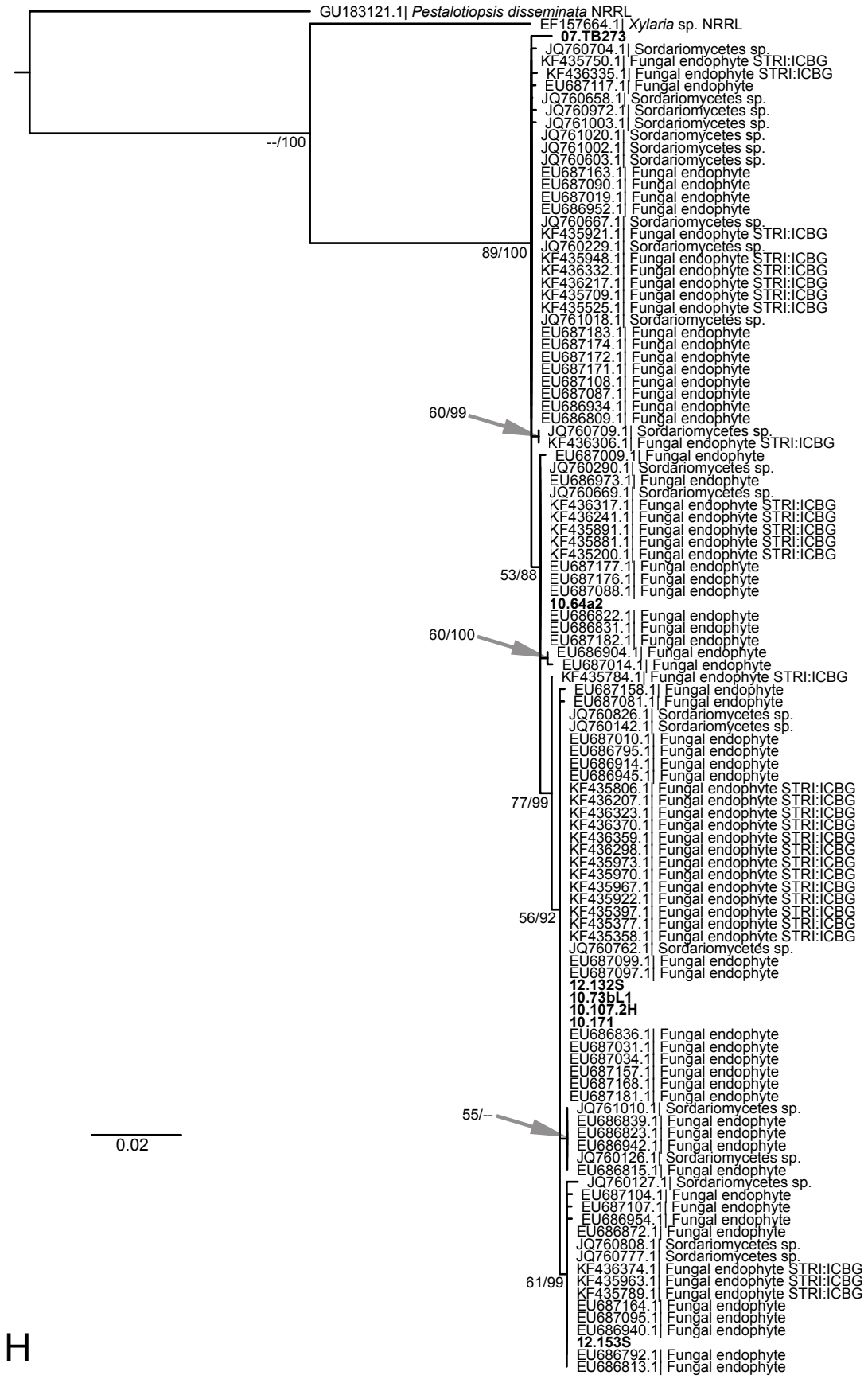
FJ613069.1| Fungal sp.
FJ612797.1| Fungal sp.
AB671500.2| Phialocephala fusca
100/100
AF486122.1| Phialocephala fusca
HQ630978.1| Chloridium sp.
97/100
GQ331985.1| Chloridium sp.
--/100
100/100
FJ612880.1| Fungal sp.
FJ612758.1| Fungal sp.
94/95
FJ612724.1| Fungal sp.
FJ612685.1| Fungal sp.
79/92
FJ613104.1| Fungal sp.
HM992500.1| Codinaeopsis sp.
88/97
KF467098.1| Fungal sp.
100/100
EF488392.1| Codinaeopsis sp.
JX244052.1| Chaetosphaeriales sp.
JX244060.1| Chaetosphaeriales sp.
80/66
JX244033.1| Chaetosphaeriales sp.
JX243974.1| Chaetosphaeriales sp.
JX244066.1| Chaetosphaeriaceae sp.
KJ188677.1| Codinaeopsis sp.
85/98
KF435517.1| Fungal endophyte STRI:ICBG
07.TB274
95/100
AB847015.1| Sordariomycetes sp.
75/83
GU191794.1| Rattania setulifera
JX243872.1| Chaetosphaeriales sp.
78/95
JX243880.1| Chaetosphaeriales sp.
100/100
JX243871.1| Chaetosphaeriales sp.
97/99
FJ612742.1| Fungal sp.
FJ612707.1| Fungal sp.
78/89
82/100
FJ612704.1| Fungal sp.
52/79
FJ612709.1| Fungal sp.
0.05

F


AF163026.1| Xylaria acuta ATCC
JF440975.1| Leiosphaerella lycopodina
JN120411.1| Ascomycota sp.
JN120371.1| Ascomycota sp.
JN120404.1| Ascomycota sp.
HM042313.1| Sordariomycetes sp.
HM439595.1| Fungal sp.
KF019266.1| Sordariomycetes sp.
AF502675.1| Leaf litter ascomycete
JX244023.1| Sordariomycetes sp.
AF502847.1| Leaf litter ascomycete
EU563602.1| Fungal sp.
AF502881.1| Leaf litter ascomycete
AF502895.1| Leaf litter ascomycete
07.TB262
07.TB50
AF502893.1| Leaf litter ascomycete
AF502810.1| Leaf litter ascomycete
AF502620.1| Leaf litter ascomycete
AF502899.1| Leaf litter ascomycete
67/60
100/100
67/95
--/100
95/100
96/100
90/100
60/64
81/98
97/98
0.04

G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JQ760658.1| Sordariomycetes sp.
JQ760972.1| Sordariomycetes sp.
JQ761003.1| Sordariomycetes sp.
JQ761020.1| Sordariomycetes sp.
JQ761002.1| Sordariomycetes sp.
JQ760603.1| Sordariomycetes sp.
EU687163.1| Fungal endophyte
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JQ760667.1| Sordariomycetes sp.
KF435921.1| Fungal endophyte STRI:ICBG
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KF435948.1| Fungal endophyte STRI:ICBG
KF436332.1| Fungal endophyte STRI:ICBG
KF436217.1| Fungal endophyte STRI:ICBG
KF435709.1| Fungal endophyte STRI:ICBG
KF435525.1| Fungal endophyte STRI:ICBG
JQ761018.1| Sordariomycetes sp.
EU687183.1| Fungal endophyte
EU687174.1| Fungal endophyte
EU687172.1| Fungal endophyte
EU687171.1| Fungal endophyte
EU687108.1| Fungal endophyte
EU687087.1| Fungal endophyte
EU686934.1| Fungal endophyte
EU686809.1| Fungal endophyte
JQ760709.1| Sordariomycetes sp.
KF436306.1| Fungal endophyte STRI:ICBG
EU687009.1| Fungal endophyte
JQ760290.1| Sordariomycetes sp.
EU686973.1| Fungal endophyte
JQ760669.1| Sordariomycetes sp.
KF436317.1| Fungal endophyte STRI:ICBG
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EU686904.1| Fungal endophyte
EU687014.1| Fungal endophyte
KF435784.1| Fungal endophyte STRI:ICBG
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EU687081.1| Fungal endophyte
JQ760826.1| Sordariomycetes sp.
JQ760142.1| Sordariomycetes sp.
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EU686795.1| Fungal endophyte
EU686914.1| Fungal endophyte
EU686945.1| Fungal endophyte
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KF435358.1| Fungal endophyte STRI:ICBG
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12.132S
10.73bL1
10.107.2H
10.171
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EU687168.1| Fungal endophyte
EU687181.1| Fungal endophyte
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JQ760127.1| Sordariomycetes sp.
EU687104.1| Fungal endophyte
EU687107.1| Fungal endophyte
EU686954.1| Fungal endophyte
EU686872.1| Fungal endophyte
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JQ760777.1| Sordariomycetes sp.
KF436374.1| Fungal endophyte STRI:ICBG
KF435963.1| Fungal endophyte STRI:ICBG
KF435789.1| Fungal endophyte STRI:ICBG
EU687164.1| Fungal endophyte
EU687095.1| Fungal endophyte
EU686940.1| Fungal endophyte
12.153S
EU686792.1| Fungal endophyte
EU686813.1| Fungal endophyte
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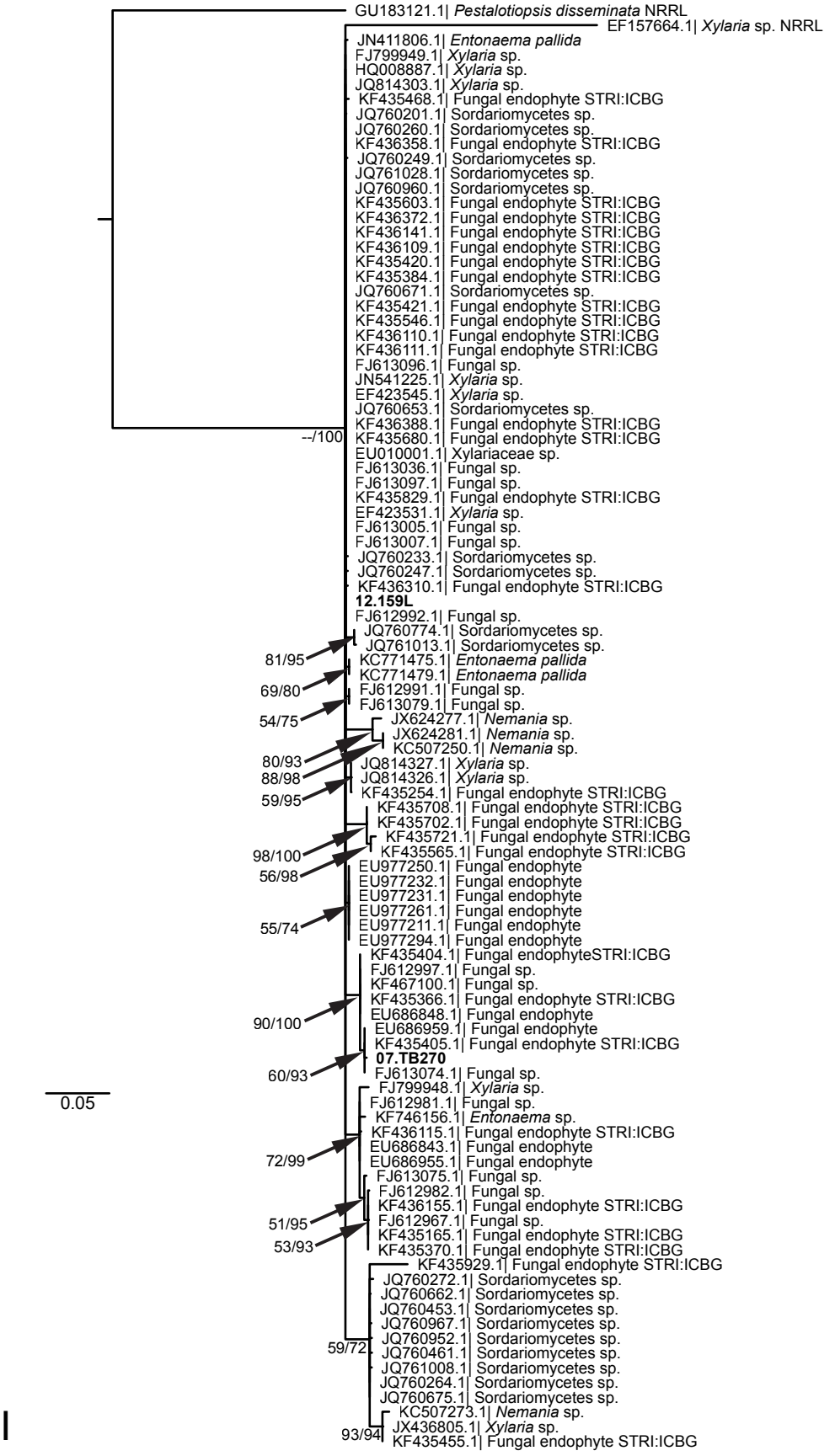
89/100
60/99
53/88
60/100
77/99
56/92
55/--
61/99
0.02

H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HQ008887.1| Xylaria sp.
JQ814303.1| Xylaria sp.
KF435468.1| Fungal endophyte STRI:ICBG
JQ760201.1| Sordariomycetes sp.
JQ760260.1| Sordariomycetes sp.
KF436358.1| Fungal endophyte STRI:ICBG
JQ760249.1| Sordariomycetes sp.
JQ761028.1| Sordariomycetes sp.
JQ760960.1| Sordariomycetes sp.
KF435603.1| Fungal endophyte STRI:ICBG
KF436372.1| Fungal endophyte STRI:ICBG
KF436141.1| Fungal endophyte STRI:ICBG
KF436109.1| Fungal endophyte STRI:ICBG
KF435420.1| Fungal endophyte STRI:ICBG
KF435384.1| Fungal endophyte STRI:ICBG
JQ760671.1| Sordariomycetes sp.
KF435421.1| Fungal endophyte STRI:ICBG
KF435546.1| Fungal endophyte STRI:ICBG
KF436110.1| Fungal endophyte STRI:ICBG
KF436111.1| Fungal endophyte STRI:ICBG
FJ613096.1| Fungal sp.
JN541225.1| Xylaria sp.
EF423545.1| Xylaria sp.
JQ760653.1| Sordariomycetes sp.
KF436388.1| Fungal endophyte STRI:ICBG
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KF435680.1| Fungal endophyte STRI:ICBG
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KF435829.1| Fungal endophyte STRI:ICBG
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KF436310.1| Fungal endophyte STRI:ICBG
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JQ760774.1| Sordariomycetes sp.
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81/95
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KC771479.1| Entonaema pallida
69/80
FJ612991.1| Fungal sp.
FJ613079.1| Fungal sp.
54/75
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JX624281.1| Nemania sp.
KC507250.1| Nemania sp.
80/93
JQ814327.1| Xylaria sp.
88/98
JQ814326.1| Xylaria sp.
59/95
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KF435708.1| Fungal endophyte STRI:ICBG
KF435702.1| Fungal endophyte STRI:ICBG
KF435721.1| Fungal endophyte STRI:ICBG
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56/98
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EU977261.1| Fungal endophyte
55/74
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EU977294.1| Fungal endophyte
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FJ612997.1| Fungal sp.
KF467100.1| Fungal sp.
KF435366.1| Fungal endophyte STRI:ICBG
90/100
EU686848.1| Fungal endophyte
EU686959.1| Fungal endophyte
KF435405.1| Fungal endophyte STRI:ICBG
07.TB270
60/93
FJ613074.1| Fungal sp.
FJ799948.1| Xylaria sp.
FJ612981.1| Fungal sp.
KF746156.1| Entonaema sp.
KF436115.1| Fungal endophyte STRI:ICBG
72/99
EU686843.1| Fungal endophyte
EU686955.1| Fungal endophyte
FJ613075.1| Fungal sp.
FJ612982.1| Fungal sp.
KF436155.1| Fungal endophyte STRI:ICBG
51/95
FJ612967.1| Fungal sp.
53/93
KF435165.1| Fungal endophyte STRI:ICBG
KF435370.1| Fungal endophyte STRI:ICBG
KF435929.1| Fungal endophyte STRI:ICBG
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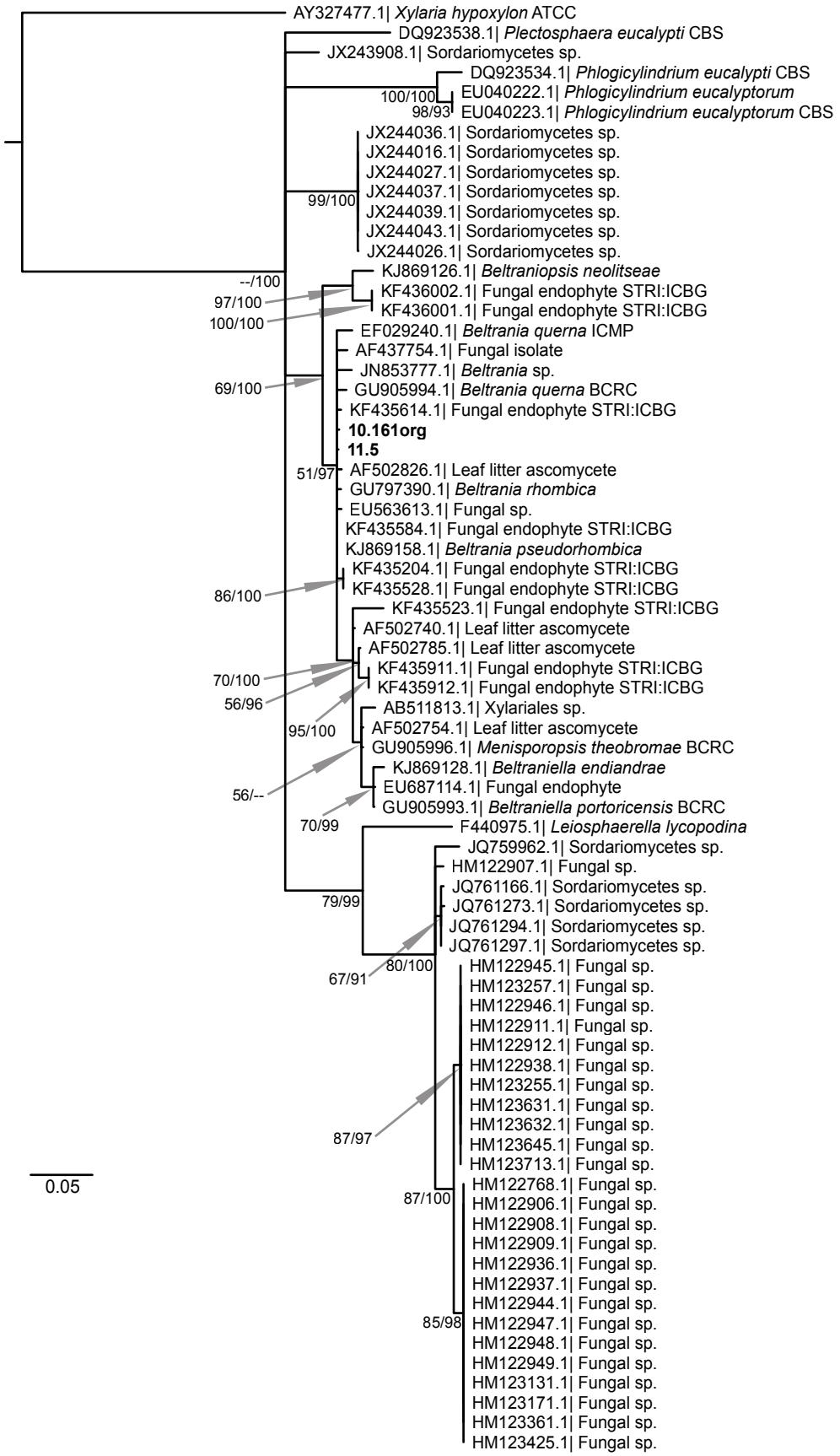
JQ760662.1| Sordariomycetes sp.
JQ760453.1| Sordariomycetes sp.
JQ760967.1| Sordariomycetes sp.
59/72
JQ760952.1| Sordariomycetes sp.
JQ760461.1| Sordariomycetes sp.
JQ761008.1| Sordariomycetes sp.
JQ760264.1| Sordariomycetes sp.
JQ760675.1| Sordariomycetes sp.
KC507273.1| Nemania sp.
93/94
JX436805.1| Xylaria sp.
KF435455.1| Fungal endophyte STRI:ICBG
0.05


