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The effects of plant pathogens on tree recruitment in the Western Amazon under a projected future climate: a dynamical systems analysis

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Summary

1. Climate change predictions in the Amazon have largely focused on carbon–water relations, while the impacts of increased air temperature and reduced precipitation on host–pathogen relationships have not been extensively explored. These relationships are known to affect recruitment of many Amazonian plant species.

2. Host–pathogen relationships are well suited to a dynamical analysis of the effects of climate change due to the direct linkages between pathogen behaviour and abiotic factors such as temperature and rainfall.

3. Seedlings of the palm *Iriartea deltoidea* experience significant mortality due to infection by the fungus *Diplodia mutila*. This host–pathogen interaction was examined by combining a semi-analytical model with field data illustrating the temperature sensitivity of *D. mutila* reproductive rates and *I. deltoidea* seedling mortality in response to infection.

4. The data–model combination shows that projected climatic shifts in rainfall and temperature for the Amazon region will tend to reduce recruitment by altering pathogen activity and reducing palm fecundity. The magnitude of the reduction is sensitive to the details of the epidemiology of the *D. mutila–I. deltoidea* host–pathogen system, and ranges from 10% to 56% under plausible scenarios.

5. Although considerable uncertainty remains, the proposed model provides a blueprint for research on one aspect of ecosystem change in future climate models.

6. *Synthesis*. The study illustrates the potential for ecosystem responses to climate change, which can be investigated through tractable models simple enough to assimilate into climate modelling frameworks. Particular environmental sensitivities in fungal dynamics are identified. The implications of combined plant physiological stress and enhanced pathogenic activity under future climate scenarios are highlighted as critical issues for projecting forest response.

Key-words: Amazon, climate change, *Diplodia mutila*, *Iriartea deltoidea*, Janzen–Connell, plant–climate interactions, plant pathogens, spatial pattern, tree recruitment

Introduction

Models of climate change across the Amazon indicate that during the 21st century, air temperatures will rise by 3–8 °C and that dry-season rainfall will decline by 20%. The implications of these climatic changes on Amazonian ecosystems are usually assessed in terms of plant physiology and carbon–water relations, highlighting the risk of Amazonian 'dieback' due to drought stress (Betts *et al.* 2004), lower net primary production (Cavaleri, Oberbauer & Ryan 2008), increased fire frequency (Cochrane & Barber 2009) and forest fragmentation (Broadbent *et al.* 2008). Although Amazonian dieback predictions appear to be robust, considerable uncertainties about the response of the ecosystem to climate change remain (Betts, Malhi & Roberts 2008; Huntingford *et al.* 2008). The net effects and complex feedbacks between deep-rooting systems, the role of carbon fertilization, nutrient dynamics and plant acclimation to higher air temperatures and CO_2 are not well understood (Betts, Malhi & Roberts 2008). Furthermore, feedbacks between climate change and ecosystem dynamics are likely to have large implications for the ecosystem's response but are not readily captured by current models (Lloyd & Farquhar 2008).

One understudied ecosystem–climate feedback in the tropics is an alteration in the activity and virulence of plant pathogens

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in response to increasing temperatures. Climatic factors constitute a primary control on plant disease (Agrios 2005), but there are relatively few studies of how climate change could influence pathogens in agricultural or natural systems, particularly in the tropics (Chakraborty, Tiedemann & Teng 2000; Boland *et al*. 2004; Garrett *et al*. 2006; Ibanez *et al*. 2006). Climate change is likely to shift the geographical distributions of pathogens, to alter host resistance and to change pathogen virulence, pathogenicity and fecundity (Coakley, Scherm & Chakraborty 1999; Chakraborty, Tiedemann & Teng 2000; Boland *et al*. 2004; Garrett *et al*. 2006; Ibanez *et al*. 2006). Air temperature governs aspects of the epidemiology of many pathogens, and many pathogens exhibit an 'ideal temperature range'. For instance, in the case of *Diplodia mutila* (the pathogen considered in this paper), *in vitro* studies indicate optimal growing temperatures above 25 °C, but dramatic reductions in growth rate once temperatures increase to 35 °C (Sanchez *et al*. 2003; Copes & Hendrix 2004).

In rain forest ecosystems, pathogenic infection can result in the death of most seeds or seedlings of several plant species, placing strong constraints on recruitment (Augspurger & Kelly 1984; Bell, Freckleton & Lewis 2006; Gallery, Dalling & Arnold 2007). Plant fungal pathogens are generally not subject to top-down control from predators in a food web but are regulated by environmental factors and host availability (Agrios 2005). Consequently, their dynamics may respond sensitively to small shifts in ambient environmental conditions (Maxwell, Kruger & Stanosz 1997; Tapsoba & Wilson 1997; Mayek-Perez, Acosta-Gallegos & Lopez-Castaneda 2002; Waugh *et al*. 2003). Given the importance of pathogens in regulating plant recruitment in the Amazon, we hypothesize that such changes in fungal abundance or activity may constitute an unquantified ecosystem response to climate change.

We examined the magnitude of climatic effects have on seedling–pathogen interactions, using the subtropical palm *Iriartea deltoidea* as an example. *Iriartea deltoidea* is a dominant tree species in wet lowland and pre-montane tropical forests of western Amazonia and the Chocó and Central American region (Wattenberg & Breckle 1995; Clark, Palmer & Clark 1999; Pitman *et al*. 2001; Macia & Svenning 2005). Under current climatic conditions, fungi infect 50–70% of *I. deltoidea* seedlings, and cause 10–15% of recorded seedling mortality (Alvarez-Loayza 2009). Data regarding the disease cycles of all fungal pathogens infecting *I. deltoidea* seedlings are not available. Consequently, a dominant endophyte and pathogen of *I. deltoidea* seedlings and adults, *D. mutila* (synonyms include *Botryosphaeria stevensii* Shoemaker; *Stenocarpella macrospora*; Botryosphaeriaceae; Botryosphaeriales; Ascomycota), was treated as a 'model pathogen' on which to base the case study. Based on field surveys undertaken at Cocha Cashu Biological Station (CCBS) in the Western Amazon, *D. mutila* infects *c*. 20% of *I. deltoidea* seedlings and 85% of adults (Alvarez-Loayza 2009). *Diplodia mutila* affects another 10 species in the Western Amazon from diverse plant families and the pathogen has a global distribution (Alvarez-Loayza 2009; Farr & Rossman 2010). *Diplodia mutila* causes mortality in 6% of *I. deltoidea* seedlings, but in adults is typically found as an endophyte (Alvarez-Loayza 2009). Endophytic fungi that asymptomatically colonize plants (Petrini 1986) are diverse and abundant in tropical ecosystems (Arnold *et al*. 2000). These organisms can be weakly pathogenic (Schulz & Boyle 2005) and/or mutualistic, frequently enabling plants to adapt to extreme environments (Rodriguez, Redman & Henson 2004). *Diplodia mutila* can be an endophyte or a pathogen of *I. deltoidea*, depending on abiotic and biotic stress and the age of the plant. However, its pathogenicity and growth rates are likely to increase with rising temperatures (Sanchez *et al*. 2003; Alvarez-Loayza 2009).

We adapted a theoretical framework proposed by Nathan & Casagrandi (2004) to synthesize the effects of *D. mutila* on the recruitment of *I. deltoidea*. The interaction of pathogens with seedling recruitment dynamics is complicated by the dependence of seed dispersal, pathogen attack and herbivore activity on space and seedling density. Nathan & Casagrandi (NC) provided a framework to account for these processes within a simple spatially explicit model. We adapted the NC framework and revised it to apply to *I. deltoidea* by incorporating the effects of fungi, specifically *D. mutila,* as informed by available literature, observations and data. Climatic sensitivity within the NC model was then considered in terms of pathogen dependence on temperature availability, and seed production dependence on rainfall availability.