KF144886.1| *Arthrinium arundinis* CBS
EU552155.1| *Sarcostroma bisetulatum* CBS
95/99
EU687040.1| Fungal endophyte
100/100
EU687178.1| Fungal endophyte
GQ328855.1| *Nigrospora oryzae* NRRL
GQ428201.1| *Nigrospora oryzae* NRRL
--/100
JQ760343.1| Sordariomycetes sp.
84/100
GQ221861.1| *Nigrospora oryzae* NRRL
95/100
GQ221860.1| *Nigrospora oryzae* NRRL
EU687125.1| Fungal endophyte
89/100
JQ760355.1| Sordariomycetes sp.
91/100
JX155957.1| Fungal endophyte
87/96
JQ759985.1| Sordariomycetes sp.
JQ759966.1| Sordariomycetes sp.
94/100
61/--
JQ761140.1| Sordariomycetes sp.
JQ761138.1| Sordariomycetes sp.
JQ761137.1| Sordariomycetes sp.
97/70
JQ761136.1| Sordariomycetes sp.
JQ761100.1| Sordariomycetes sp.
JQ761139.1| Sordariomycetes sp.
0.02
10.151

J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JX243908.1| Sordariomycetes sp.
DQ923534.1| Phlogicylindrium eucalypti CBS
100/100
EU040222.1| Phlogicylindrium eucalyptorum
98/93
EU040223.1| Phlogicylindrium eucalyptorum CBS
JX244036.1| Sordariomycetes sp.
JX244016.1| Sordariomycetes sp.
JX244027.1| Sordariomycetes sp.
99/100
JX244037.1| Sordariomycetes sp.
JX244039.1| Sordariomycetes sp.
JX244043.1| Sordariomycetes sp.
JX244026.1| Sordariomycetes sp.
KJ869126.1| Beltraniopsis neolitseae
--/100
KF436002.1| Fungal endophyte STRI:ICBG
97/100
KF436001.1| Fungal endophyte STRI:ICBG
100/100
EF029240.1| Beltrania querna ICMP
AF437754.1| Fungal isolate
JN853777.1| Beltrania sp.
69/100
GU905994.1| Beltrania querna BCRC
KF435614.1| Fungal endophyte STRI:ICBG
10.161org
11.5
51/97
AF502826.1| Leaf litter ascomycete
GU797390.1| Beltrania rhombica
EU563613.1| Fungal sp.
KF435584.1| Fungal endophyte STRI:ICBG
KJ869158.1| Beltrania pseudorhombica
KF435204.1| Fungal endophyte STRI:ICBG
86/100
KF435528.1| Fungal endophyte STRI:ICBG
KF435523.1| Fungal endophyte STRI:ICBG
AF502740.1| Leaf litter ascomycete
AF502785.1| Leaf litter ascomycete
70/100
KF435911.1| Fungal endophyte STRI:ICBG
56/96
KF435912.1| Fungal endophyte STRI:ICBG
AB511813.1| Xylariales sp.
95/100
AF502754.1| Leaf litter ascomycete
GU905996.1| Menisporopsis theobromae BCRC
KJ869128.1| Beltraniella endiandrae
56/--
EU687114.1| Fungal endophyte
GU905993.1| Beltraniella portoricensis BCRC
70/99
F440975.1| Leiosphaerella lycopodina
JQ759962.1| Sordariomycetes sp.
HM122907.1| Fungal sp.
79/99
JQ761166.1| Sordariomycetes sp.
JQ761273.1| Sordariomycetes sp.
JQ761294.1| Sordariomycetes sp.
JQ761297.1| Sordariomycetes sp.
80/100
67/91
HM122945.1| Fungal sp.
HM123257.1| Fungal sp.
HM122946.1| Fungal sp.
HM122911.1| Fungal sp.
HM122912.1| Fungal sp.
HM122938.1| Fungal sp.
HM123255.1| Fungal sp.
HM123631.1| Fungal sp.
87/97
HM123632.1| Fungal sp.
HM123645.1| Fungal sp.
HM123713.1| Fungal sp.
0.05
HM122768.1| Fungal sp.
87/100
HM122906.1| Fungal sp.
HM122908.1| Fungal sp.
HM122909.1| Fungal sp.
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HM122937.1| Fungal sp.
HM122944.1| Fungal sp.
85/98
HM122947.1| Fungal sp.
HM122948.1| Fungal sp.
HM122949.1| Fungal sp.
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HM123171.1| Fungal sp.
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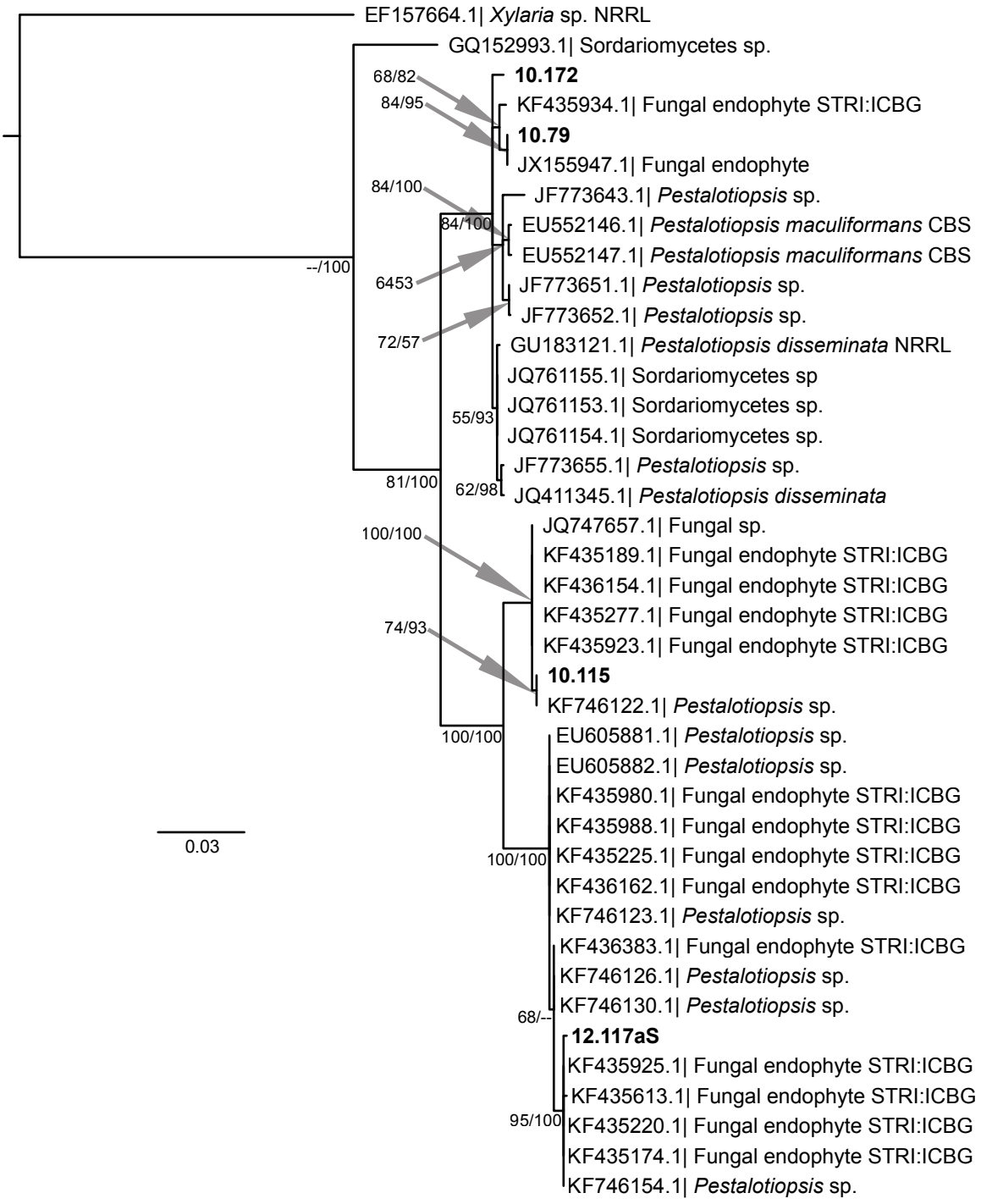
HM123425.1| Fungal sp.

K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68/82
84/95
10.172
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JX155947.1| Fungal endophyte
84/100
JF773643.1| Pestalotiopsis sp.
84/100
EU552146.1| Pestalotiopsis maculiformans CBS
EU552147.1| Pestalotiopsis maculiformans CBS
--/100
6453
JF773651.1| Pestalotiopsis sp.
JF773652.1| Pestalotiopsis sp.
72/57
GU183121.1| Pestalotiopsis disseminata NRRL
JQ761155.1| Sordariomycetes sp
JQ761153.1| Sordariomycetes sp.
55/93
JQ761154.1| Sordariomycetes sp.
JF773655.1| Pestalotiopsis sp.
81/100
62/98
JQ411345.1| Pestalotiopsis disseminata
JQ747657.1| Fungal sp.
100/100
KF435189.1| Fungal endophyte STRI:ICBG
KF436154.1| Fungal endophyte STRI:ICBG
KF435277.1| Fungal endophyte STRI:ICBG
74/93
KF435923.1| Fungal endophyte STRI:ICBG
10.115
KF746122.1| Pestalotiopsis sp.
100/100
EU605881.1| Pestalotiopsis sp.
EU605882.1| Pestalotiopsis sp.
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KF435988.1| Fungal endophyte STRI:ICBG
0.03
100/100
KF435225.1| Fungal endophyte STRI:ICBG
KF436162.1| Fungal endophyte STRI:ICBG
KF746123.1| Pestalotiopsis sp.
KF436383.1| Fungal endophyte STRI:ICBG
KF746126.1| Pestalotiopsis sp.
KF746130.1| Pestalotiopsis sp.
68/--
12.117aS
KF435925.1| Fungal endophyte STRI:ICBG
KF435613.1| Fungal endophyte STRI:ICBG
95/100
KF435220.1| Fungal endophyte STRI:ICBG
KF435174.1| Fungal endophyte STRI:ICBG
KF746154.1| Pestalotiopsis sp.

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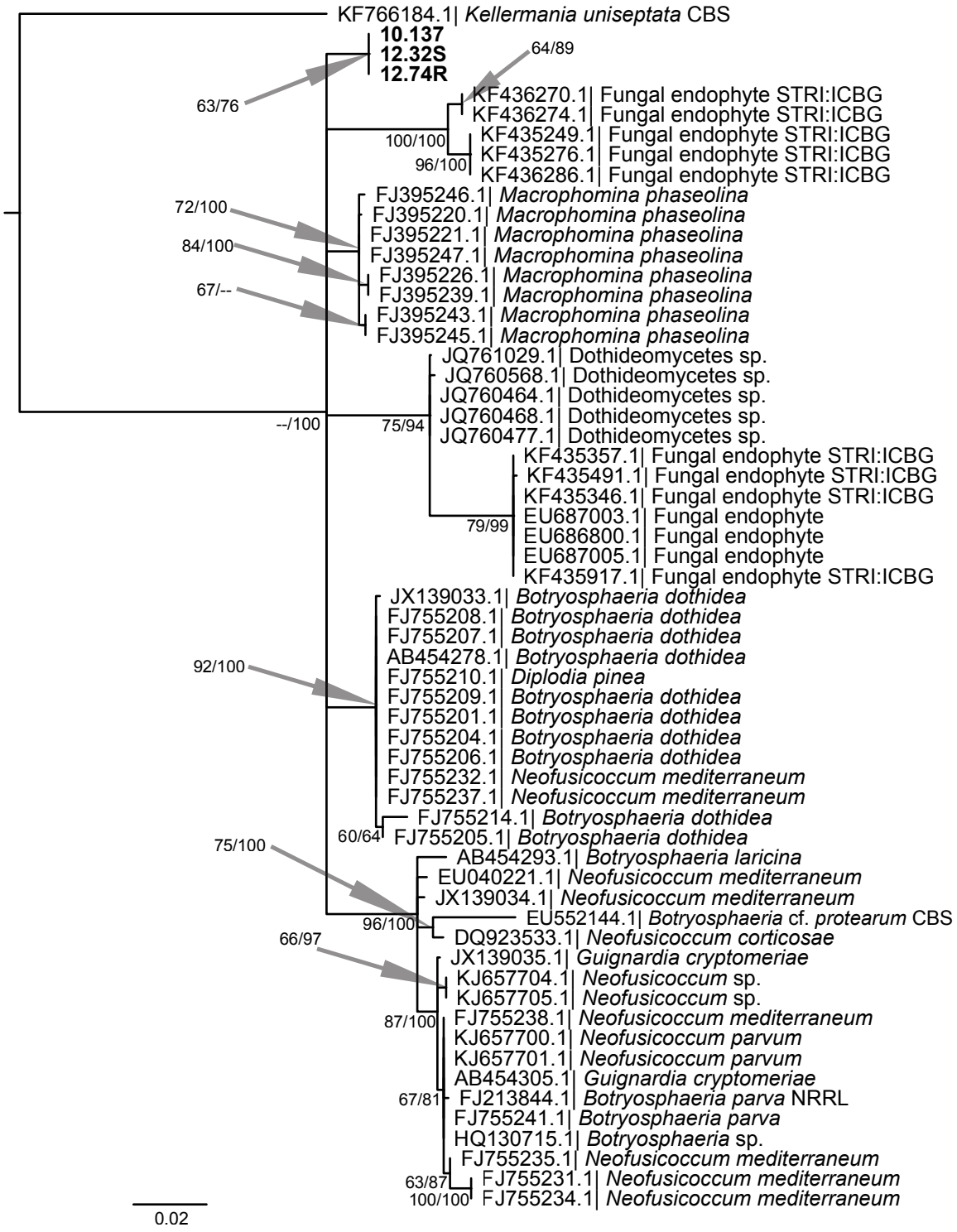
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12.32S
12.74R
64/89
63/76
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KF436274.1| Fungal endophyte STRI:ICBG
100/100
KF435249.1| Fungal endophyte STRI:ICBG
96/100
KF435276.1| Fungal endophyte STRI:ICBG
KF436286.1| Fungal endophyte STRI:ICBG
72/100
FJ395246.1| Macrophomina phaseolina
FJ395220.1| Macrophomina phaseolina
84/100
FJ395221.1| Macrophomina phaseolina
FJ395247.1| Macrophomina phaseolina
67/--
FJ395226.1| Macrophomina phaseolina
FJ395239.1| Macrophomina phaseolina
FJ395243.1| Macrophomina phaseolina
FJ395245.1| Macrophomina phaseolina
JQ761029.1| Dothideomycetes sp.
JQ760568.1| Dothideomycetes sp.
JQ760464.1| Dothideomycetes sp.
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75/94
JQ760468.1| Dothideomycetes sp.
JQ760477.1| Dothideomycetes sp.
KF435357.1| Fungal endophyte STRI:ICBG
KF435491.1| Fungal endophyte STRI:ICBG
KF435346.1| Fungal endophyte STRI:ICBG
79/99
EU687003.1| Fungal endophyte
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EU687005.1| Fungal endophyte
KF435917.1| Fungal endophyte STRI:ICBG
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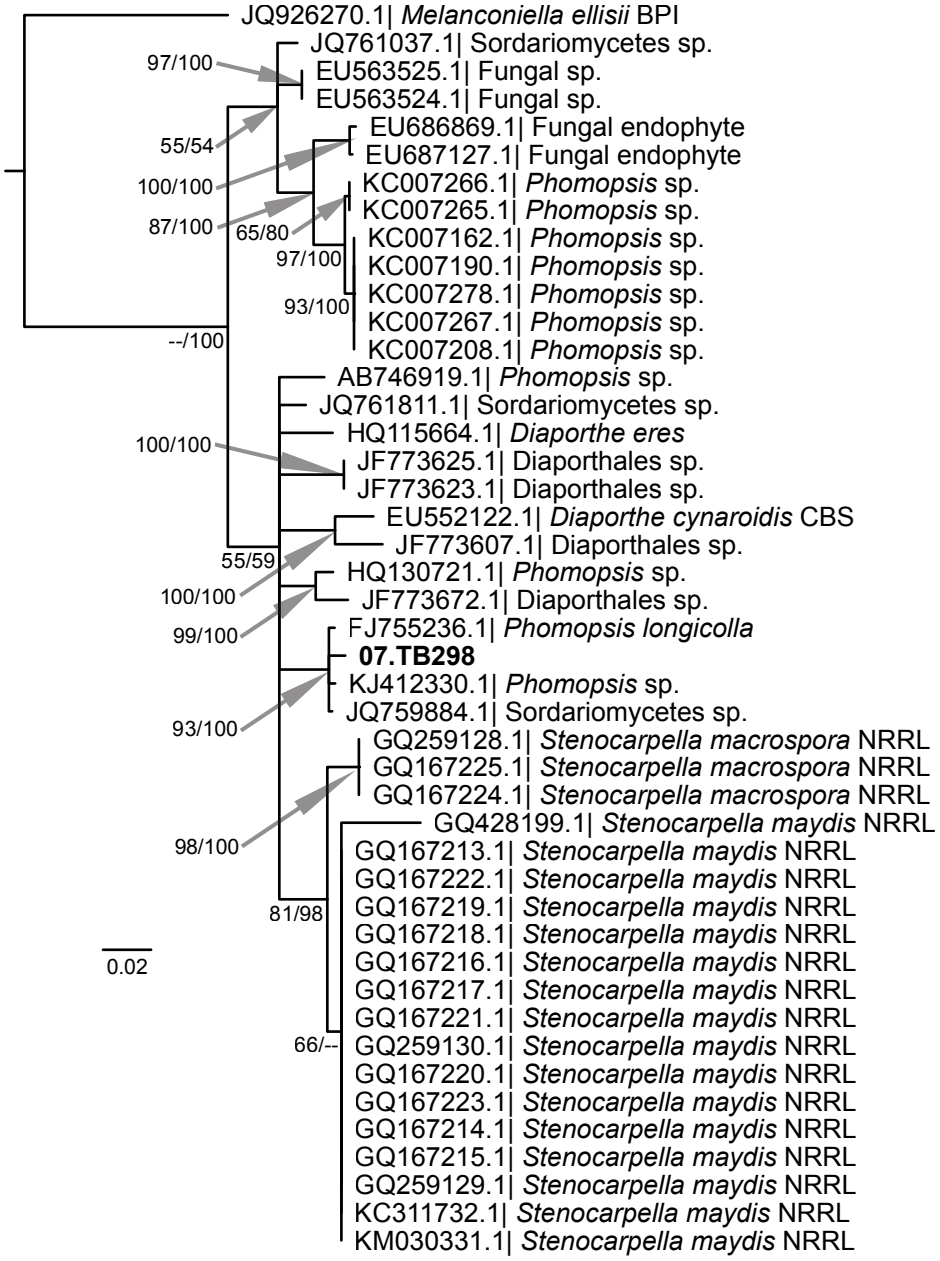
FJ755208.1| Botryosphaeria dothidea
FJ755207.1| Botryosphaeria dothidea
AB454278.1| Botryosphaeria dothidea
92/100
FJ755210.1| Diplodia pinea
FJ755209.1| Botryosphaeria dothidea
FJ755201.1| Botryosphaeria dothidea
FJ755204.1| Botryosphaeria dothidea
FJ755206.1| Botryosphaeria dothidea
FJ755232.1| Neofusicoccum mediterraneum
FJ755237.1| Neofusicoccum mediterraneum
FJ755214.1| Botryosphaeria dothidea
60/64
FJ755205.1| Botryosphaeria dothidea
75/100
AB454293.1| Botryosphaeria laricina
EU040221.1| Neofusicoccum mediterraneum
JX139034.1| Neofusicoccum mediterraneum
96/100
EU552144.1| Botryosphaeria cf. protearum CBS
DQ923533.1| Neofusicoccum corticosae
66/97
JX139035.1| Guignardia cryptomeriae
KJ657704.1| Neofusicoccum sp.
KJ657705.1| Neofusicoccum sp.
87/100
FJ755238.1| Neofusicoccum mediterraneum
KJ657700.1| Neofusicoccum parvum
KJ657701.1| Neofusicoccum parvum
AB454305.1| Guignardia cryptomeriae
67/81
FJ213844.1| Botryosphaeria parva NRRL
FJ755241.1| Botryosphaeria parva
HQ130715.1| Botryosphaeria sp.
FJ755235.1| Neofusicoccum mediterraneum
63/87
FJ755231.1| Neofusicoccum mediterraneum
100/100
FJ755234.1| Neofusicoccum mediterraneum
0.02