Although the *I. deltoidea–D. mutila* host–pathogen pair was used as a case study to illustrate the potential implications of climate change on fungal pathogens in Amazonia, there is a huge spectrum of plant–pathogen interactions with variable sensitivities to temperature and precipitation shifts. Resolving the diversity of all these interactions and their trajectories under variable climatic drivers is well beyond the scope of a single study. The aim of this study was to develop a modelling framework suitable for upscaling interactions that pertain to survival of tree recruits, incorporating the effects of abiotic shifts, and illustrating the potential magnitude of such 'ecosystem-driven' responses. Indeed, even within the deliberately simple framework adopted here, the challenges of linking observed shifts in pathogen behaviour under controlled conditions to the likely implications at the ecosystem level remain formidable and illuminate ongoing challenges in data collection and observation. Ultimately, one of the aims of this approach was to illustrate the potential to couple ecological approaches to future generations of climate models, and this aim has motivated an intentional simplicity in the modelling framework.

Materials and methods

FIELD DATA

The research was conducted at two field sites in lowland tropical forests in Peru: Cocha Cashu Biological Station (CCBS), Manu National Park (11°54′ S, 71°22′ W, elevation *c*. 350 m a.s.l.) and Los Amigos Biological Station (LABS) (12°34′07″ S, 70°05′57″ W, elevation *c*. 268 m a.s.l.) (Terborgh 1983; Gentry 1990; Pitman 2009). Ten plots were established in primary floodplain forest, each with similar floristic composition and topographic characteristics. Five plots were located at CCBS and five at LABS. Nine plots measured 900 m^2 and

one plot located at CCBS measured 2.25 ha. In each plot all *I. deltoidea* plants were tagged and mapped in a Cartesian coordinate system. The height of the tallest leaf (cm), the number of leaves, colour of leaves, diseases on the leaves and stem and percentage of disease in the affected leaves were recorded for all *I. deltoidea* plants < 6 m tall. A total of 1068 *I. deltoidea* plants were mapped in the first census (March 2007). Four age classes were defined based on leaf morphology and ease of accessibility for the evaluation of diseases: (i) young seedlings, (ii) old seedlings, (iii) juveniles and adults and (iv) fruiting trees. Young seedlings had one or two simple round leaves measuring < 25 cm. Remnants of the seed were still attached to the newly germinated seedling. Old seedlings had two or more simple or compound leaves but measured < 50 cm tall. Old and new seedlings were distinguished by the presence of remnants of the seed on young seedlings and the presence of algae and epifoliar fungi on old ones.

Sixty-three adults produced fruit between December 2006 and March 2007. Three hundred and seventeen seedlings were considered young at the time of the first census, another 491 old. One hundred and ninety-seven trees were considered juveniles and adults (non-fruiting). The remaining 63 trees were the fruiting adults. Common pathogens other than *D. mutila* and insect predators affecting *I. deltoidea* are identified and discussed elsewhere (Alvarez-Loayza 2009).

TRANSPLANT EXPERIMENTS

Transplant experiments were used to assess the effect of air temperature and radiance on plant infection by *D. mutila*. In these experiments, 30 seedlings were transplanted from one plot at CCBS where adults, juveniles, seeds and fruits were colonized by *D. mutila*. Ten seedlings were transplanted into the field under shade conditions (light intensity of *c.* 55 ± 15 μmol m^{-2} s^{-1}), 10 in a shaded glasshouse (light intensity of *c.* 491 ± 34 μmol m^{-2} s^{-1}) and 10 under full sun (light intensity of *c.* 1058 ± 23 μmol m^{-2} s^{-1}). All seedlings had two leaves and did not have any visible disease symptoms produced by *D. mutila* or any other foliar spot when transplanted, but were subsequently inoculated with *D. mutila*. The seedlings were the same age, derived from the same parental tree and same seed cohort, were planted in the same soil type and were exposed to the same watering conditions. Variability of infection is associated with different ages, different parental trees, and different soils, so this design provided a highly controlled experiment. Light availability was measured three times per day (6 AM, 12 PM and 5 PM) for a period of 10 days and all disease symptoms and insect damage were recorded and measured daily. The average daily temperature in understorey and full sun conditions was 23 °C (± 3), and 28 °C (± 5) in the glasshouse. These temperature ranges correspond to the predictions of the Hadley Centre's climate projections for 2070–2100 from the HadCM3 IS92a model.

IMPACTS OF CLIMATE CHANGE

Field and glasshouse results from Alvarez-Loayza (2009) and Terborgh (unpublished data) were used to explore temperature and precipitation effects on model parameters. We considered the impact of a 5 °C increase in air temperature from the baseline ambient temperature of 23 °C at CCBS, and a 20% decline in rainfall. These climatic and hydrologic alterations are based on current Hadley Centre predictions for air temperature and a consensus position of recent IPCC models for the Western Amazon in the case of precipitation (Betts, Malhi & Roberts 2008). The data were used to obtain proportional changes in measured parameters associated with such temperature and precipitation changes. These proportional changes were then applied to the model parameters. We considered three parameters describing pathogen behaviour that were considered likely to respond to climate change: the production of conidia, the virulence of fungi, i.e. the rate at which they induced mortality in palm seedlings, and the fecundity of the palms.

The data from the glasshouse experiments and field measurements used to assess the climate sensitivity of these parameters are provided in Fig. 1. No data were available to relate precipitation rates to the pathogen dynamics, but rainfall could be assessed against *Iriartea* fecundity. Based on 6 years of sampling at CCBC, a near linear relationship between rainfall and fecundity of *I. deltoidea* held with a slope of 0.12 with the 95% confidence limits on this slope bounded by 0 and 0.3. Based on this relationship, a 20% decline in rainfall, as predicted by *c.* 70% of models used by the IPCC for the Amazon region (Betts, Malhi & Roberts 2008), would result in a 15% (95% confidence intervals (CIs) 0–20%) decline in fecundity. Similar results were found over a 3-year period by Martinez-Ramos, Anten & Ackerly (2009) when studying the Central American understorey palm species *Chamaedorea elegans*, for which total fruit production dropped by a factor of 40–50% during El Nino Southern Oscillation events, which are associated with drought and heat stress.

Mortality rates due to fungal predation were measured in the glasshouse experiment over a 12-day period at 23 and 28 °C and shown to approximately double with the increased air temperature. Given the relatively small data set, the 95% CI on this proportional increase ranged from a factor of 1.3 to a factor of 5.4.

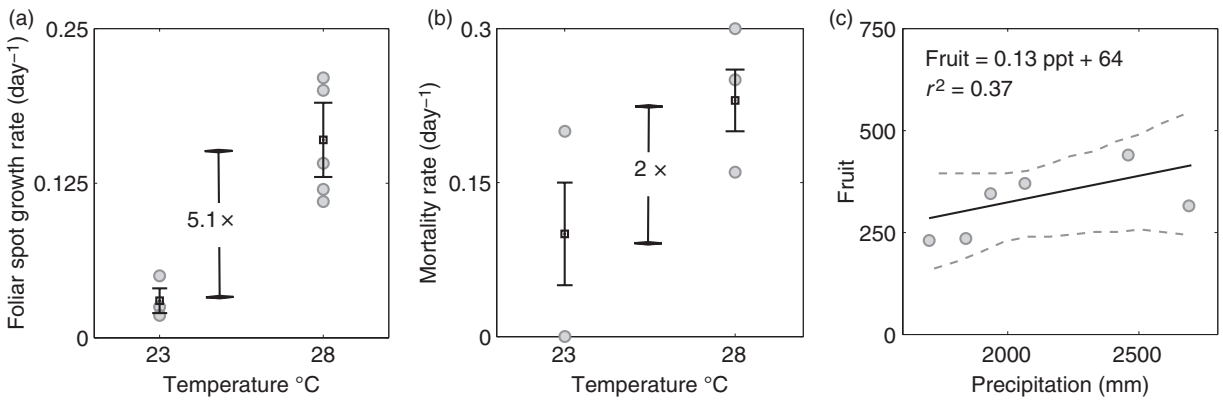

Fig. 1. (a) Impact of a 5 °C air temperature rise on *Diplodia mutila* foliar spot growth rate. Grey circles indicate measured values, black squares the means. (b) Effect of a 5 °C air temperature rise on mortality rates of infected palms. (c) Linear trend in fecundity (mean fruit per *Iriartea deltoidea* tree) with precipitation (mm) and the linear fit to the data.