M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EU563524.1| Fungal sp.
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EU687127.1| Fungal endophyte
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KC007265.1| Phomopsis sp.
KC007162.1| Phomopsis sp.
KC007190.1| Phomopsis sp.
KC007278.1| Phomopsis sp.
KC007267.1| Phomopsis sp.
KC007208.1| Phomopsis sp.
AB746919.1| Phomopsis sp.
JQ761811.1| Sordariomycetes sp.
HQ115664.1| Diaporthe eres
JF773625.1| Diaporthales sp.
JF773623.1| Diaporthales sp.
EU552122.1| Diaporthe cynaroidis CBS
JF773607.1| Diaporthales sp.
HQ130721.1| Phomopsis sp.
JF773672.1| Diaporthales sp.
FJ755236.1| Phomopsis longicolla
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KJ412330.1| Phomopsis sp.
JQ759884.1| Sordariomycetes sp.
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GQ167225.1| Stenocarpella macrospora NRRL
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GQ428199.1| Stenocarpella maydis NRRL
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GQ167222.1| Stenocarpella maydis NRRL
GQ167219.1| Stenocarpella maydis NRRL
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GQ167217.1| Stenocarpella maydis NRRL
GQ167221.1| Stenocarpella maydis NRRL
GQ259130.1| Stenocarpella maydis NRRL
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97/100
55/54
100/100
87/100
65/80
97/100
93/100
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100/100
55/59
100/100
99/100
93/100
98/100
81/98
66/--
0.02

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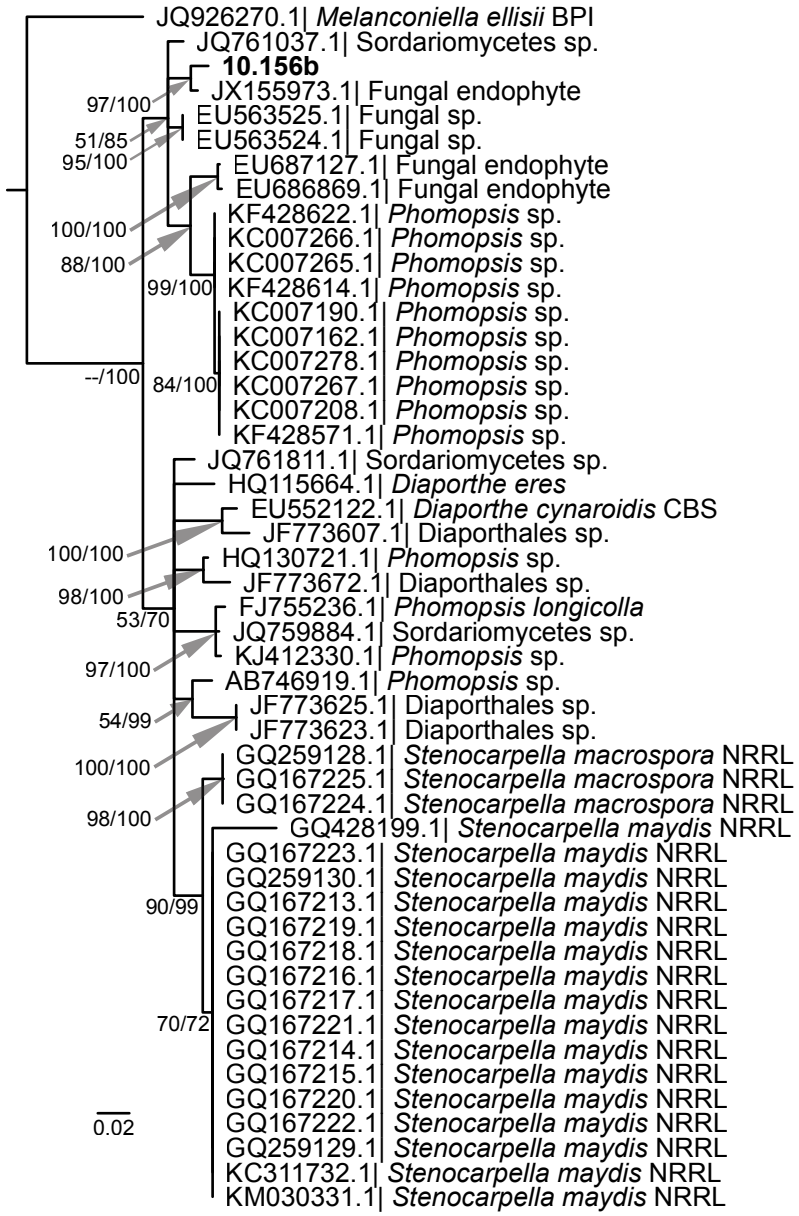
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KC007278.1| *Phomopsis* sp.
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KJ412330.1| *Phomopsis* sp.
AB746919.1| *Phomopsis* sp.
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KM030331.1| *Stenocarpella maydis* NRRL
97/100
51/85
95/100
100/100
88/100
99/100
--/100
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100/100
98/100
90/99
70/72
0.02

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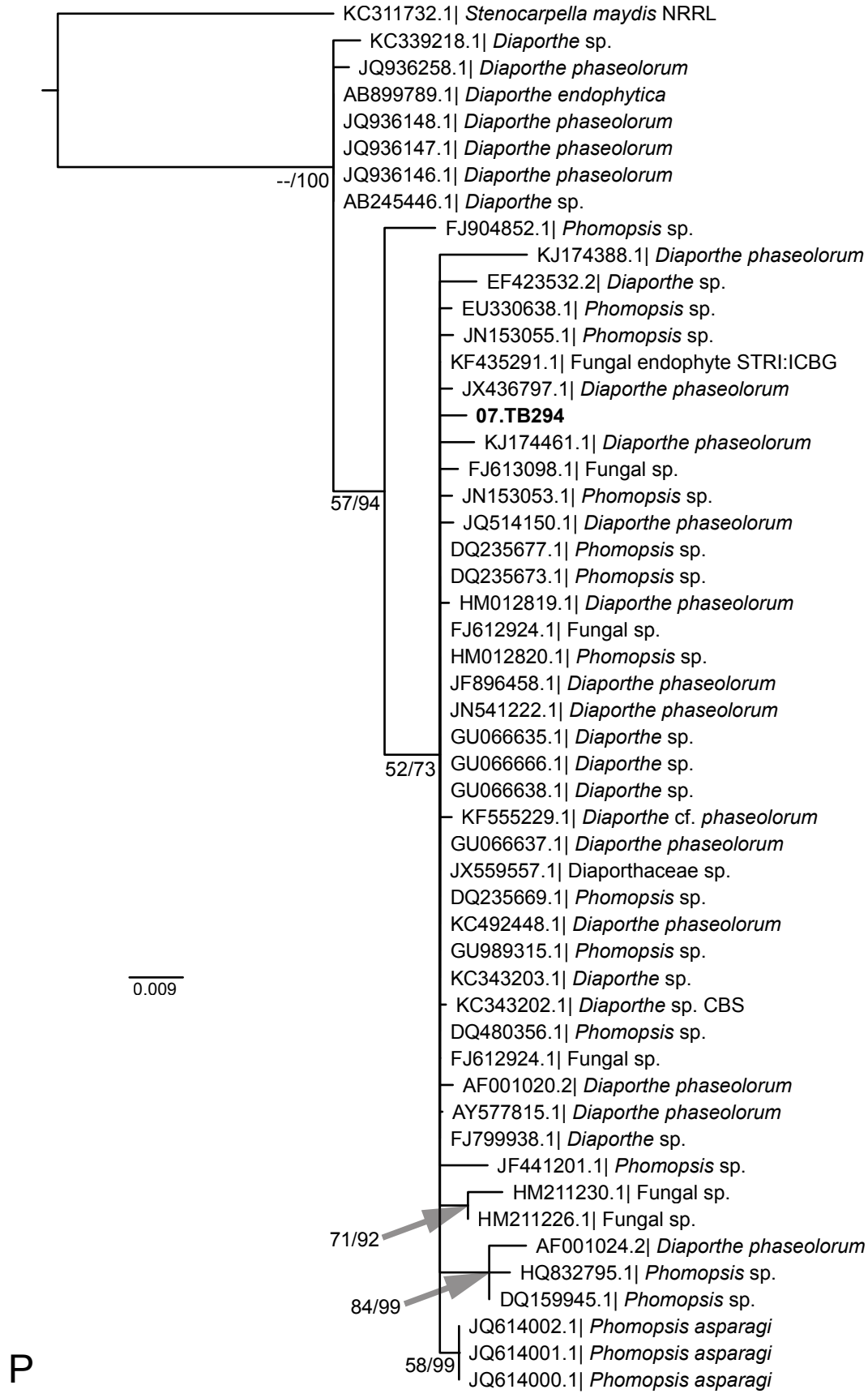
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JQ936148.1| Diaporthe phaseolorum
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57/94
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HM012820.1| Phomopsis sp.
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71/92
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84/99
DQ159945.1| Phomopsis sp.
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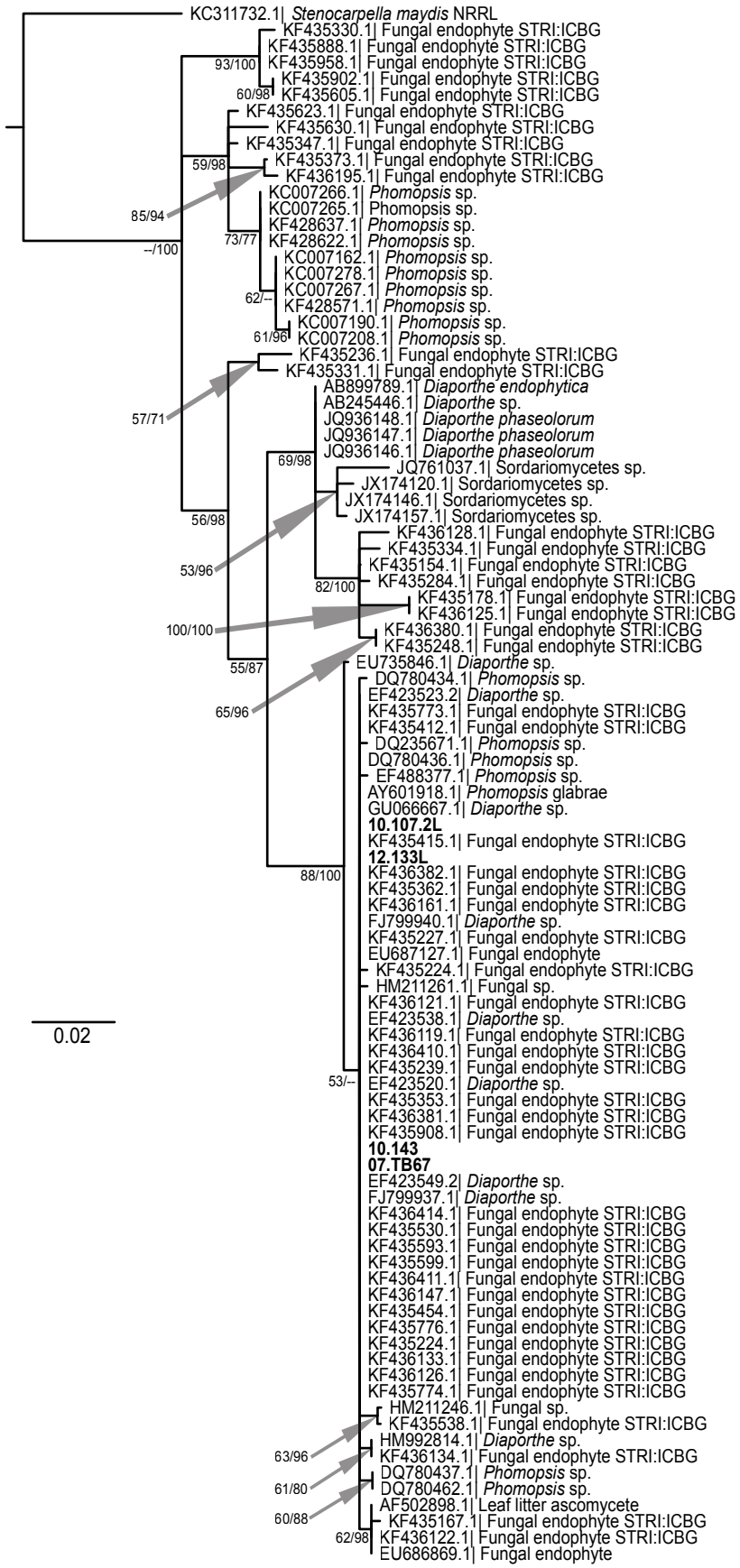
58/99
JQ614000.1| Phomopsis asparagi