Conidia densities were measured as a function of foliar spot diameter in a replicated glasshouse experiment ($n = 42$). For each foliar spot, the measured conidia densities (and the spot area) increased with time. At a given time, conidia densities were uncorrelated to the spot size, suggesting that the total rate of conidia production is best represented as the rate of foliar spot growth (see Fig. 2). Mean conidia density increased with temperature from an average of 40 conidia cm^{-2} at 23 °C to an average of 58 conidia cm^{-2} at 28 °C (see Fig. 2). These conidia densities were measured after 4 days of infection: after this time, the least stressed plants (23 °C) abscised their leaves, preventing further damage to the plant. Under higher temperature conditions, conidia densities continued to increase to over 200 conidia cm^{-2} in some cases. In addition to increases in conidia density, the rate at which foliar spots grew at the elevated temperatures increased by a factor of 5 (95% CI 3.1–7.7), or from an average growth rate of 0.03 to 1.6 per day (Fig. 1).

MODEL DEVELOPMENT

We developed an extension to the Nathan & Casagrandi (2004) seedling distribution model (hereafter the NC model) by incorporating two new features: (i) pathogen impacts and (ii) long-distance dispersal of seed, which is common to many tropical species and specifically to *I. deltoidea* (Losos 1995). The modified model was parameterized by fitting it to the CCBS and LABS field data documenting the location and health of seedlings of *I. deltoidea* dispersed over the course of a reproductive season. The impact of climate change on *I. deltoidea* and *D. mutila* seedling–pathogen dynamics was incorporated into the model by relating measured effects to the NC model parameters as described in the previous section. The model was re-run using these parameters, and estimates of seedling distributions and impacts on recruitment were made.

The Nathan and Casagrandi (NC) model

The NC model interprets observed patterns of seedling distribution as the steady state of the interacting processes of seed dispersal and germination, random mortality of the growing seedlings and mortality due to predation. That is:

$$\frac{\partial S(x,t)}{\partial t} = \varphi(x) - \mu\, S(x) - N(x)\ \psi(S,x) \qquad \text{eqn 1}$$

where S (seedlings m^{-2}) is the density of seedlings, ϕ (seedlings m^{-2} day^{-1}) is a function describing seedling production rates through space (i.e. a seed dispersal kernel multiplying germination probabilities), μ (1 day^{-1}) is the rate at which 'random' mortality agents impact seedlings, N (predators m^{-2}) is the density of predators, ψ (seedlings predator^{-1} day^{-1}) is a specific predation rate, x (m) is a radial distance from the parent tree and t (days) is time. Over the course of a reproductive season, the balance of seed production and mortality may be assumed to reach a steady state, allowing the distribution of surviving seedlings to be predicted only as a function of the dispersal and mortality parameters by setting $\partial S/\partial t = 0$. The model is then applied in a radial sense under the idealized assumption that dispersal and predation lack any preferential direction with respect to fruiting trees. The NC model takes ϕ to be an exponentially declining function:

$$\varphi = \frac{2\alpha}{\pi D^2}\exp\left(-\frac{2x}{D}\right), \qquad \text{eqn 2}$$

where α (seedlings day^{-1}) represents the product of fecundity and the germination rate and D (m) is the mean distance a population of seeds travel from the parent tree. Unlike agents contributing to random mortality, predators were assumed to actively seek out seedlings, so N also varies exponentially in space:

$$N = \frac{2\beta}{\pi q^2}\exp\left(\frac{-2x}{q}\right), \qquad \text{eqn 3}$$

where β is the abundance of predators and q is the mean distance from the parent tree at which predators are located. Nathan and Casagrandi assumed that the specific predation rate followed a Holling (1959) Type II function, commonly employed to describe plant–herbivore interactions; such that

$$\psi(x) = \frac{a\ S(x)}{1 + a\ T_h S(x)}, \qquad \text{eqn 4}$$

where a (seedlings predator^{-1} day^{-1}) may be interpreted as a spatial predation rate and T_h is a 'handling time' describing the timescale on which a herbivore interacts with a plant (day seed-

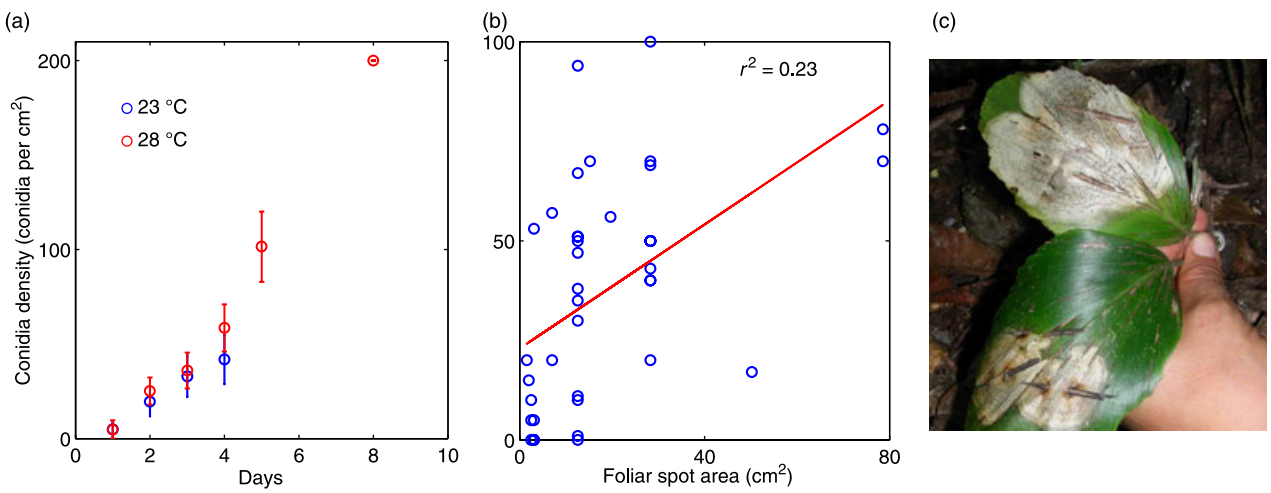

Fig. 2. Conidia density as a function of temperature and foliar spot area, for *Iriartea deltoidea* seedlings infected with *Diplodia mutila* as recorded in a glasshouse experiment ($n = 42$). As shown in (a) the mean density of conidia on the surface of foliar spots increases as the spots develop over time and does so at a more rapid rate at elevated temperature. However, at any given time, no clear correlation could be ascertained between spot area and conidia density, suggesting that the total rate of conidia production is most strongly determined by the growth of the foliar spot (b). The data were generated from inoculation studies (c), the black dots within each foliar spot are the pycnidia, which shed conidia.

ling^{-1}). Here, the family of Holling functions $y(p, \theta)$of type p have a mathematical form $y(p, \theta) = \theta^{(p-1)}/(1 + \theta^{p-1})$. Nathan and Casagrandi solved this model numerically, but analytical solutions for the steady state case do exist given these particular parameterizations of ϕ, N and ψ (and for many other ecologically meaningful spatio-temporal descriptors of the behaviour of seedlings and their predators) (Nathan & Casagrandi 2004).

Modifications to the NC model

Two modifications were made to the NC model. First, the dispersal term was changed from an exponential distribution to a 'fat-tailed' distribution that can account for long-distance dispersal. We adopted a Wald distribution that multiplicatively combines exponential-type behaviour with a power-law tail (Katul *et al.* 2005). The Wald distribution is parameterized with mean dispersal distance D and a shape parameter λ that determines the relative mass contained in the tail of the distribution. This adaptation allows the known long-distance dispersal of *I. deltoidea* seeds to be accounted for in the model (Sezen, Chazdon & Holsinger 2007).