P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KC007278.1| Phomopsis sp.
KC007267.1| Phomopsis sp.
KF428571.1| Phomopsis sp.
KC007190.1| Phomopsis sp.
KC007208.1| Phomopsis sp.
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KF436128.1| Fungal endophyte STRI:ICBG
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KF435284.1| Fungal endophyte STRI:ICBG
KF435178.1| Fungal endophyte STRI:ICBG
KF436125.1| Fungal endophyte STRI:ICBG
KF436380.1| Fungal endophyte STRI:ICBG
KF435248.1| Fungal endophyte STRI:ICBG
EU735846.1| Diaporthe sp.
DQ780434.1| Phomopsis sp.
EF423523.2| Diaporthe sp.
KF435773.1| Fungal endophyte STRI:ICBG
KF435412.1| Fungal endophyte STRI:ICBG
DQ235671.1| Phomopsis sp.
DQ780436.1| Phomopsis sp.
EF488377.1| Phomopsis sp.
AY601918.1| Phomopsis glabrae
GU066667.1| Diaporthe sp.
10.107.2L
KF435415.1| Fungal endophyte STRI:ICBG
12.133L
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FJ799940.1| Diaporthe sp.
KF435227.1| Fungal endophyte STRI:ICBG
EU687127.1| Fungal endophyte
KF435224.1| Fungal endophyte STRI:ICBG
HM211261.1| Fungal sp.
KF436121.1| Fungal endophyte STRI:ICBG
EF423538.1| Diaporthe sp.
KF436119.1| Fungal endophyte STRI:ICBG
KF436410.1| Fungal endophyte STRI:ICBG
KF435239.1| Fungal endophyte STRI:ICBG
EF423520.1 Diaporthe sp.
KF435353.1| Fungal endophyte STRI:ICBG
KF436381.1| Fungal endophyte STRI:ICBG
KF435908.1| Fungal endophyte STRI:ICBG
10.143
07.TB67
EF423549.2| Diaporthe sp.
FJ799937.1| Diaporthe sp.
KF436414.1| Fungal endophyte STRI:ICBG
KF435530.1| Fungal endophyte STRI:ICBG
KF435593.1| Fungal endophyte STRI:ICBG
KF435599.1| Fungal endophyte STRI:ICBG
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KF435776.1| Fungal endophyte STRI:ICBG
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KF436133.1| Fungal endophyte STRI:ICBG
KF436126.1| Fungal endophyte STRI:ICBG
KF435774.1| Fungal endophyte STRI:ICBG
HM211246.1| Fungal sp.
KF435538.1| Fungal endophyte STRI:ICBG
HM992814.1| Diaporthe sp.
KF436134.1| Fungal endophyte STRI:ICBG
DQ780437.1| Phomopsis sp.
DQ780462.1| Phomopsis sp.
AF502898.1| Leaf litter ascomycete
KF435167.1| Fungal endophyte STRI:ICBG
KF436122.1| Fungal endophyte STRI:ICBG
EU686869.1| Fungal endophyte
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60/98
59/98
85/94
--/100
73/77
62/--
61/96
57/71
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56/98
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65/96
88/100
53/--
63/96
61/80
60/88
62/98
0.02

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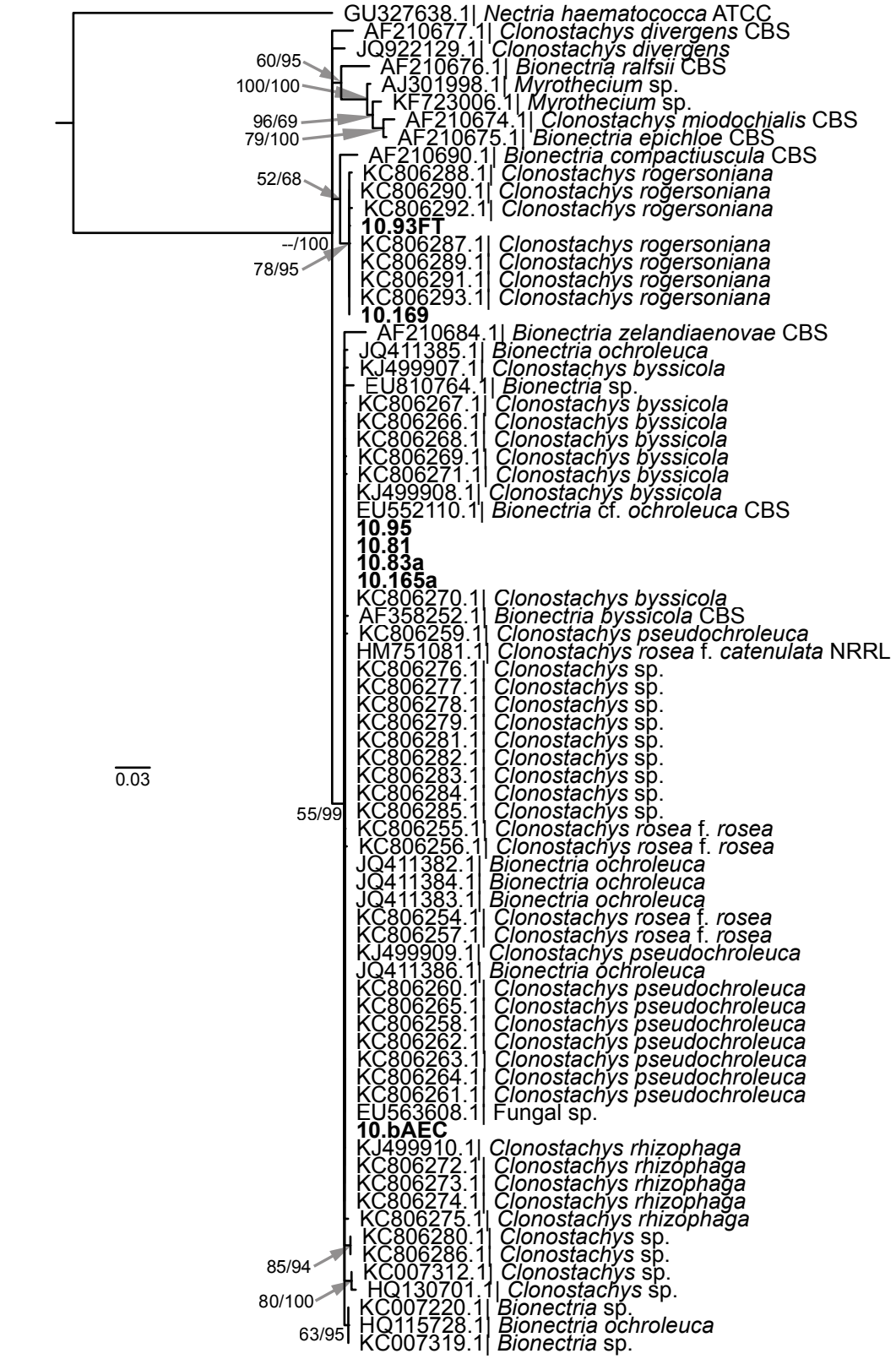
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100/100
AJ301998.1| Myrothecium sp.
KF723006.1| Myrothecium sp.
96/69
AF210674.1| Clonostachys miodochialis CBS
79/100
AF210675.1| Bionectria epichloe CBS
AF210690.1| Bionectria compactiuscula CBS
52/68
KC806288.1| Clonostachys rogersoniana
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10.93FT
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KC806287.1| Clonostachys rogersoniana
78/95
KC806289.1| Clonostachys rogersoniana
KC806291.1| Clonostachys rogersoniana
KC806293.1| Clonostachys rogersoniana
10.169
AF210684.1| Bionectria zelandiaenovae CBS
JQ411385.1| Bionectria ochroleuca
KJ499907.1| Clonostachys byssicola
EU810764.1| Bionectria sp.
KC806267.1| Clonostachys byssicola
KC806266.1| Clonostachys byssicola
KC806268.1| Clonostachys byssicola
KC806269.1| Clonostachys byssicola
KC806271.1| Clonostachys byssicola
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10.95
10.81
10.83a
10.165a
KC806270.1| Clonostachys byssicola
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KC806276.1| Clonostachys sp.
KC806277.1| Clonostachys sp.
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KC806282.1| Clonostachys sp.
0.03
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KC806284.1| Clonostachys sp.
55/99
KC806285.1| Clonostachys sp.
KC806255.1| Clonostachys rosea f. rosea
KC806256.1| Clonostachys rosea f. rosea
JQ411382.1| Bionectria ochroleuca
JQ411384.1| Bionectria ochroleuca
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KC806254.1| Clonostachys rosea f. rosea
KC806257.1| Clonostachys rosea f. rosea
KJ499909.1| Clonostachys pseudochroleuca
JQ411386.1| Bionectria ochroleuca
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KC806258.1| Clonostachys pseudochroleuca
KC806262.1| Clonostachys pseudochroleuca
KC806263.1| Clonostachys pseudochroleuca
KC806264.1| Clonostachys pseudochroleuca
KC806261.1| Clonostachys pseudochroleuca
EU563608.1| Fungal sp.
10.bAEC
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KC806272.1| Clonostachys rhizophaga
KC806273.1| Clonostachys rhizophaga
KC806274.1| Clonostachys rhizophaga
KC806275.1| Clonostachys rhizophaga
KC806280.1| Clonostachys sp.
85/94
KC806286.1| Clonostachys sp.
KC007312.1| Clonostachys sp.
80/100
HQ130701.1| Clonostachys sp.
KC007220.1| Bionectria sp.
63/95
HQ115728.1| Bionectria ochroleuca
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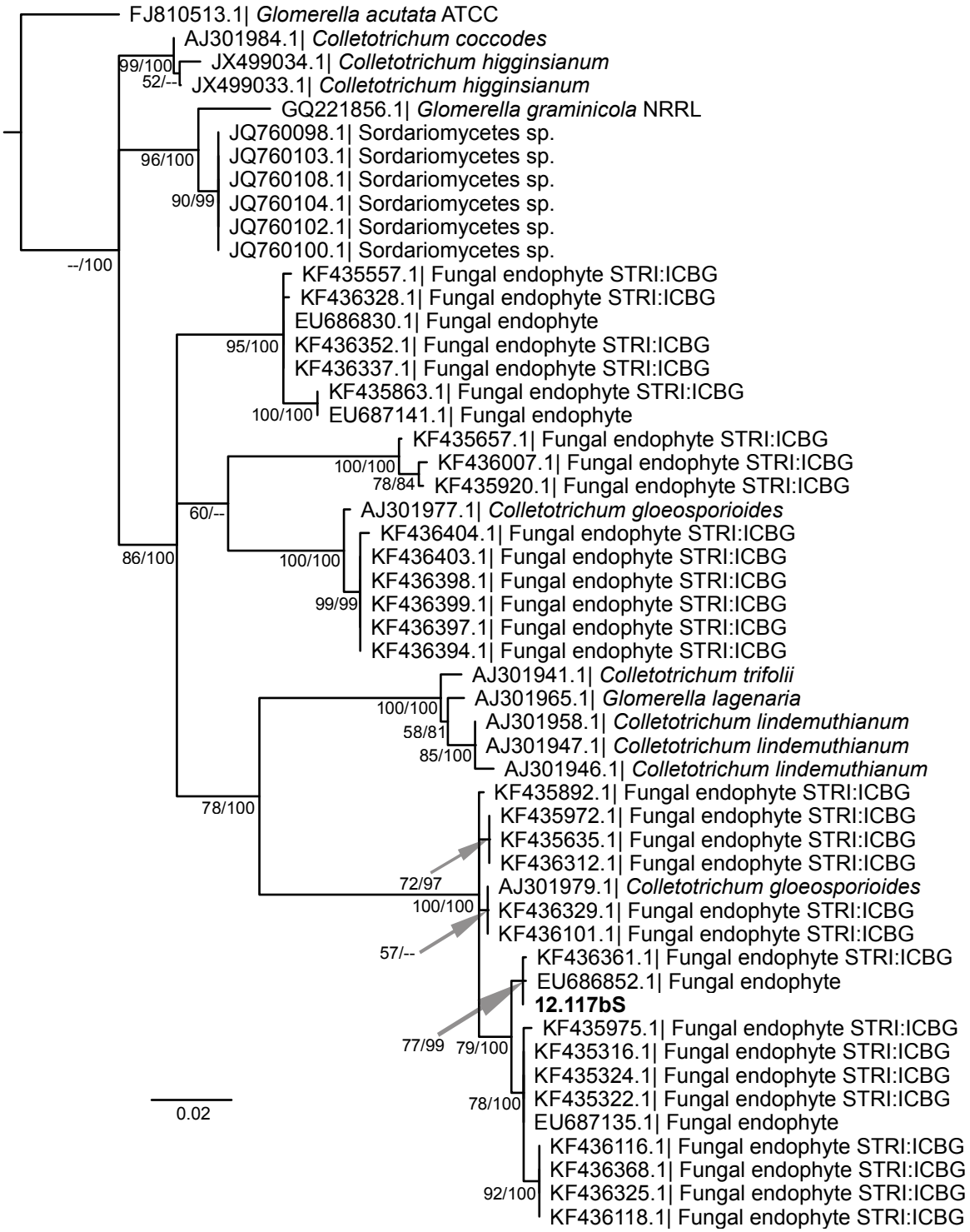
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JQ760098.1| Sordariomycetes sp.
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AJ301965.1| Glomerella lagenaria
AJ301958.1| Colletotrichum lindemuthianum
AJ301947.1| Colletotrichum lindemuthianum
AJ301946.1| Colletotrichum lindemuthianum
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KF436312.1| Fungal endophyte STRI:ICBG
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KF436101.1| Fungal endophyte STRI:ICBG
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12.117bS
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99/100
52/--
96/100
90/99
--/100
95/100
100/100
100/100
78/84
60/--
86/100
100/100
99/99
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79/100
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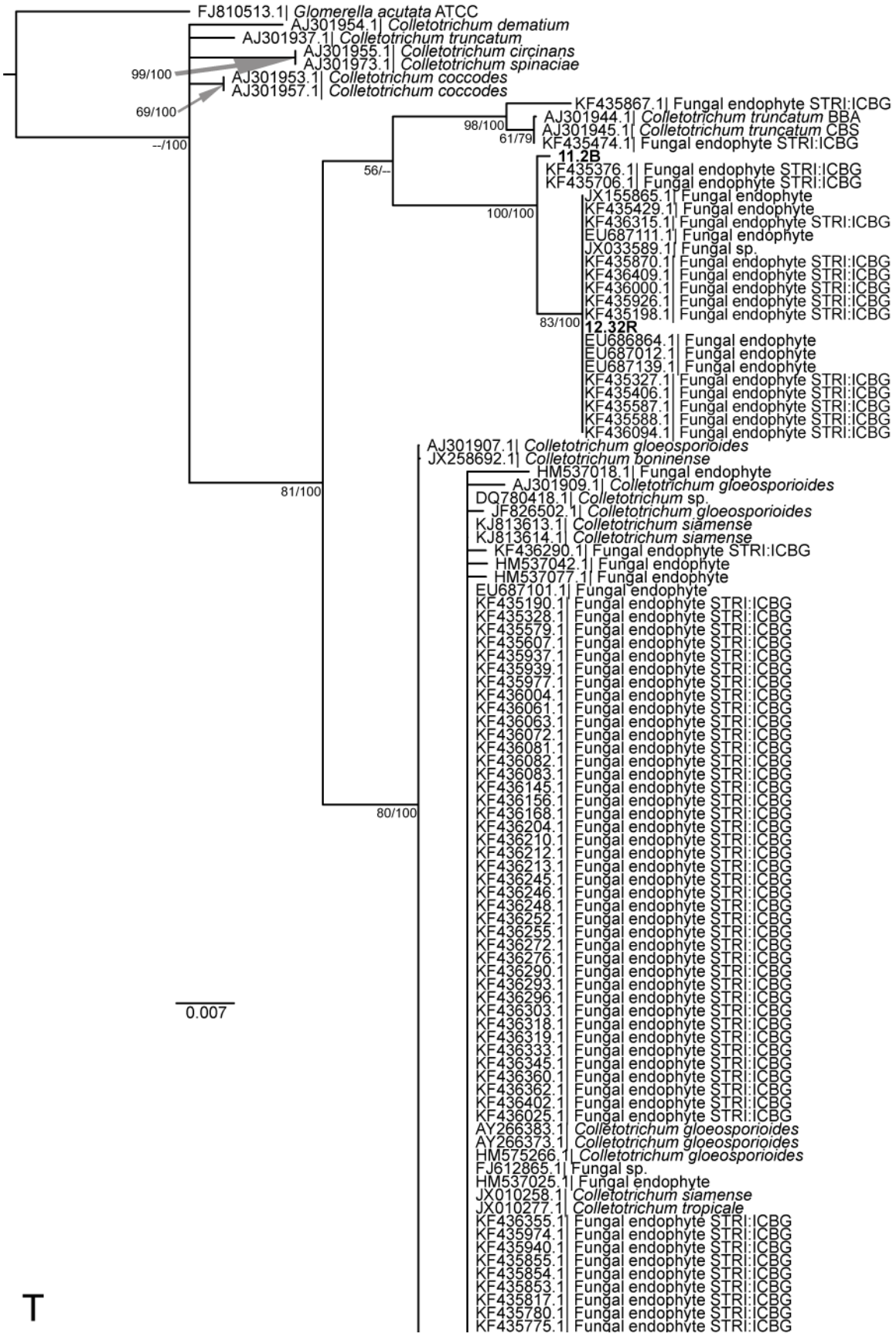
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99/100
69/100
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98/100
61/79
11.2B
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KF435706.1| Fungal endophyte STRI:ICBG
56/--
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KF436315.1| Fungal endophyte STRI:ICBG
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83/100
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AJ301909.1| Colletotrichum gloeosporioides
DQ780418.1| Colletotrichum sp.
JF826502.1| Colletotrichum gloeosporioides
KJ813613.1| Colletotrichum siamense
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KF436290.1| Fungal endophyte STRI:ICBG
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EU687101.1| Fungal endophyte
KF435190.1| Fungal endophyte STRI:ICBG
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HM575266.1| Colletotrichum gloeosporioides
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JX010258.1| Colletotrichum siamense
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80/100
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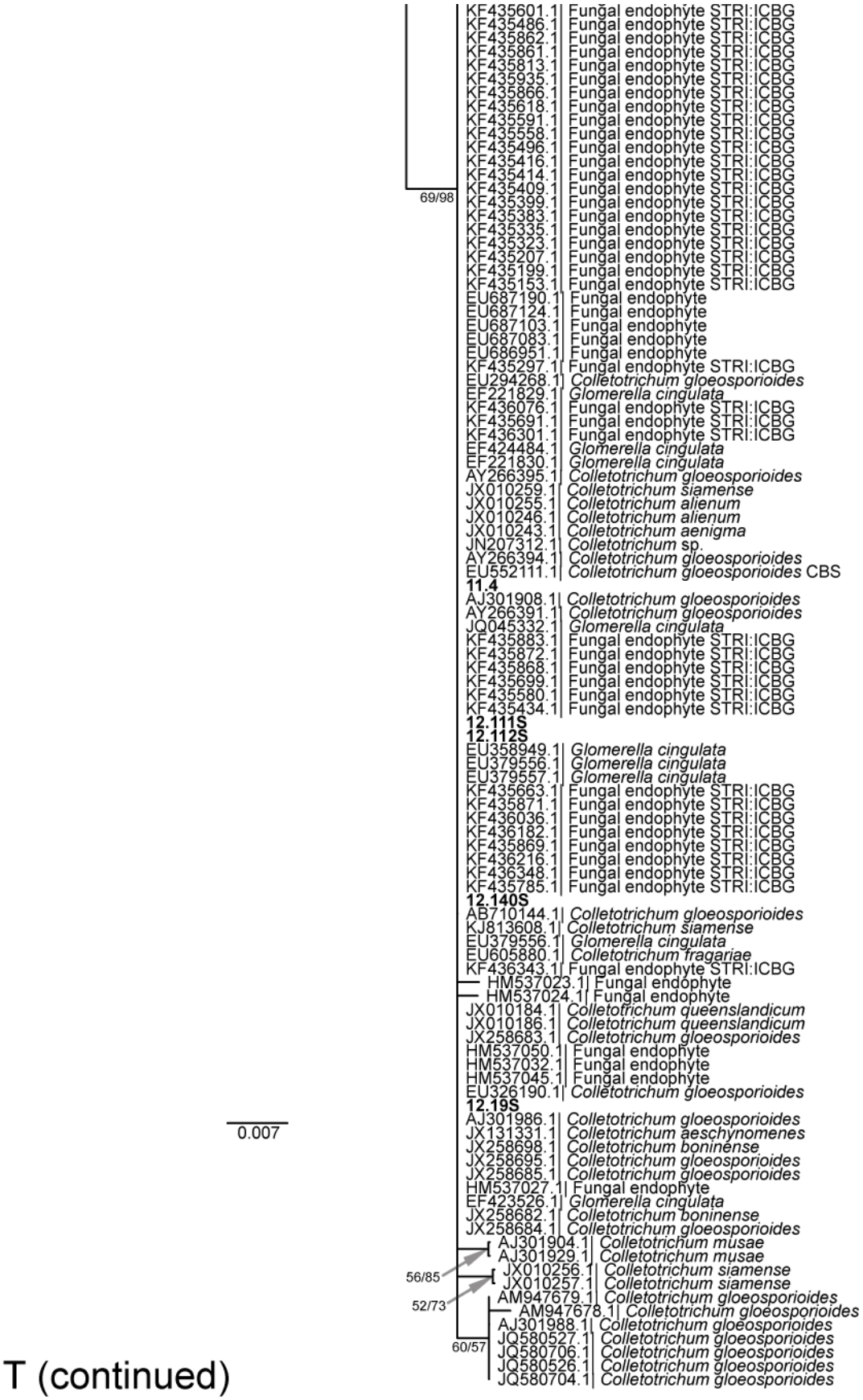
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JX010246.1| Colletotrichum alienum
JX010243.1| Colletotrichum aenigma
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11.4
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KF436348.1 Fungal endophyte STRI:ICBG
KF435785.1 Fungal endophyte STRI:ICBG
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KJ813608.1| Colletotrichum siamense
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EU605880.1| Colletotrichum fragariae
KF436343.1| Fungal endophyte STRI:ICBG
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56/85
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52/73
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60/57
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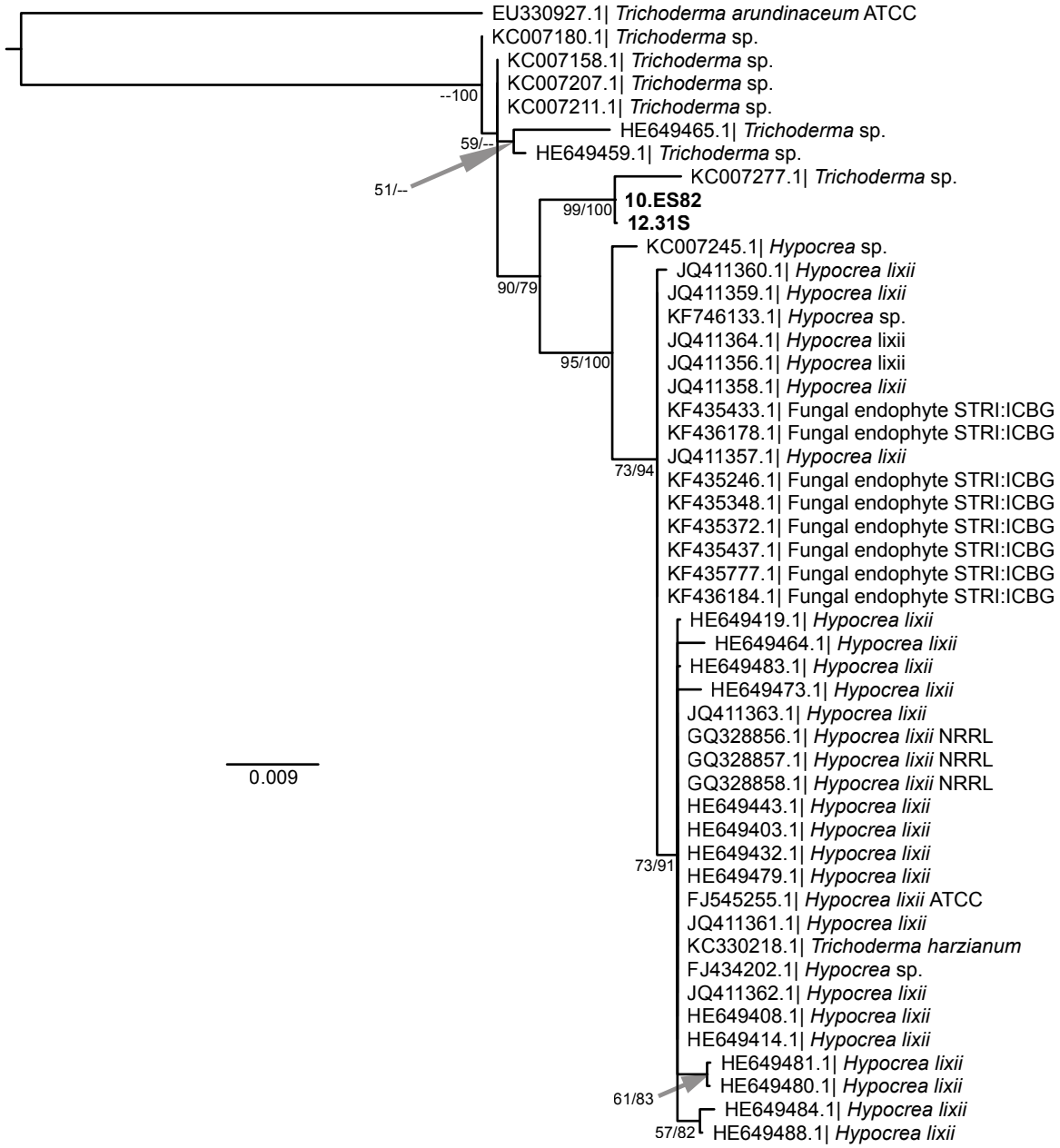
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KF436184.1| Fungal endophyte STRI:ICBG
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HE649464.1| Hypocrea lixii
HE649483.1| Hypocrea lixii
HE649473.1| Hypocrea lixii
JQ411363.1| Hypocrea lixii
GQ328856.1| Hypocrea lixii NRRL
GQ328857.1| Hypocrea lixii NRRL
GQ328858.1| Hypocrea lixii NRRL
HE649443.1| Hypocrea lixii
HE649403.1| Hypocrea lixii
HE649432.1| Hypocrea lixii
HE649479.1| Hypocrea lixii
FJ545255.1| Hypocrea lixii ATCC
JQ411361.1| Hypocrea lixii
KC330218.1| Trichoderma harzianum
FJ434202.1| Hypocrea sp.
JQ411362.1| Hypocrea lixii
HE649408.1| Hypocrea lixii
HE649414.1| Hypocrea lixii
HE649481.1| Hypocrea lixii
HE649480.1| Hypocrea lixii
HE649484.1| Hypocrea lixii
HE649488.1| Hypocrea lixii
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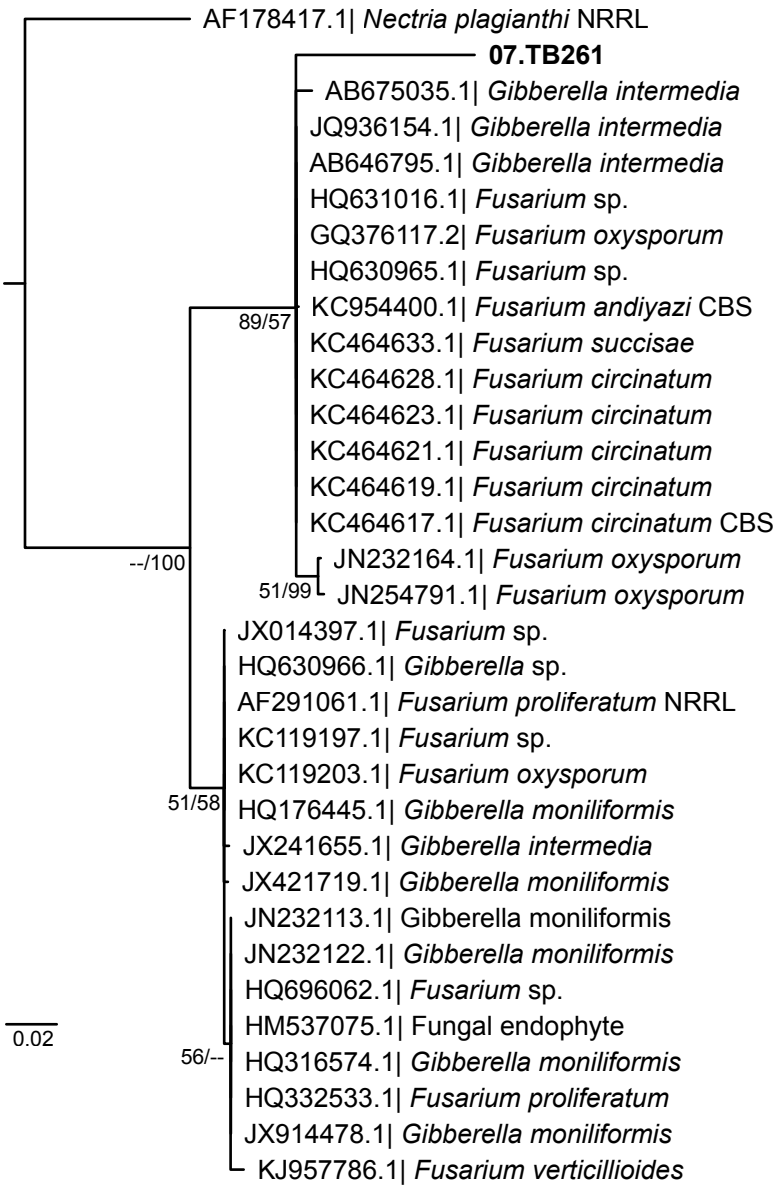
59/--
51/--
99/100
90/79
95/100
73/94
73/91
61/83
57/82
0.009