In principle, the NC model may be extended *ad infinitum* to account for multiple predator behaviours by adding additional mortality terms. It is more parsimonious, however, to group predators by type and functional response (Alvarez-Loayza 2009). Field data suggest that some 35% of *I. deltoidea* seedlings are currently killed by insect herbivores, motivating the retention of both insects and fungi in the proposed reformulation of the model. To account for both insect herbivores and fungal pathogens, which have substantially different abundance and behaviour, the original NC model is now modified as:

$$\frac{\partial S(x,t)}{\partial t} = \varphi(\mathrm{x}) - \mu S(x,t) - N_\mathrm{i}(x)\psi_\mathrm{i}(S,x) - N_\mathrm{f}(x)\psi_\mathrm{f}(S,x), \quad \text{eqn 5}$$

where the subscripts i and f refer to insects and fungi, respectively.

The representation of insect herbivory by an exponential spatial distribution has been confirmed by coupling seed distributions and generalized foraging models for insects (Mari *et al.* 2008). The use of a Holling Type II specific predation rate was maintained as in the original NC approach, such that N_i and ψ_i followed the definitions in eqns (3 and 4). However, the primary departure from NC is in the addition of fungal dynamics.

DIPLODIA MUTILA DYNAMICS

The development of a conceptual model of *D. mutila* dynamics at CCBS is based on field observations and data collected by and described in Alvarez-Loayza (2009). The disease cycle of *D. mutila* at the CCBS site commences with conidia production by endophytic fungi colonizing adult *I. deltoidea* and other canopy host species. Rain and aerial dispersal from the upper canopy transport the conidia into the subcanopy where they infect *I. deltoidea* seedlings. Given the abundance of adult *I. deltoidea* at the study sites, this source of primary inoculant (from adult endophytes) is approximately uniform in space. Disease prevalence, as measured by the percentage of seedlings with fungal foliar spots, showed no trend with distance.

Diplodia mutila infections proceed with the development of necrotic foliar spots that produce conidia as they grow. Production of conidia in the subcanopy creates the capacity for secondary infection of seedlings, if the seedling density is sufficiently high. Such polycyclic behaviour is known to occur in *D. mutila* (Brown 1970; Srivastava 1972; Lo & Clark 1988; Olatinwo *et al.* 1999) and adds a non-uniform component to the fungal population density. Polycyclic infections can produce more than a 10-fold increase of inoculum with each disease cycle, generating epidemics (Agrios 2005). Conceptually, the density of *D. mutila* inoculant (N_f) can be considered to follow its own evolution equation, increasing with increased foliar spot area and decreasing at some net removal rate that accounts for settling and degradation. This budget yields:

$$\frac{\partial N_\mathrm{f}}{\partial t} = E + \rho_\mathrm{c}(r_1 - r_2)S_\mathrm{f}(S,x) - dN_\mathrm{f}, \quad \text{eqn 6}$$

where E is the assumed uniform input of primary inoculant from canopy endophytes, r_1 is the rate of growth of foliar spots, r_2 is the rate at which the surface area of foliar spots ceases conidia production, ρ_c is the density of conidia production per area of foliar spot per seedling, S_f is the density of infected seedlings, itself a function of space and the local seedling density, while d is the rate at which the dispersed fungal inoculant degrades or becomes immobilized.

Full parameterization of this evolution equation is not possible with existing data for *D. mutila*. However, some observations can be made regardless of the connection to ψ_f (the primary thrust of the work here). The first is that over seasonal timescales, we would expect N_f to be pseudo-steady and approximately uniform in space with a mean value we term $b_\mathrm{f} = E/d$ as inferred from eqn 6. Although we cannot explicitly test this assumption given the lack of data of conidia shedding from the canopy, we computed the rate of change of infections during the 3-month period studied and found it to be relatively low (< 15% per month), and that the spatial distribution of these infections remains essentially unchanged in time. The consequence of this steady state is that the rate of inoculum shedding by the upper canopy and the removal processes parameterized by d approximately balance each other out, and r_1–r_2 is assumed small. At steady state, we can also approximate:

$$S_\mathrm{f} \approx T\psi_\mathrm{f}N_\mathrm{f} \quad \text{eqn 7}$$

which implies that for low levels of secondary infection,

$$\left(\frac{\psi_\mathrm{f}}{S_\mathrm{f}}\right) \approx \frac{d}{TE}, \quad \text{eqn 8}$$

where T is a timescale associated with mortality.

This approximation accounts reasonably for primary infection (i.e. a constant rate of infection) but becomes less reasonable if the contribution of conidia from infected seedlings becomes non-negligible. If this is the case, then at steady state, the expression takes the form:

$$\left(\frac{\psi_\mathrm{f}}{S_\mathrm{f}}\right) \approx \frac{d}{T\rho_\mathrm{c}(r_1 - r_2)} \frac{1}{\left(\frac{E}{\rho_\mathrm{c}(r_1 - r_2)} + S_\mathrm{f}\right)}, \quad \text{eqn 9}$$

The interpretation of eqn 9 depends on the relationship between S_f and S, i.e. between the infected seedling density and the total seedling density. For a case where only primary infection occurs, S_f is logically proportional to S and Holling Type II dynamics naturally arise from eqn 9. If secondary infections occur, however, the dependence of the infection rate on the seedling density may no longer be linear, and S_f may vary nonlinearly with S.

To account for all these scenarios in a unifying framework, we assume that S_f is proportional to S^n (i.e. $S_\mathrm{f} = \chi_n S^n$, χ_n is a proportionality constant that varies only with n) where for $n = 0$, we obtain a constant ψ_f (or Holling Type I), and for $n = 1$, we recover the primary infection case leading to Holling Type II. If $n = 2$ (or higher),

then this dependence suggests Holling Type III predator–prey interactions (Holling 1959), where the parameters of the Holling Type III function can now be directly related to the relative production of conidia in the subcanopy. Hence, for any $n \geq 0$, we have

$$\frac{\psi_f}{S^n} \approx \frac{d}{T\rho_c(r_1 - r_2)} \frac{1}{\left(\frac{E}{\chi_n \rho_c (r_1 - r_2)} + S^n\right)} \approx \frac{a_f}{c_n + S^n}; \text{ or}$$
$$\psi_f = \frac{a_f S^n}{c_n + S^n}, \qquad \text{eqn 10}$$

where a_f is the infection rate (seedlings m^{-2} fungi^{-1} day^{-1}) and c can be interpreted as the square of a threshold in seedling density at which fungal infection escalates due to the impact of repeated cycles of infection when $n = 2$ (Holling Type III). For simplicity, we refer to c_n as c hereafter.

There is not sufficient epidemiological data to fully characterize the epidemiology of the *D. mutila–I. deltoidea* system (encoded in n) and evaluate the cases above. However, a number of the features of the spatial distribution of fungal foliar infections observed in field plots at CCBS are illuminating and may assist in discerning n. First, the distance dependence in the rate or absolute value of the fungal infections amongst palm seedlings is small in comparison to the value of a fitted intercept across the range of observed infections (Fig. 3a), consistent with the assumption of uniformity in the distribution of primarily inoculum. The infection prevalence (P) can be predicted as a function of distance (x) according to the expression: $P = -0.029x + 0.77$

Infections were not observed at distances > 15 m from the tree. Within the range $0 < x < 15$, the sensitivity of disease prevalence to distance is small. Given that the majority of observed dynamics occur within this region, the assumption of uniformity appears reasonable. Secondly, it is apparent that at the relatively low seedling densities observed, discriminating between linear, Holling Type II ($n = 1$) or Holling Type III ($n = 2$) dynamics is rather challenging on the basis of this data set alone (Fig. 3b). All fits describe the data reasonably well and exhibit meaningful r^2 values ($r^2 = 0.76$ for the linear and Holling II case, $r^2 = 0.68$ for the Holling III case). Consequently, we tested all three approaches in the NC formulation and found that only when density effects were incorporated via use of the Holling III ($n = 2$) could we simultaneously reproduce the correct seed dispersal pattern, survival probabilities and seedling distribution (discussed further below). In particular we found that where Holling II ($n = 1$) or linear dynamics ($n = 0$) were employed, survival probabilities could not be well reconstructed and tend to saturate at short distances (< 5 m) from the parent tree. We attribute this saturation to the lack of sensitivity of these dynamics to the local density of seedlings (thereby necessitating the use of an $n \geq 2$).

Several literature sources indicate that there is potential for secondary infection to develop with *Diplodia* species and their allies, particularly when, as is the case for *I. deltoidea* seedlings, wounding is prevalent (Brown 1970; Srivastava 1972; Lo & Clark 1988; Olatinwo *et al.* 1999). Increasing survival probabilities at low seed densities are consistent with Holling III dynamics and the possibility of secondary infection. Consequently, the Holling III model is discussed in greatest detail in the remainder of the paper. Given the difficulty of definitively discriminating between the cases, however, we have also evaluated the implications of climate change in parallel assessments using linear and Holling II functional forms.

THE MULTI-PREDATOR MODEL

The resulting budget with multiple predators, including the Holling Type III fungi–seedling interaction, takes the overall form:

$$\frac{\partial S(x,t)}{\partial t} = \frac{\alpha}{\pi D}\left(\frac{\lambda}{2\pi\pi^3}\right)^{1/2} \exp\left[\frac{-\lambda(x-D)^2}{2D^2 x}\right] - \mu S(x,t)$$
$$- \frac{2b_i}{\pi q_i^2} \exp\left[\frac{-q_i x}{2}\right] \frac{a_i S}{1 - T_h a_i S} - b_f \frac{a_f S^2}{c + S^2} \qquad \text{eqn 11}$$

and the seedling distribution may be obtained by solving the model for S under the conditions $\partial S(x,t)/\partial t = 0$. An analytical solution for this equation may be obtained, but in practice, it is too unwieldy to deal with in 'closed form'. Instead S may be solved for using a standard root finding procedure applied at sequential spatial increments from the parent tree. Hereafter, this model and its solution are referred to as the multi-predator model (MPM).

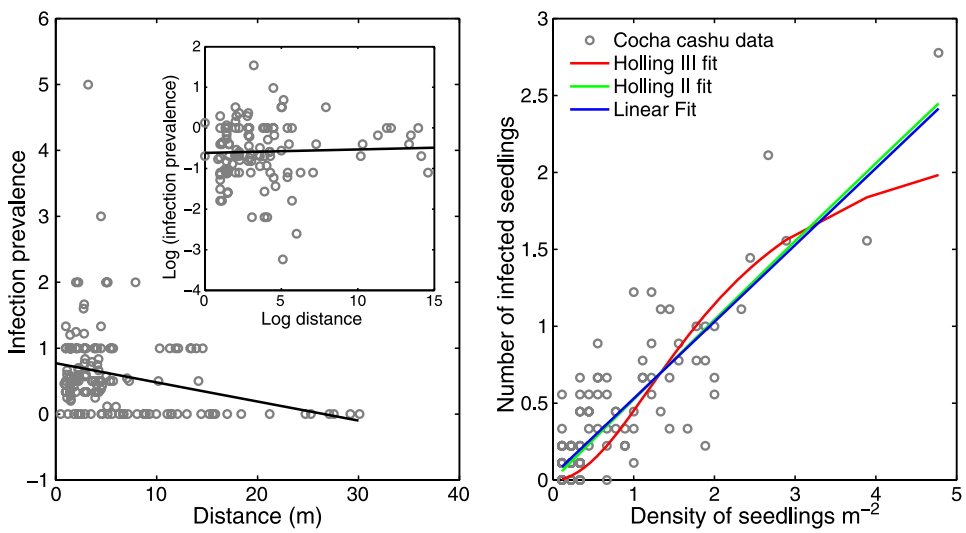

Fig. 3. Distance and density relationships with the occurrence of fungal foliar infection during the reproductive season of *Iriartea deltoidea*. Weakly decreasing relationships are evident between infection prevalence (and its log) and distance. However, the magnitude of the slope is low in comparison to the intercept. Across the range where infected seedlings were found, an approximation of the inoculum shedding as near uniform is appropriate. In contrast, the number of infected seedlings increased with seedling density, although given the generally low densities considered, linear ($r^2 = 0.76$), Holling II ($r^2 = 0.76$) and Holling III ($r^2 = 0.68$) dynamics all appear to be consistent with the observations.

MODEL PARAMETERIZATION WITH FIELD DATA

The MPM was fitted to field data from CCBS and LABS for the 3-month reproductive season of *I. deltoidea*. Regression fitting was conducted on three distributions: that of all recorded dispersal locations across the growing season (i.e. anywhere a seedling commenced growth), the distribution of surviving seedlings at the end of the growing season and the distribution of survival probabilities in space. The first distribution was taken to approximate the seed dispersal kernel at seasonal timescales. These data were compared to additional seed count data for *I. deltoidea* (Terborgh, unpublished data) and were found to be consistent with these data (e.g. seed densities of *c*. 3 seeds m^{-2} at distances *c*. 30 m from adult trees and lower densities (0.3 seeds m^{-2}) at greater distances again). Given the large number of parameters and the lack of an analytical function to fit, we used a search algorithm to maximize the coefficient of determination for the model fit to the measured survival, dispersal and final seedling distributions, and then refined the estimates locally for each of these variables. Fits were improved by smoothing the tail of the distribution over the last three data points. This smoothing simply accounts for the fact that the representation of discontinuous dispersal by a continuous distribution becomes more tenuous at longer distances from the parent tree, and larger averaging intervals are needed to capture the spatial behaviour. The results of the optimal parameterization provided estimates of the proportional influence of insects, fungi and random agents on palm seedling mortality.

As discussed above, various plausible options (linear, Holling II and Holling III) for the fungal dynamics were trialled in this phase. We could not simultaneously reproduce the increasing trend in the survival probabilities and the observed division of mortality amongst predators with the Holling II or linear models, largely because these models saturate at low seedling densities. Thus, they cannot in themselves generate the appropriate sensitivity in the survival distribution away from the fruiting trees. The appropriate survival trends can be induced with a Holling II model by simultaneously inflating the rate of fungal mortality and the spatial extent of insect predators, but this result in 92% of seedling mortality arising from fungal predators, rather than the observed 15% under contemporary conditions (Alvarez-Loayza 2009). By contrast, the Holling III model incorporates additional sensitivity to seedling densities, allowing the continued increase in survival probabilities with distance and the division of mortality to be captured appropriately (namely *c*.15% fungal mortality, 85% insects and random agents).

IMPACTS OF CLIMATE CHANGE ON MODEL PARAMETERS

Field and glasshouse data indicated that climate change was likely to reduce *I. deltoidea* fecundity, increase the rate of conidia production in association with accelerated growth of foliar spots, and increase the rate of seedling mortality. Given the observed linear relationship between rainfall and fecundity, and accounting for the 95% CI on the estimation of this relationship, the predicted 20% decline in rainfall over the Amazon region would result in a 15% (95% CI 0–20%) decline in fecundity.

We related the variation in seedling mortality to the value of the interaction rate a_f (seedling m^{-2} $fungi^{-1}$ day^{-1}). As identified in eqn 10, the timescale of mortality (T) is a linear divisor in the estimation of the a_f term. Thus, although a_f is not strictly a mortality rate, its value directly depends on variations in mortality, and allowing a_f to vary linearly with the mortality rate is reasonable. Consequently, we have represented the effect of climate change on a_f as a twofold (1.3–5.4) increase in T over the temperature change from 23 to 28 °C.

Determining what, if any, impact to attribute to the observed increase in conidia production rate (and, if the observed increases in conidia density with temperature are also included, bulk conidia production) is much more problematic, given the difficulties associated with clearly determining the appropriate interaction model between *D. mutila* and *I. deltoidea*.

Accordingly, we explored two bounding scenarios that might be expected to frame the plausible range of responses of the epidemiology to altered conidia production rates: (i) insensitivity and (ii) linear sensitivity in the density and mortality parameters. In the first case, model parameters were assumed to be invariant with respect to the rate of conidia production (cf. eqn 8). The alternative case considered the limiting behaviour of eqn 10 as the growth rates of fungal foliar spots increase: this affected not only the density parameter but also the estimation of a_f, which was shown to be inversely related to the rate of conidia production. The acceleration of the foliar spot growth rates was applied to the difference term (r_1-r_2), which states that the both the growth rate of foliar spots and the release of conidia from these spots increase with fungal metabolism (the presumed cause of enhanced growth rate at higher temperatures).