U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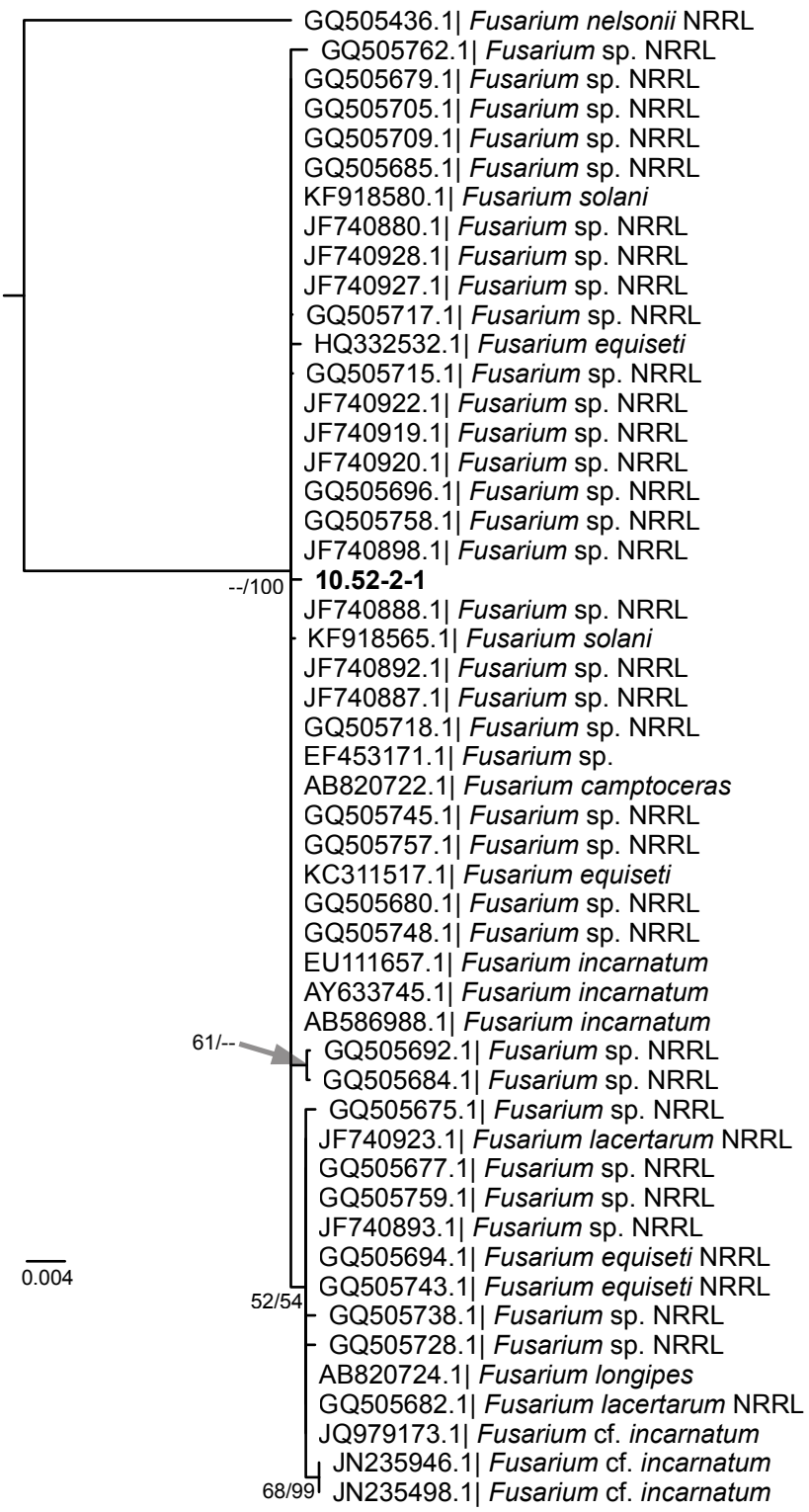
JQ936154.1| *Gibberella intermedia*
AB646795.1| *Gibberella intermedia*
HQ631016.1| *Fusarium* sp.
GQ376117.2| *Fusarium oxysporum*
HQ630965.1| *Fusarium* sp.
KC954400.1| *Fusarium andiyazi* CBS
89/57
KC464633.1| *Fusarium succisae*
KC464628.1| *Fusarium circinatum*
KC464623.1| *Fusarium circinatum*
KC464621.1| *Fusarium circinatum*
KC464619.1| *Fusarium circinatum*
KC464617.1| *Fusarium circinatum* CBS
--/100
JN232164.1| *Fusarium oxysporum*
51/99
JN254791.1| *Fusarium oxysporum*
JX014397.1| *Fusarium* sp.
HQ630966.1| *Gibberella* sp.
AF291061.1| *Fusarium proliferatum* NRRL
KC119197.1| *Fusarium* sp.
KC119203.1| *Fusarium oxysporum*
51/58
HQ176445.1| *Gibberella moniliformis*
JX241655.1| *Gibberella intermedia*
JX421719.1| *Gibberella moniliformis*
JN232113.1| Gibberella moniliformis
JN232122.1| *Gibberella moniliformis*
HQ696062.1| *Fusarium* sp.
HM537075.1| Fungal endophyte
0.02
56/--
HQ316574.1| *Gibberella moniliformis*
HQ332533.1| *Fusarium proliferatum*
JX914478.1| *Gibberella moniliformis*
KJ957786.1| *Fusarium verticillioides*