Thus, the scenarios considered in the modelling sensitivity analysis were:

1. A 100% increase in $1/T$ while holding b_f and c constant (cf. eqn 8): this results in a 100% increase in a_f.
2. A 100% increase in $1/T$, while allowing (r_1-r_2) to increase by a factor of five: this results in a 60% decrease in a_f and a 80% reduction in c (cf. eqn 10).
3. A 15% decline in fecundity, considered in isolation from and in addition to the previous cases.

A significant uncertainty persists across these scenarios, namely the effect (if any) of increased temperature on the production of primary inoculant from canopy endophytes (E from eqns 6–10), which would be anticipated to increase the overall abundance (b_f) of conidia. No data are available to assess the likelihood of such a change, nor its potential magnitude. However, the response of conidia production on seedling foliar spots (i.e. a fivefold increase) could reasonably be taken as the 'upper limit' of any such increase, given that contemporary observations suggest that sporulation rates are greatest on seedlings. Decreases are also plausible, however, as the projected reductions in precipitation may reduce the rate of transport of conidia to the subcanopy. Thus, we undertook an order of magnitude sensitivity analysis by allowing b_f to vary from 5 to 2500, or 10 to 500% of contemporary values. The effects of these scenarios and sensitivity analyses on the model parameters are presented in Table 1.

Results

The results section documents the form of the modifications made to the NC model and then considers its suitability to represent survival and distributions of *I. deltoidea*. The ultimate effects of climate change on predicted *I. deltoidea* recruitment are presented. The results are intended to illustrate how climate induced increases in mean air temperature and reduced precipitation impact host–pathogen interactions and concomitant recruitment and survival.

Table 1. Inferred parameters by fitting the multi-predator model to seed dispersal, seedling survival and seedling distribution data for *Iriartea deltoidea*. The climate sensitivity scenarios examined for a 5 °C rise in temperature are shown in the right hand columns

Parameter		23 °C (inferred)	28 °C (predicted, *lower CI, upper CI*)		
			Case 1: Limited secondary infection	Case 2: Enhanced secondary infection	Case 3: Sensitivity to fungal abundance
a_f	seedlings m^{-2} $predator^{-1}$ day	0.1	0.2 *(0.13,0.5)*	0.04 *(0.016–0.17)*	0.04, 0.2
a_i	seedlings $m^{-2}predator^{-1}$ day	1	1	1	1
c	$(seedlings\ m^{-2})^2$	30	30	6 *(3.9–9.8)*	6, 30
q	m	0.1	0.1	0.1	0.1
D	m	0.8	0.8	0.8	0.8
λ		0.1	0.1	0.1	0.1
α	seeds day^{-1}	100	85 *(80,100)*	85 *(80,100)*	85
μ	day^{-1}	0.1	0.1	0.1	0.1
T_h	day $seedling^{-1}$	1.9	1.9	1.9	1.9
b_f	fungi numbers	500	500	500	Varied: 50–2500
b_i	insect numbers	100	100	100	100

SOLUTIONS OF THE STEADY STATE NC MODEL

The appeal of the original NC model is that it demonstrates that many of the forms of seedling survival probability and spatial distributions that are observed in the field can be generated as solutions of eqn 1. In particular, Janzen–Connell effects (where survival increases with distance, resulting in a peak of the recruitment curve away from the parent plant) were found to occur when the mean dispersal distance (D) exceeded the mean distance at which predators were found (q) (Janzen 1970). Adopting a 'fat-tailed' dispersal kernel such as the Wald (or others) in the framework of eqn 1 ensures that survival increases with distance regardless of the relative magnitudes of D and q (a primary departure from the results obtained with exponential dispersal kernels). This is a reflection of the increased probability of some seeds being dispersed at a long-distance from the parent tree and 'escaping' the concentration of herbivorous predators (Janzen 1970). We tested a wide variety of predation functions (linear, Holling Type II, Holling Type III), predator distributions (uniform, exponential) in conjunction with different seed dispersal kernels (exponential, Wald) and were able to reconstruct the full spectrum of seed dispersal patterns reported by Nathan and Casagrandi (Nathan & Casagrandi 2004; Mari *et al.* 2008).

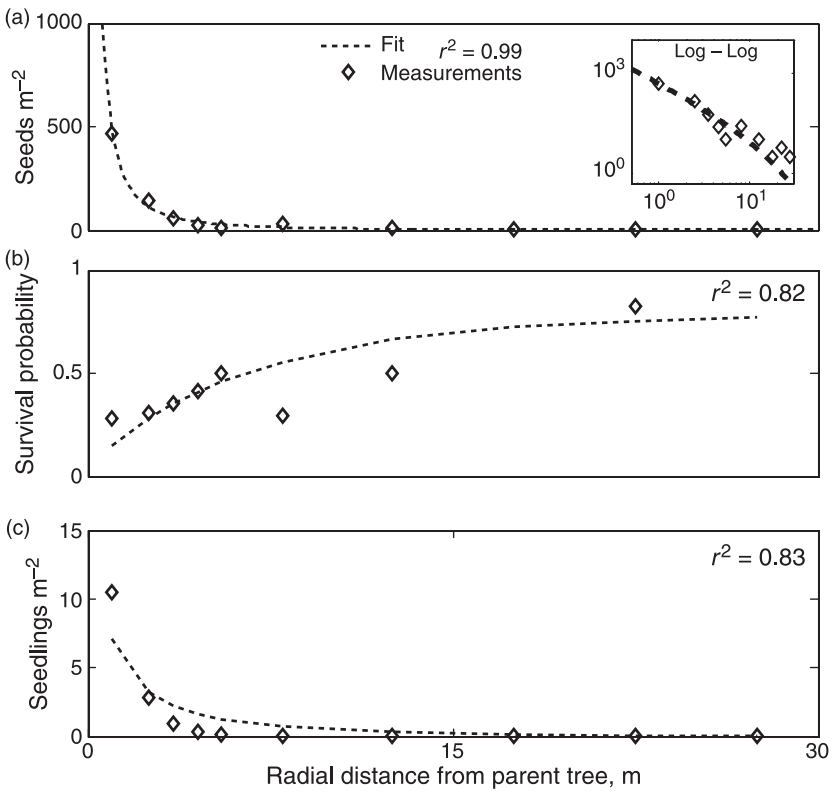

Fig. 4. Model parameter estimation by simultaneously matching the seed dispersal kernel, survival probabilities and final seedling distribution using the multi-predator model. (a) Dispersal kernel for seeds (log-log plot shown as an insert). (b) Survival probability of seedlings. (c) Seedling distribution at the end of the reproductive season. This parameterization also reproduces the relative mortality due to fungi and insects under contemporary climatic conditions (*c.* 15% fungi).

MODEL RESULTS

Using the MPM, with the Holling III assumption, good fits to the dispersal, survival and seedling distribution data for *I. deltoidea* were obtained (see Fig. 4) for the parameters shown in Table 1. For these parameters, 16% of the steady-state seedling mortality occurred due to fungal predation, and 84% due to insects and random mortality agents. This division in mortality is close to that observed in the field where 15% of mortality is associated with fungal pathogens (Alvarez-Loayza 2009). The effects of climate change in each scenario (as outlined previously in the 'Impacts of Climate Change' section) were considered in isolation and together. Decreases in palm fecundity in the order of 15% did not have a significant impact on the survival probability distribution through space. In fact, survival probabilities improved due to a reduction in density-dependent herbivory (see Fig. 5). The reduction in the total number of seeds dispersed resulted in an *c*. 10% drop in seedling abundance.

Scenario 1 assumed that secondary infection probabilities would be unchanged under future climate scenarios, such that a_f increased in isolation. This scenario resulted in a 25% reduction in seedling abundance (32% if fecundity effects were included), and reduced survival probabilities. The alternative scenario, in which increased rates of conidia production are sufficient to increase the probabilities of secondary infection and thus the density dependence of the *D. mutila–I. deltoidea* relationship had the strongest impact of the scenarios considered, reducing overall survival to 44% of 2009 values, or 38% if the fecundity effects are also included (Fig. 6, Table 2).

The sensitivity analysis on b_f values in the range of 10–500% of contemporary levels showed that increases in fungal abundance resulted in large declines in seedling survival numbers. These decreases were exacerbated when combined with the potential for higher mortality rates or enhanced secondary infection probabilities. Conversely, reductions in fungal abundance in the order of 20% (i.e. in line with projected rainfall declines) increased seedling survival, but not sufficiently to alleviate the projected affects of increased temperature (Fig. 7).

Discussion

Under current climate scenarios, *c*. 0.5% of seeds dispersed by an *I. deltoidea* tree over the course of a reproductive season survives to become seedlings. Of these, only 44% are likely to mature into juvenile plants. The model results indicate, across a range of scenarios, higher temperatures are likely to reduce the size of new seedling cohorts due to reduced seed production, increased rates of mortality induced by fungal infection, and, in the most severe circumstances, increased probability of secondary infection cycles. The implications of such a reduction on the Western Amazonian ecosystems are difficult to assess, given the long generational timescales of *I. deltoidea* and the complexity of the local ecosystem. Reduction in the recruitment of this abundant understorey species might result in improved niche availability, or perturb existing trophic relationships. Reduced seedling survival would, however, be expected to negatively impact disturbed forests, limiting the capacity for *I. deltoidea* to re-establish following deforestation. This is of particular concern when the additional effects of light on *D. mutila* (discussed below), and the likelihood of strong dispersal limitation on *I. deltoidea* (as documented by Losos (1995), for instance) are considered.

The broader implications of these findings for ongoing research into the response of Amazonian ecosystems to climate change are quite clear. Evidently, it is possible for increased temperature and reduced precipitation to initiate shifts in pathogen behaviour and host–pathogen interactions. Where the impact of these shifts is to increase pathogen abundance or virulence, the consequences on plant populations may be severe. The difficulties associated with making clear predictions even in this intensively studied case offer an opportunity to assess areas where further research is needed.

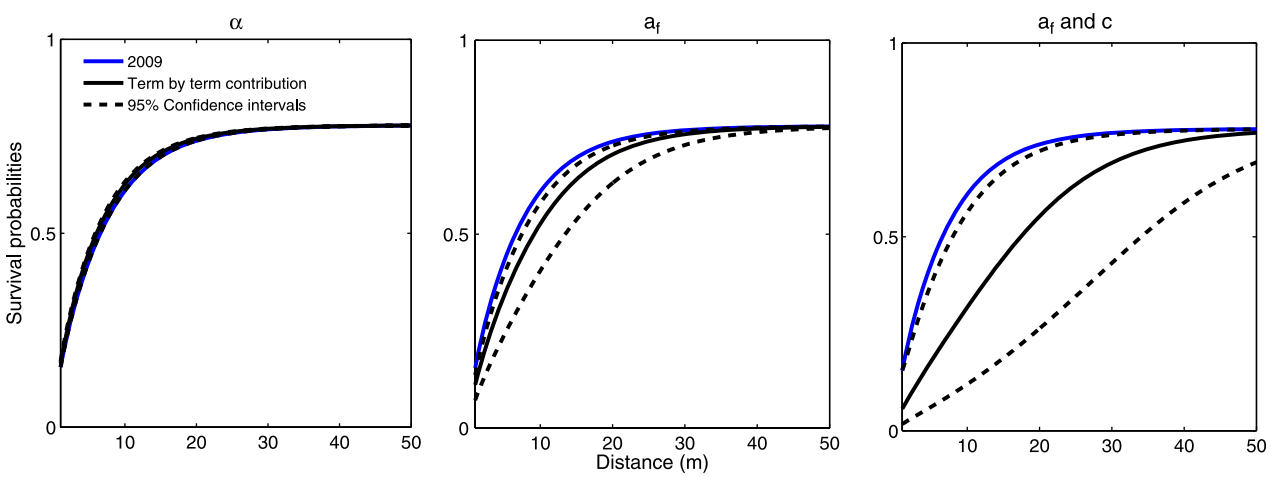

Fig. 5. Predicted survival probability of *Iriartea deltoidea* from 2009 to 2100, showing the contribution of shifts in fecundity (left panel) and the two scenarios of fungal response that were explored (no density response, center panel, and enhanced density dependence, right panel). The dashed lines indicate the 95% confidence intervals associated with extrapolating from the glasshouse experiment.

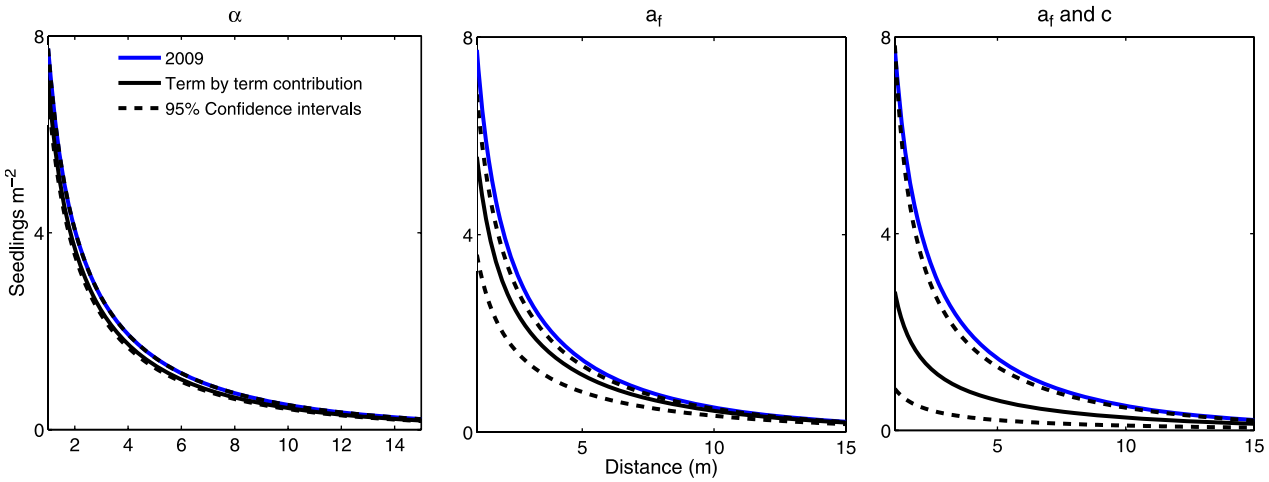

Fig. 6. Predicted *Iriartea deltoidea* seedling distribution from 2009 to 2100, showing the contribution of each aspect of fungal pathogenicity and shifts in fecundity. The dashed lines indicate the 95% confidence intervals associated with extrapolating from the glasshouse experiment.

Table 2. Predicted future seedling abundance as a proportion of the 2009 seedling abundance associated with the tested fungal response scenarios to future climatic conditions

Scenarios		Seedling abundance cf. 2009 conditions	Linear dynamics case	Holling II case
	Reduced fecundity in isolation	0.9 *(0.85, 1)*	0.81 *(0.74, 1.0)*	0.80 *(0.74,1.0)*
(1)	Increased mortality, limited secondary infection	0.76 *(0.52,0.90)*	0.96 *(0.88, 0.98)*	0.93 *(0.78,0.98)*
	+ reduced fecundity	0.68 *(0.45, 0.90)*	0.77 *(0.65, 0.98)*	0.75 *(0.58,0.98)*
(2)	Increased mortality, increased secondary infection	0.44 *(0.14, 1.3)*		
	+ reduced fecundity	0.38 *(0.13, 1.3)*		
(3)	Increased primary inoculant (in isolation)	0.52		
	+ (1)	0.40 *(0.26,0.47)*		
	+ (1) + reduced fecundity	0.35 *(0.23, 0.47)*		
	+ (2)	0.20 *(0.07, 0.46)*		
	+ (2) + reduced fecundity	0.18 *(0.06, 0.46)*		

First, it has been clearly shown that the implications of climatic changes on fungal pathogen behaviour are not in themselves sufficient to predict the impacts for the host–pathogen system, at least in the absence of detailed epidemiological knowledge. The lack of such knowledge in the case of *D. mutila–I. deltoidea* has forced us to address this problem by testing competing models against bulk survival data and by considering competing scenarios to address the potential for increased conidia production by seedlings to significantly alter the epidemiology of the infection. Although both scenarios considered predict reductions in seedling survival, the magnitude of these reductions is dramatically different between the scenarios, illustrating the sensitivity of the system to the epidemiological details. As shown in Table 2, these sensitivities are further compounded when alternative fungal–host interaction models are considered.