V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GQ505679.1| Fusarium sp. NRRL
GQ505705.1| Fusarium sp. NRRL
GQ505709.1| Fusarium sp. NRRL
GQ505685.1| Fusarium sp. NRRL
KF918580.1| Fusarium solani
JF740880.1| Fusarium sp. NRRL
JF740928.1| Fusarium sp. NRRL
JF740927.1| Fusarium sp. NRRL
GQ505717.1| Fusarium sp. NRRL
HQ332532.1| Fusarium equiseti
GQ505715.1| Fusarium sp. NRRL
JF740922.1| Fusarium sp. NRRL
JF740919.1| Fusarium sp. NRRL
JF740920.1| Fusarium sp. NRRL
GQ505696.1| Fusarium sp. NRRL
GQ505758.1| Fusarium sp. NRRL
JF740898.1| Fusarium sp. NRRL
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10.52-2-1
JF740888.1| Fusarium sp. NRRL
KF918565.1| Fusarium solani
JF740892.1| Fusarium sp. NRRL
JF740887.1| Fusarium sp. NRRL
GQ505718.1| Fusarium sp. NRRL
EF453171.1| Fusarium sp.
AB820722.1| Fusarium camptoceras
GQ505745.1| Fusarium sp. NRRL
GQ505757.1| Fusarium sp. NRRL
KC311517.1| Fusarium equiseti
GQ505680.1| Fusarium sp. NRRL
GQ505748.1| Fusarium sp. NRRL
EU111657.1| Fusarium incarnatum
AY633745.1| Fusarium incarnatum
AB586988.1| Fusarium incarnatum
61/--
GQ505692.1| Fusarium sp. NRRL
GQ505684.1| Fusarium sp. NRRL
GQ505675.1| Fusarium sp. NRRL
JF740923.1| Fusarium lacertarum NRRL
GQ505677.1| Fusarium sp. NRRL
GQ505759.1| Fusarium sp. NRRL
JF740893.1| Fusarium sp. NRRL
GQ505694.1| Fusarium equiseti NRRL
GQ505743.1| Fusarium equiseti NRRL
0.004
52/54
GQ505738.1| Fusarium sp. NRRL
GQ505728.1| Fusarium sp. NRRL
AB820724.1| Fusarium longipes
GQ505682.1| Fusarium lacertarum NRRL
JQ979173.1| Fusarium cf. incarnatum
JN235946.1| Fusarium cf. incarnatum
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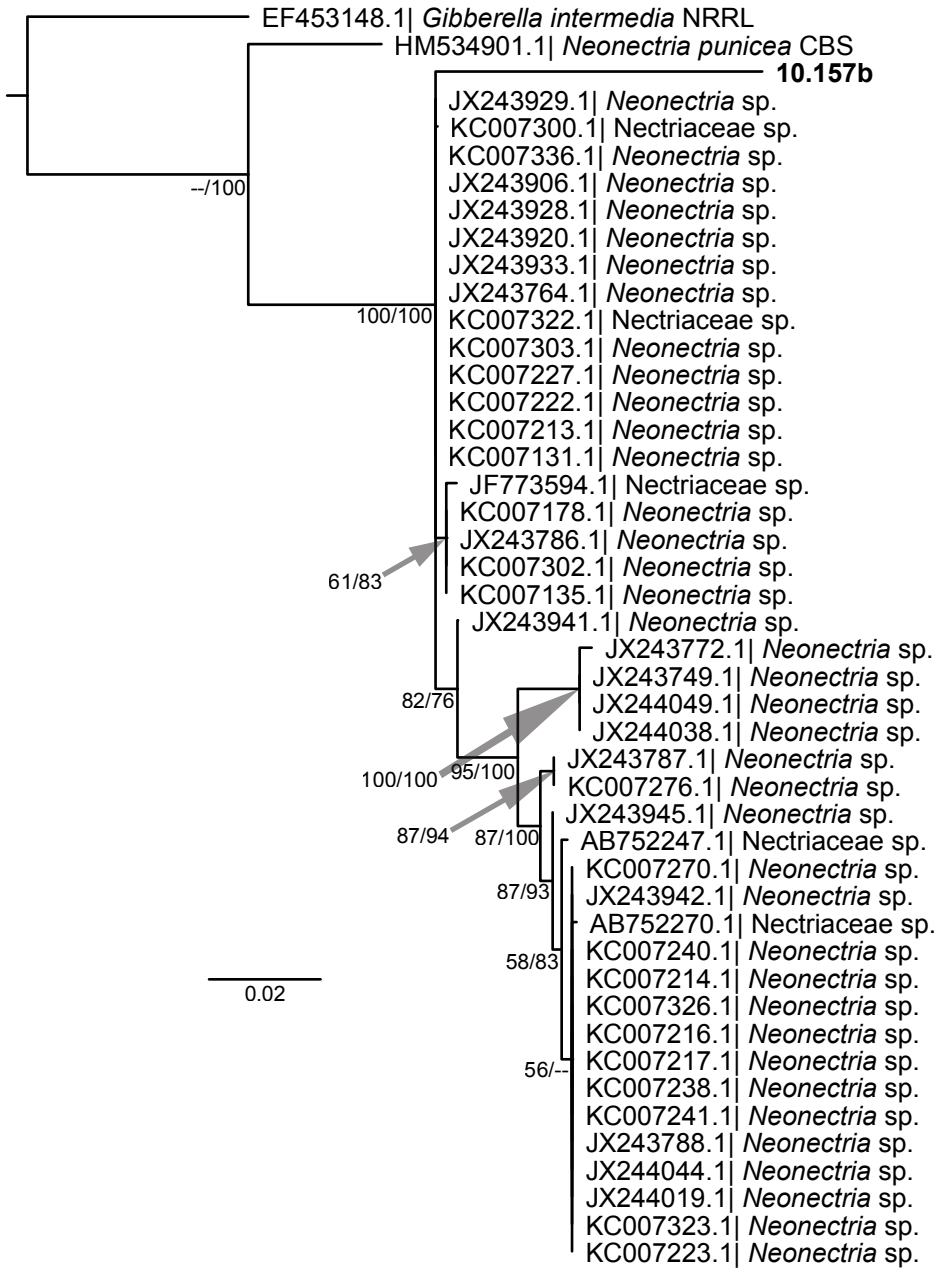
JN235498.1| Fusarium cf. incarnatum

W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KC007336.1| Neonectria sp.
JX243906.1| Neonectria sp.
JX243928.1| Neonectria sp.
JX243920.1| Neonectria sp.
JX243933.1| Neonectria sp.
JX243764.1| Neonectria sp.
KC007322.1| Nectriaceae sp.
KC007303.1| Neonectria sp.
KC007227.1| Neonectria sp.
KC007222.1| Neonectria sp.
KC007213.1| Neonectria sp.
KC007131.1| Neonectria sp.
JF773594.1| Nectriaceae sp.
KC007178.1| Neonectria sp.
JX243786.1| Neonectria sp.
KC007302.1| Neonectria sp.
KC007135.1| Neonectria sp.
JX243941.1| Neonectria sp.
JX243772.1| Neonectria sp.
JX243749.1| Neonectria sp.
JX244049.1| Neonectria sp.
JX244038.1| Neonectria sp.
JX243787.1| Neonectria sp.
KC007276.1| Neonectria sp.
JX243945.1| Neonectria sp.
AB752247.1| Nectriaceae sp.
KC007270.1| Neonectria sp.
JX243942.1| Neonectria sp.
AB752270.1| Nectriaceae sp.
KC007240.1| Neonectria sp.
KC007214.1| Neonectria sp.
KC007326.1| Neonectria sp.
KC007216.1| Neonectria sp.
KC007217.1| Neonectria sp.
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KC007241.1| Neonectria sp.
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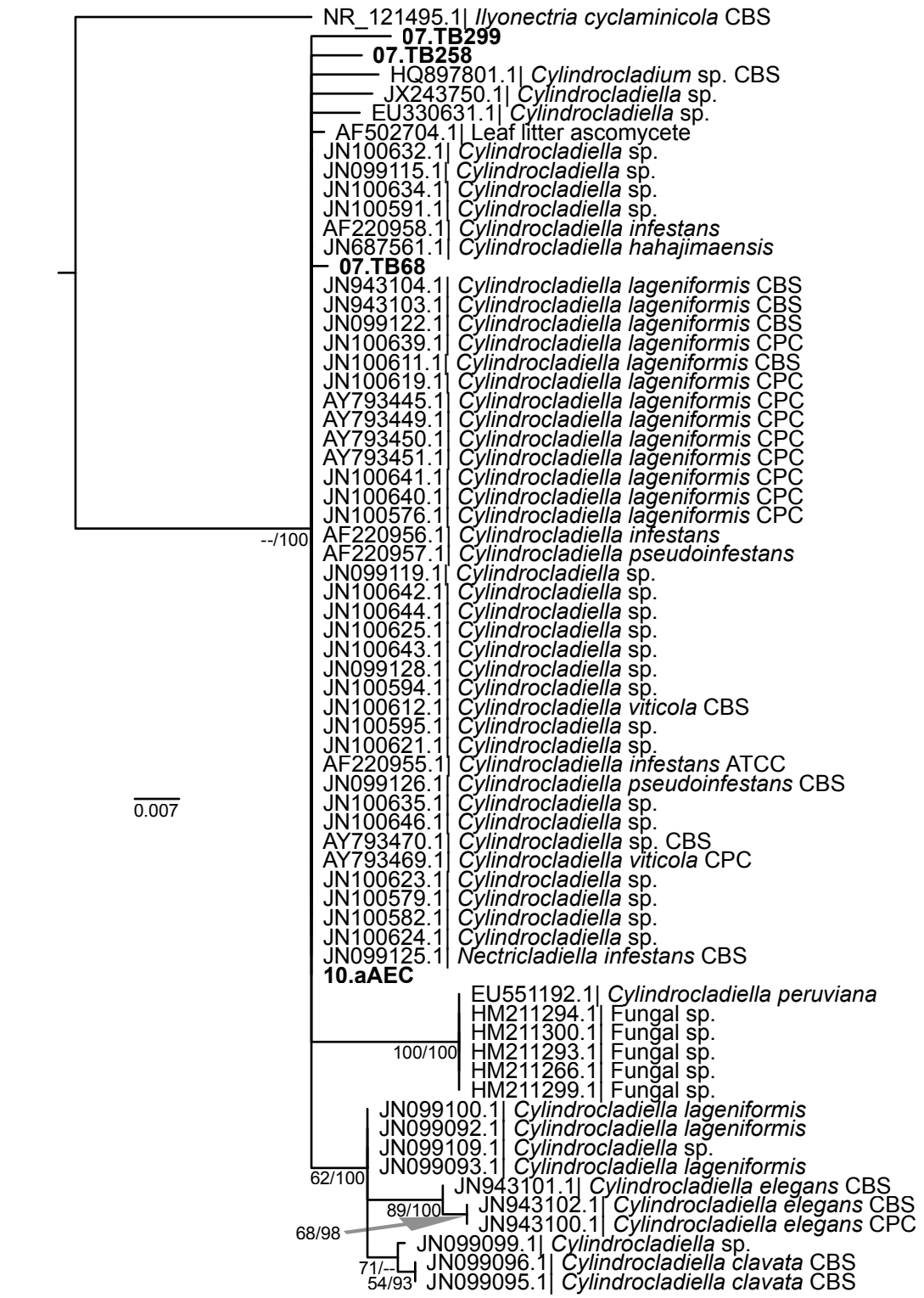
JX244044.1| Neonectria sp.
JX244019.1| Neonectria sp.
KC007323.1| Neonectria sp.
KC007223.1| Neonectria sp.
--/100
100/100
61/83
82/76
100/100
95/100
87/94
87/100
87/93
58/83
56/--
0.02

X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07.TB258
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EU330631.1| Cylindrocladiella sp.
AF502704.1| Leaf litter ascomycete
JN100632.1| Cylindrocladiella sp.
JN099115.1| Cylindrocladiella sp.
JN100634.1| Cylindrocladiella sp.
JN100591.1| Cylindrocladiella sp.
AF220958.1| Cylindrocladiella infestans
JN687561.1| Cylindrocladiella hahajimaensis
07.TB68
JN943104.1| Cylindrocladiella lageniformis CBS
JN943103.1| Cylindrocladiella lageniformis CBS
JN099122.1| Cylindrocladiella lageniformis CBS
JN100639.1| Cylindrocladiella lageniformis CPC
JN100611.1| Cylindrocladiella lageniformis CBS
JN100619.1| Cylindrocladiella lageniformis CPC
AY793445.1| Cylindrocladiella lageniformis CPC
AY793449.1| Cylindrocladiella lageniformis CPC
AY793450.1| Cylindrocladiella lageniformis CPC
AY793451.1| Cylindrocladiella lageniformis CPC
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JN100640.1| Cylindrocladiella lageniformis CPC
JN100576.1| Cylindrocladiella lageniformis CPC
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AF220957.1| Cylindrocladiella pseudoinfestans
JN099119.1| Cylindrocladiella sp.
JN100642.1| Cylindrocladiella sp.
JN100644.1| Cylindrocladiella sp.
JN100625.1| Cylindrocladiella sp.
JN100643.1| Cylindrocladiella sp.
JN099128.1| Cylindrocladiella sp.
JN100594.1| Cylindrocladiella sp.
JN100612.1| Cylindrocladiella viticola CBS
JN100595.1| Cylindrocladiella sp.
JN100621.1| Cylindrocladiella sp.
AF220955.1| Cylindrocladiella infestans ATCC
JN099126.1| Cylindrocladiella pseudoinfestans CBS
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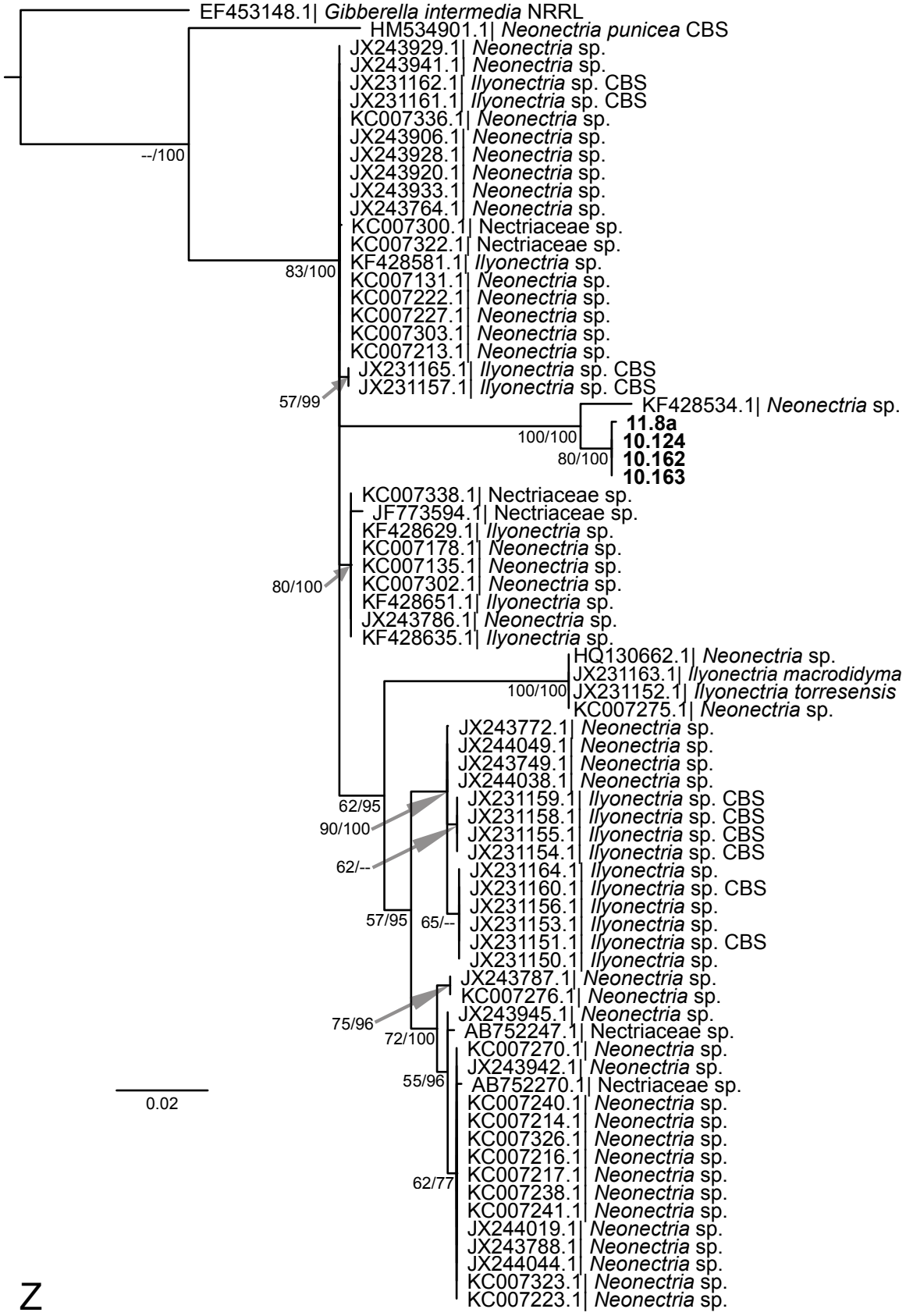
JN100635.1| Cylindrocladiella sp.
JN100646.1| Cylindrocladiella sp.
AY793470.1| Cylindrocladiella sp. CBS
AY793469.1| Cylindrocladiella viticola CPC
JN100623.1| Cylindrocladiella sp.
JN100579.1| Cylindrocladiella sp.
JN100582.1| Cylindrocladiella sp.
JN100624.1| Cylindrocladiella sp.
JN099125.1| Nectricladiella infestans CBS
10.aAEC
EU551192.1| Cylindrocladiella peruviana
HM211294.1| Fungal sp.
HM211300.1| Fungal sp.
100/100
HM211293.1| Fungal sp.
HM211266.1| Fungal sp.
HM211299.1| Fungal sp.
JN099100.1| Cylindrocladiella lageniformis
JN099092.1| Cylindrocladiella lageniformis
JN099109.1| Cylindrocladiella sp.
JN099093.1| Cylindrocladiella lageniformis
62/100
JN943101.1| Cylindrocladiella elegans CBS
89/100
JN943102.1| Cylindrocladiella elegans CBS
68/98
JN943100.1| Cylindrocladiella elegans CPC
JN099099.1| Cylindrocladiella sp.
71/--
JN099096.1| Cylindrocladiella clavata CBS
54/93
JN099095.1| Cylindrocladiella clavata CBS

Y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JX243929.1| Neonectria sp.
JX243941.1| Neonectria sp.
JX231162.1| Ilyonectria sp. CBS
JX231161.1| Ilyonectria sp. CBS
KC007336.1| Neonectria sp.
JX243906.1| Neonectria sp.
JX243928.1| Neonectria sp.
JX243920.1| Neonectria sp.
JX243933.1| Neonectria sp.
JX243764.1| Neonectria sp.
KC007300.1| Nectriaceae sp.
KC007322.1| Nectriaceae sp.
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KC007222.1| Neonectria sp.
KC007227.1| Neonectria sp.
KC007303.1| Neonectria sp.
KC007213.1| Neonectria sp.
JX231165.1| Ilyonectria sp. CBS
JX231157.1| Ilyonectria sp. CBS
KF428534.1| Neonectria sp.
11.8a
10.124
10.162
10.163
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JF773594.1| Nectriaceae sp.
KF428629.1| Ilyonectria sp.
KC007178.1| Neonectria sp.
KC007135.1| Neonectria sp.
KC007302.1| Neonectria sp.
KF428651.1| Ilyonectria sp.
JX243786.1| Neonectria sp.
KF428635.1| Ilyonectria sp.
HQ130662.1| Neonectria sp.
JX231163.1| Ilyonectria macrodidyma
JX231152.1| Ilyonectria torresensis
KC007275.1| Neonectria sp.
JX243772.1| Neonectria sp.
JX244049.1| Neonectria sp.
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JX244038.1| Neonectria sp.
JX231159.1| Ilyonectria sp. CBS
JX231158.1| Ilyonectria sp. CBS
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JX231154.1| Ilyonectria sp. CBS
JX231164.1| Ilyonectria sp.
JX231160.1| Ilyonectria sp. CBS
JX231156.1| Ilyonectria sp.
JX231153.1| Ilyonectria sp.
JX231151.1| Ilyonectria sp. CBS
JX231150.1| Ilyonectria sp.
JX243787.1| Neonectria sp.
KC007276.1| Neonectria sp.
JX243945.1| Neonectria sp.
AB752247.1| Nectriaceae sp.
KC007270.1| Neonectria sp.
JX243942.1| Neonectria sp.
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KC007323.1| Neonectria sp.
KC007223.1| Neonectria sp.
--/100
83/100
57/99
100/100
80/100
80/100
62/95
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62/--
57/95
65/--
75/96
72/100
55/96
62/77
0.02
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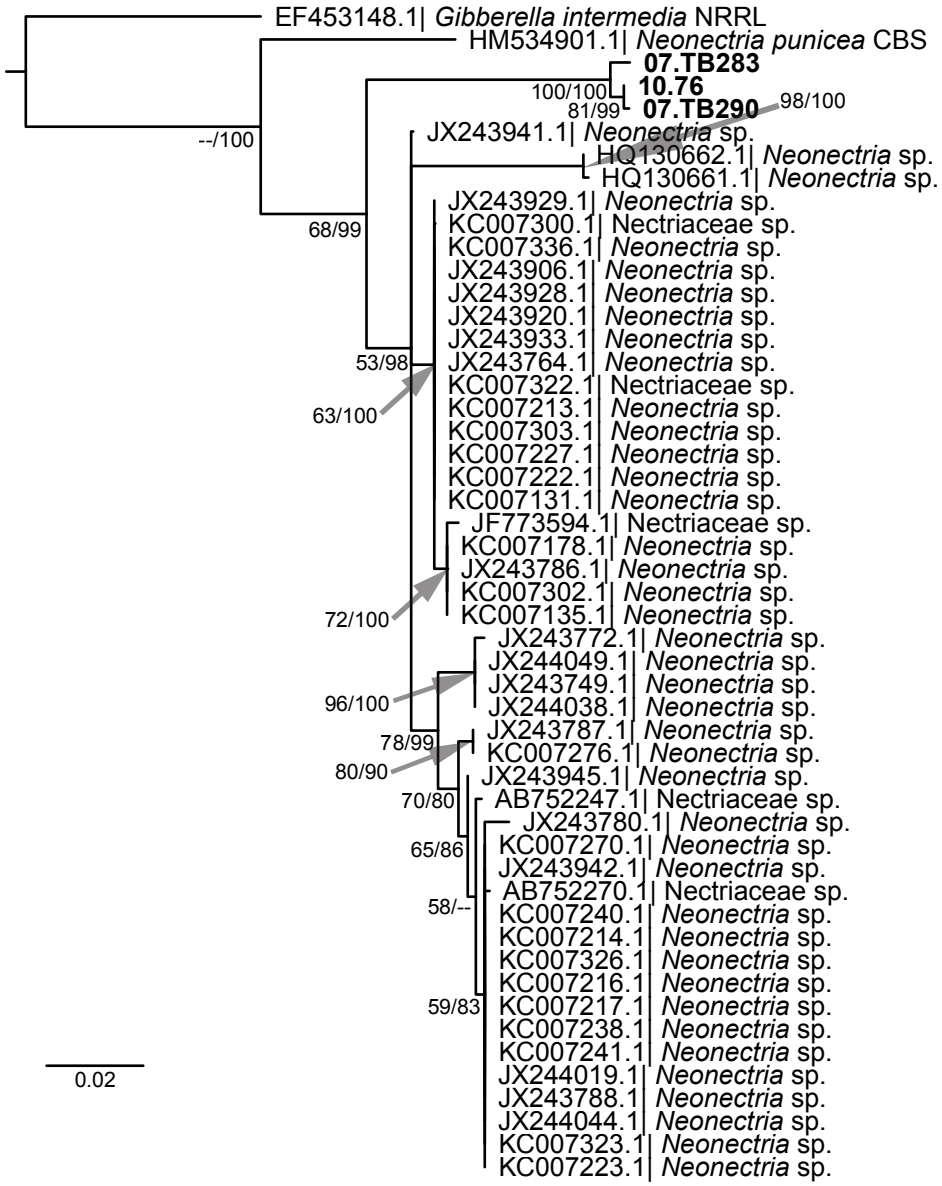
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07.TB290
100/100
81/99
98/100
--/100
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HQ130662.1| Neonectria sp.
HQ130661.1| Neonectria sp.
68/99
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KC007300.1| Nectriaceae sp.
KC007336.1| Neonectria sp.
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JX243928.1| Neonectria sp.
JX243920.1| Neonectria sp.
JX243933.1| Neonectria sp.
53/98
JX243764.1| Neonectria sp.
KC007322.1| Nectriaceae sp.
63/100
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KC007131.1| Neonectria sp.
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KC007302.1| Neonectria sp.
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96/100
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78/99
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KC007276.1| Neonectria sp.
80/90
JX243945.1| Neonectria sp.
70/80
AB752247.1| Nectriaceae sp.
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65/86
KC007270.1| Neonectria sp.
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AB752270.1| Nectriaceae sp.
58/--
KC007240.1| Neonectria sp.
KC007214.1| Neonectria sp.
KC007326.1| Neonectria sp.
KC007216.1| Neonectria sp.
59/83
KC007217.1| Neonectria sp.
KC007238.1| Neonectria sp.
KC007241.1| Neonectria sp.
JX244019.1| Neonectria sp.
JX243788.1| Neonectria sp.
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KC007323.1| Neonectria sp.
KC007223.1| Neonectria sp.
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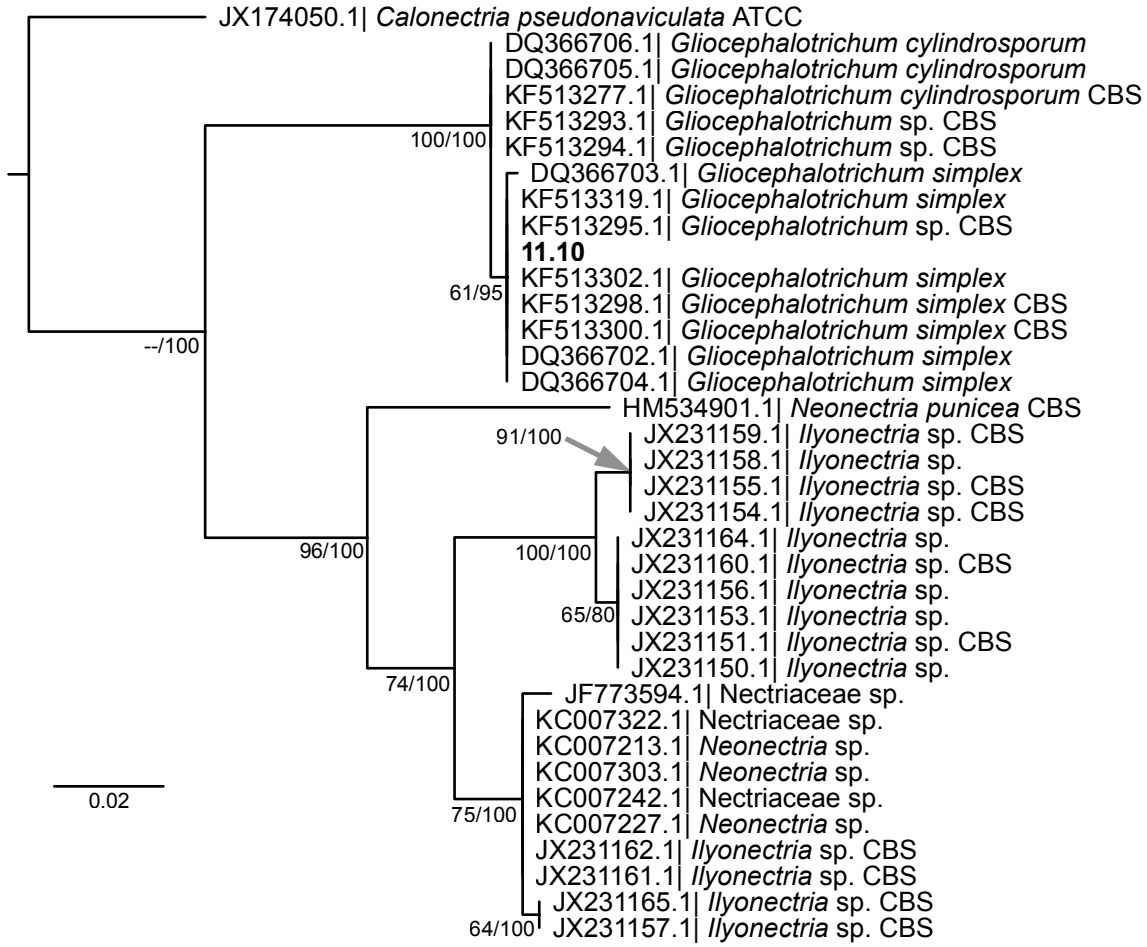
AA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DQ366705.1| Gliocephalotrichum cylindrosporum
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KF513293.1| Gliocephalotrichum sp. CBS
100/100
KF513294.1| Gliocephalotrichum sp. CBS
DQ366703.1| Gliocephalotrichum simplex
KF513319.1| Gliocephalotrichum simplex
KF513295.1| Gliocephalotrichum sp. CBS
11.10
KF513302.1| Gliocephalotrichum simplex
61/95
KF513298.1| Gliocephalotrichum simplex CBS
KF513300.1| Gliocephalotrichum simplex CBS
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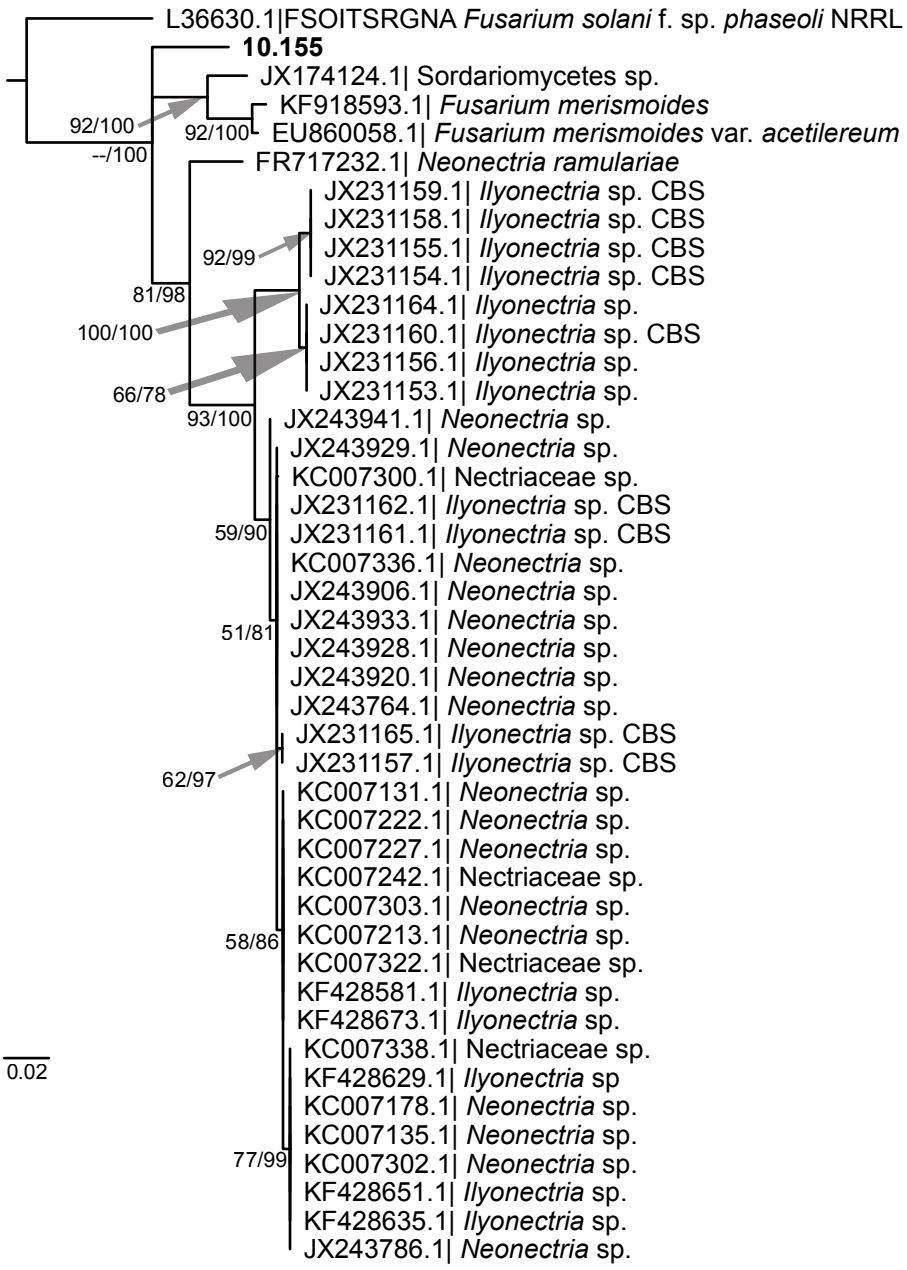
DQ366702.1| Gliocephalotrichum simplex
DQ366704.1| Gliocephalotrichum simplex
HM534901.1| Neonectria punicea CBS
91/100
JX231159.1| Ilyonectria sp. CBS
JX231158.1| Ilyonectria sp.
JX231155.1| Ilyonectria sp. CBS
JX231154.1| Ilyonectria sp. CBS
96/100
JX231164.1| Ilyonectria sp.
100/100
JX231160.1| Ilyonectria sp. CBS
JX231156.1| Ilyonectria sp.
65/80
JX231153.1| Ilyonectria sp.
JX231151.1| Ilyonectria sp. CBS
JX231150.1| Ilyonectria sp.
74/100
JF773594.1| Nectriaceae sp.
KC007322.1| Nectriaceae sp.
KC007213.1| Neonectria sp.
KC007303.1| Neonectria sp.
0.02
KC007242.1| Nectriaceae sp.
75/100
KC007227.1| Neonectria sp.
JX231162.1| Ilyonectria sp. CBS
JX231161.1| Ilyonectria sp. CBS
JX231165.1| Ilyonectria sp. CBS
64/100
JX231157.1| Ilyonectria sp. CBS

AB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JX174124.1| Sordariomycetes sp.
KF918593.1| *Fusarium merismoides*
EU860058.1| *Fusarium merismoides* var. *acetilereum*
FR717232.1| *Neonectria ramulariae*
JX231159.1| *Ilyonectria* sp. CBS
JX231158.1| *Ilyonectria* sp. CBS
JX231155.1| *Ilyonectria* sp. CBS
JX231154.1| *Ilyonectria* sp. CBS
JX231164.1| *Ilyonectria* sp.
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JX231153.1| *Ilyonectria* sp.
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KC007336.1| *Neonectria* sp.
JX243906.1| *Neonectria* sp.
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JX243920.1| *Neonectria* sp.
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JX231165.1| *Ilyonectria* sp. CBS
JX231157.1| *Ilyonectria* sp. CBS
KC007131.1| *Neonectria* sp.
KC007222.1| *Neonectria* sp.
KC007227.1| *Neonectria* sp.
KC007242.1| Nectriaceae sp.
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KC007213.1| *Neonectria* sp.
KC007322.1| Nectriaceae sp.
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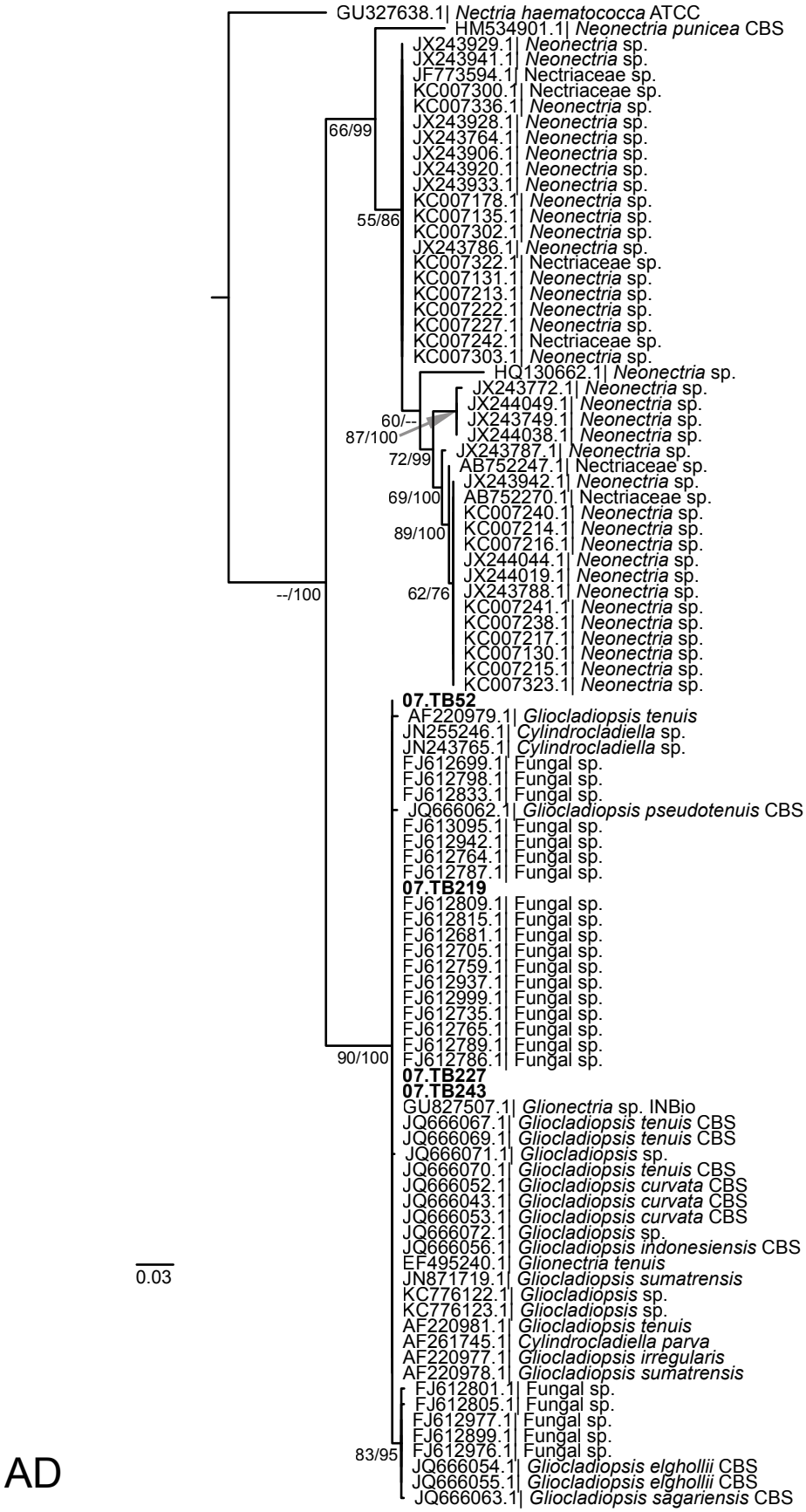
KF428673.1| *Ilyonectria* sp.
KC007338.1| Nectriaceae sp.
KF428629.1| *Ilyonectria* sp
KC007178.1| *Neonectria* sp.
KC007135.1| *Neonectria* sp.
KC007302.1| *Neonectria* sp.
KF428651.1| *Ilyonectria* sp.
KF428635.1| *Ilyonectria* sp.
JX243786.1| *Neonectria* sp.
92/100
--/100
92/100
92/99
81/98
100/100
66/78
93/100
59/90
51/81
62/97
58/86
77/99
0.02

AC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AB752247.1| Nectriaceae sp.
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AB752270.1| Nectriaceae sp.
KC007240.1| Neonectria sp.
KC007214.1| Neonectria sp.
KC007216.1| Neonectria sp.
JX244044.1| Neonectria sp.
JX244019.1| Neonectria sp.
JX243788.1| Neonectria sp.
KC007241.1| Neonectria sp.
KC007238.1| Neonectria sp.
KC007217.1| Neonectria sp.
KC007130.1| Neonectria sp.
KC007215.1| Neonectria sp.
KC007323.1| Neonectria sp.
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JN255246.1| Cylindrocladiella sp.
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FJ612798.1| Fungal sp.
FJ612833.1| Fungal sp.
JQ666062.1| Gliocladiopsis pseudotenuis CBS
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FJ612942.1| Fungal sp.
FJ612764.1| Fungal sp.
FJ612787.1| Fungal sp.
07.TB219
FJ612809.1| Fungal sp.
FJ612815.1| Fungal sp.
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FJ612705.1| Fungal sp.
FJ612759.1| Fungal sp.
FJ612937.1| Fungal sp.
FJ612999.1| Fungal sp.
FJ612735.1| Fungal sp.
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FJ612789.1| Fungal sp.
FJ612786.1| Fungal sp.
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07.TB243
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JQ666067.1| Gliocladiopsis tenuis CBS
JQ666069.1| Gliocladiopsis tenuis CBS
JQ666071.1| Gliocladiopsis sp.
JQ666070.1| Gliocladiopsis tenuis CBS
JQ666052.1| Gliocladiopsis curvata CBS
JQ666043.1| Gliocladiopsis curvata CBS
JQ666053.1| Gliocladiopsis curvata CBS
JQ666072.1| Gliocladiopsis sp.
JQ666056.1| Gliocladiopsis indonesiensis CBS
EF495240.1| Glionectria tenuis
JN871719.1| Gliocladiopsis sumatrensis
KC776122.1| Gliocladiopsis sp.
KC776123.1| Gliocladiopsis sp.
AF220981.1| Gliocladiopsis tenuis
AF261745.1| Cylindrocladiella parva
AF220977.1| Gliocladiopsis irregularis
AF220978.1| Gliocladiopsis sumatrensis
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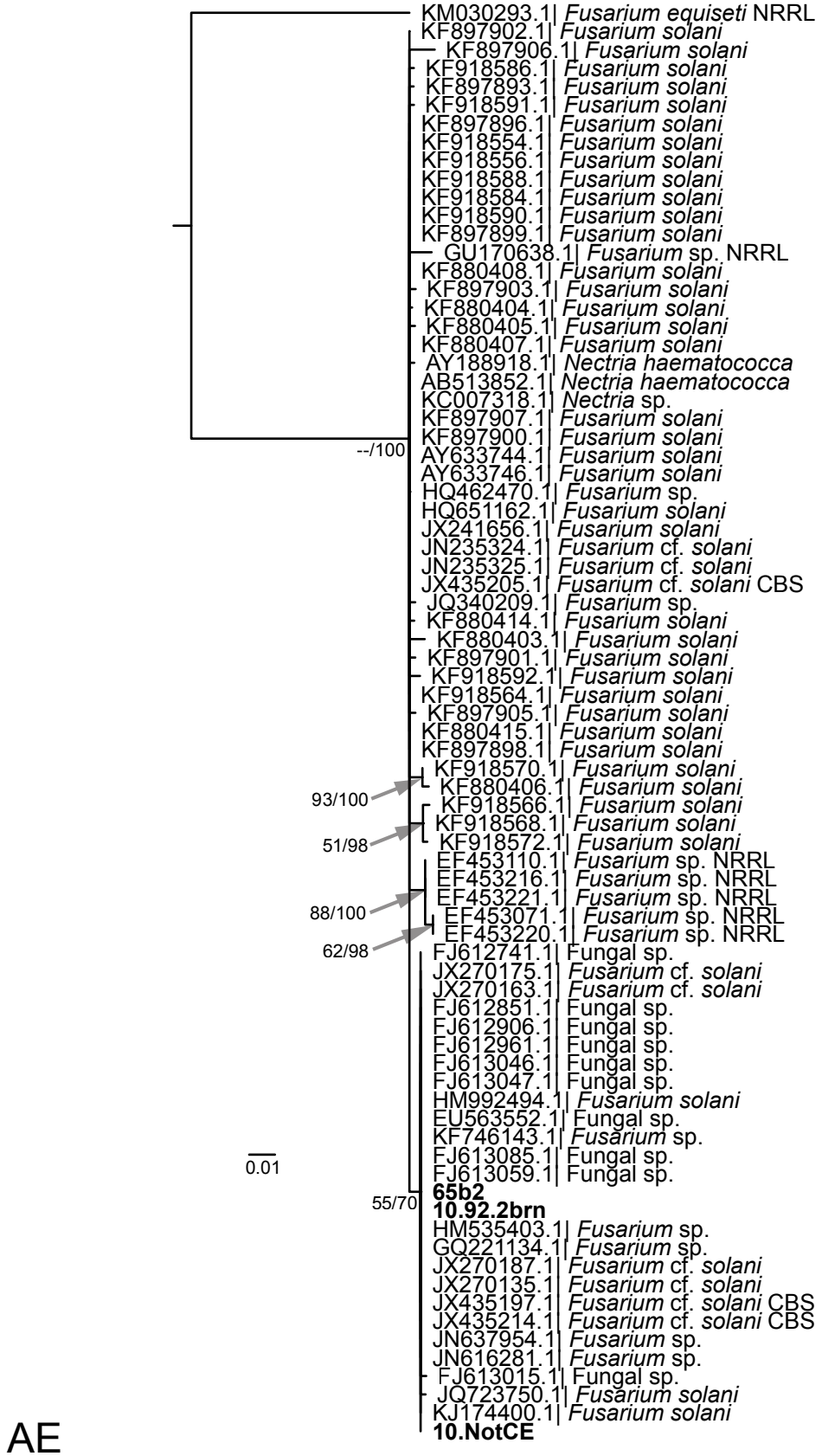
FJ612805.1| Fungal sp.
FJ612977.1| Fungal sp.
FJ612899.1| Fungal sp.
FJ612976.1| Fungal sp.
JQ666054.1| Gliocladiopsis elghollii CBS
JQ666055.1| Gliocladiopsis elghollii CBS
JQ666063.1| Gliocladiopsis sagariensis CBS
66/99
55/86
60/--
87/100
72/99
69/100
89/100
--/100
62/76
90/100
83/95
0.03

AD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KF897899.1| Fusarium solani
GU170638.1| Fusarium sp. NRRL
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KF897900.1| Fusarium solani
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AY633744.1| Fusarium solani
AY633746.1| Fusarium solani
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HQ651162.1| Fusarium solani
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JN235324.1| Fusarium cf. solani
JN235325.1| Fusarium cf. solani
JX435205.1| Fusarium cf. solani CBS
JQ340209.1| Fusarium sp.
KF880414.1| Fusarium solani
KF880403.1| Fusarium solani
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93/100
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51/98
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88/100
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62/98
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FJ613059.1| Fungal sp.
65b2
55/70
10.92.2brn
HM535403.1| Fusarium sp.
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JX435197.1| Fusarium cf. solani CBS
JX435214.1| Fusarium cf. solani CBS
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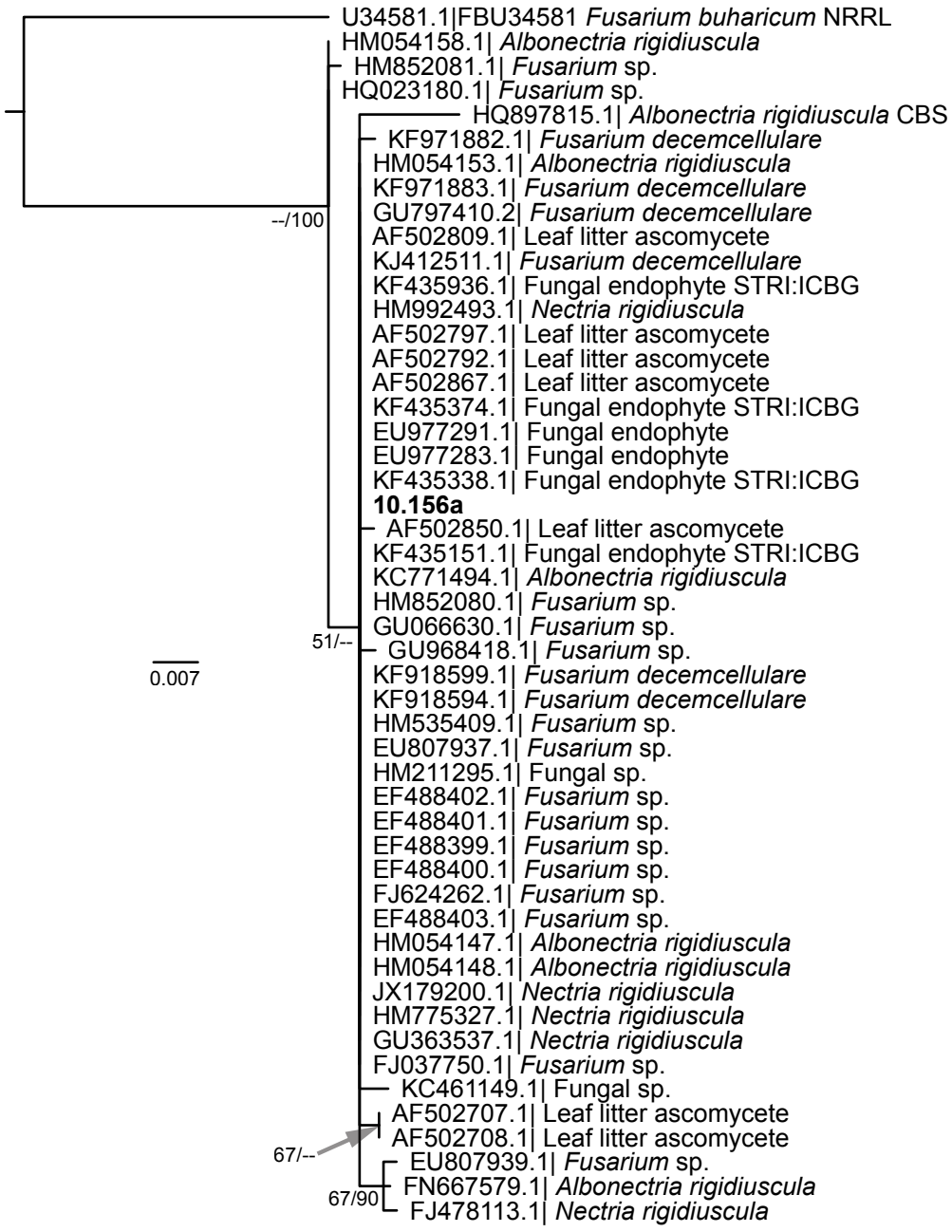
AE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HQ023180.1| *Fusarium* sp.
HQ897815.1| *Albonectria rigidiuscula* CBS
KF971882.1| *Fusarium decemcellulare*
HM054153.1| *Albonectria rigidiuscula*
KF971883.1| *Fusarium decemcellulare*
GU797410.2| *Fusarium decemcellulare*
AF502809.1| Leaf litter ascomycete
KJ412511.1| *Fusarium decemcellulare*
KF435936.1| Fungal endophyte STRI:ICBG
HM992493.1| *Nectria rigidiuscula*
AF502797.1| Leaf litter ascomycete
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AF502867.1| Leaf litter ascomycete
KF435374.1| Fungal endophyte STRI:ICBG
EU977291.1| Fungal endophyte
EU977283.1| Fungal endophyte
KF435338.1| Fungal endophyte STRI:ICBG
10.156a
AF502850.1| Leaf litter ascomycete
KF435151.1| Fungal endophyte STRI:ICBG
KC771494.1| *Albonectria rigidiuscula*
HM852080.1| *Fusarium* sp.
GU066630.1| *Fusarium* sp.
GU968418.1| *Fusarium* sp.
KF918599.1| *Fusarium decemcellulare*
KF918594.1| *Fusarium decemcellulare*
HM535409.1| *Fusarium* sp.
EU807937.1| *Fusarium* sp.
HM211295.1| Fungal sp.
EF488402.1| *Fusarium* sp.
EF488401.1| *Fusarium* sp.
EF488399.1| *Fusarium* sp.
EF488400.1| *Fusarium* sp.
FJ624262.1| *Fusarium* sp.
EF488403.1| *Fusarium* sp.
HM054147.1| *Albonectria rigidiuscula*
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JX179200.1| *Nectria rigidiuscula*
HM775327.1| *Nectria rigidiuscula*
GU363537.1| *Nectria rigidiuscula*
FJ037750.1| *Fusarium* sp.
KC461149.1| Fungal sp.
AF502707.1| Leaf litter ascomycete
AF502708.1| Leaf litter ascomycete
EU807939.1| *Fusarium* sp.
FN667579.1| *Albonectria rigidiuscula*
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51/--
67/--
67/90
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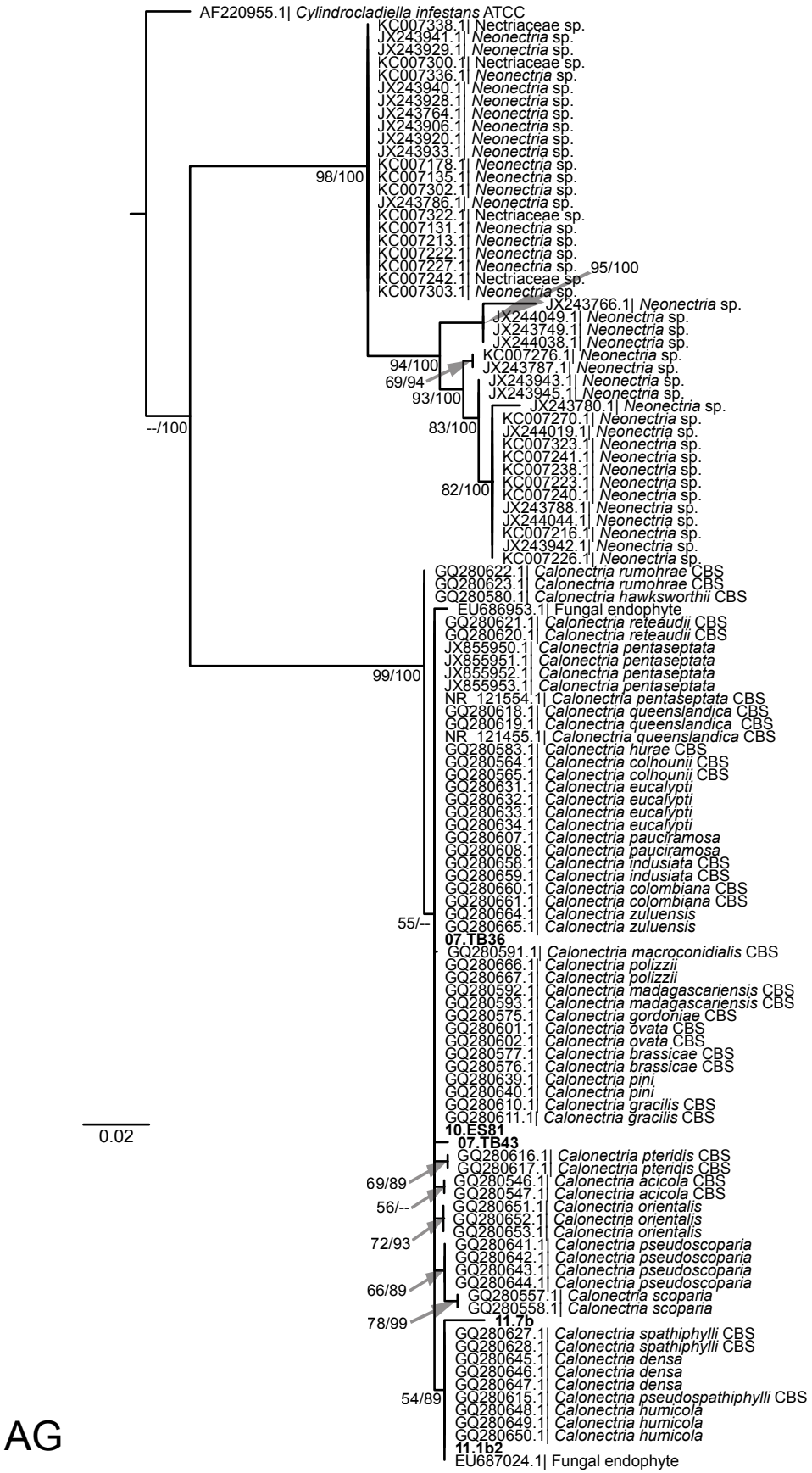
AF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98/100
95/100
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94/100
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69/94
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93/100
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83/100
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82/100
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55/--
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56/--
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GQ280644.1| Calonectria pseudoscoparia
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GQ280557.1| Calonectria scoparia
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GQ280628.1| Calonectria spathiphylli CBS
GQ280645.1| Calonectria densa
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54/89
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11.1b2
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AG