Secondly, the response of infected adult trees (and endophytic fungi) to temperature shifts was identified as a key area of uncertainty. Manipulative experiments often consider seedlings for practical reasons, but pathogenic fungi may also affect adult trees, where the potential for rapid ecosystem change to result from increased mortality due to fungi is considerably greater than in the case of seedlings. Furthermore, when the canopy acts as a key source of fungal propagules, the feedbacks between adults, seedlings and fungi may prove important. The near-ubiquity of *D. mutila* as an endophyte in adult *Iriartea* may imply that the observed increases in seedling mortality and conidia production are unlikely to have an impact on adult palms. If however, climate change induced similar changes amongst endophytic fungi, then the ubiquity of *D. mutila* suggests that such changes could have widespread effects. Endophytic fungi may confer plant resistance to abiotic and biotic stress factors (Arnold *et al.* 2000; Rodriguez, Redman & Henson 2004; Schulz & Boyle 2005; Clarke *et al.* 2006), but the effect of climate change on endophytes is not definitively understood. Furthermore, the susceptibility of adult trees to pathogens is altered when they experience physiological stress due to increased heat and drought (Maxwell, Kruger & Stanosz 1997; Mayek-Perez, Acosta-Gallegos & Lopez-Castaneda 2002; Mittler 2006), although in some cases stressors may increase host resistance (Pennypacker, Leath & Hill 1991). Consequently, determining the likelihood of increased pathogen impact on adult tree species in response to climate change should be a research priority.

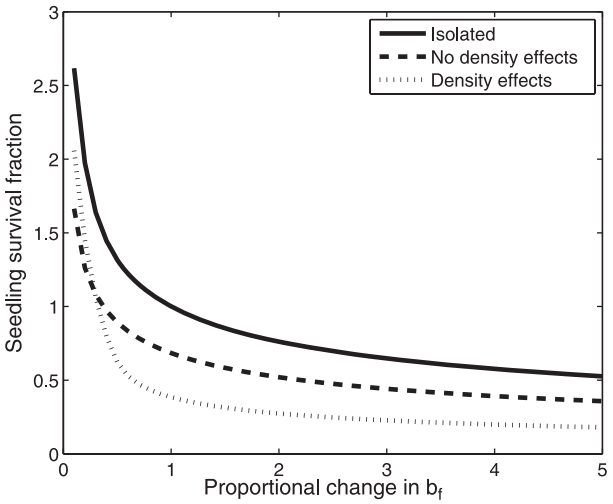

Fig. 7. Sensitivity of seedling survival fraction (in percent relative to 2009 values) resulting from changes in the fungal abundance b_f. Changes in b_f cause a response in survival whether considered in isolation (solid line) or in conjunction with increased virulence and conidia production and decreased fecundity (dashed line). Based on seedling conidia production responses, a fivefold increase represents a reasonable upper margin of potential increases in fungal abundance, while decreases in rainfall may be associated with reduced transport of conidia from canopy endophytes to the subcanopy.

Even if *D. mutila* is representative of only a subset of climate sensitive pathogens, the magnitude of the effects predicted for *I. deltoidea* suggests that ecosystem-wide ramifications could result, due to changes in abundance or genetic diversity in affected host plants. Furthermore, given that *D. mutila* is also a generalist pathogen with a world-wide distribution and is able to infect a wide range of angiosperms (Jacobs & Rehner 1998) including pines, eucalypts and grapes (De Wet *et al.* 2008), the predicted enhancement of its virulence and growth rates at elevated temperature may have important ramifications for agricultural and forestry production globally.

UNCERTAINTIES

It is estimated that only 5% of plant fungal pathogens are well understood and characterized (Ingram 1999). Even when considering a relatively well-studied pathogen such as *Diplodia*, it is difficult to obtain robust estimates of the parameters governing its epidemiology. Furthermore, extrapolations from glasshouse conditions to field conditions have been necessary. Consequently, the parameterization of this model and the interpretation of the results obtained must be approached with caution. Numerous other potential feedbacks including changes in insect abundance or foraging behaviour (Givnish 1999), adaptation of *D. mutila* or *I. deltoidea* to changed climate (Rodriguez, Redman & Henson 2004), the impacts of changing precipitation on *D. mutila* or the impacts of changing temperature on fecundity remain poorly quantified.

The quantitative predictions made here are totally subject to the constraints associated with predictions of future climate in the Amazonian basin. These uncertainties are particularly problematic for rainfall predictions in the Western Amazon, largely due to confounding effects of topography (Seth *et al.* 2007). The temperature changes predicted, however, appear to be more robust (Coppola & Giorgi 2005), and given that temperature was predominantly responsible for the predicted changes in the *Diplodia–Iriartea* relationships, the qualitative predictions should be less affected. Uncertainties in the timing and degree of climate change, however, permeate all these predictions.

An additional source of uncertainty relates to the potential response of *D. mutila* to predicted reductions in precipitation, which remains largely unexplored, beyond the potential sensitivity of? conidia transport to precipitation. Assuming no change in response to reduced precipitation may be regarded as unreasonable given the commonly held premise that increased humidity facilitates predation of plants by desiccation-intolerant insects and pathogens (Givnish 1999). Indeed, drought stress is believed to enhance resistance to plant infection by inducing stomatal closure and denying inoculum entry to the plant tissue (Garrett *et al.* 2006). Conversely, however, *D. mutila* has been shown to grow faster and cause more extensive damage in water-stressed oak species (Ragazzi, Moricca & Dellavalle 1999). Water stress was hypothesized to impair the defence mechanisms of the trees and to be related to widespread *D. mutila* damage amongst oaks in Italy, suggesting that water stress may actually enhance the susceptibility of some host species to infection and mortality.

As highlighted in the model development, some uncertainty remains as to whether *D. mutila* dynamics are best represented as linear, Holling II or Holling III functions. Although use of the Holling III function was most effective at representing the trends in survival probability of the seedlings, the more parsimonious Holling II and linear dynamics cannot be discounted. The effects of climate change on the predicted seedlings survival by the model as parameterized with the best fit using Holling II and linear dynamics are summarized in Table 2 for the cases of altered fecundity and altered seedling mortality. Certainly the choice of Holling III function appears to be the most sensitive to climate dynamics of the possibilities. However, in all cases, non-trivial responses in seedling survival are anticipated following the predicted climate shifts.

Finally, there are additional feedbacks between plant physiology and pathogen activity that might be mediated by climate change. The first of these pertains to light. Experimental work indicates that *D. mutila* virulence increases with light intensity, leading to increased mortality amongst infected *Iriartea deltoidea* seedlings exposed to a high-light regime (Alvarez-Loayza 2009). *Diplodia mutila* has enhanced mycelial growth and melanin production with increased exposure to light. Light-induced pathogenicity of *D. mutila* may constrain seedling recruitment of the host to the shaded understorey by limiting survival of seedlings in direct light (Alvarez-Loayza 2009). A potential adaptation of Amazon species to reductions in precipitation is a reduction in leaf area index in the order of 20–25% (Asner, Townsend & Braswell 2000; Nepstad *et al.* 2002; Asner *et al.* 2004). While such a reduction may buffer trees against water loss, it would also likely increase the light

intensity at the forest floor and magnify the effects of *Diplodia* on seedling mortality.

Positive feedbacks may also exist between pathogens and disturbance. Recent research in Mexico shows that herbivory and pathogen damage were more significant in forest edges than within patches (Benitez-Malvido & Lemus-Albor 2005). Fragmentation can interrupt plant dispersal processes, reducing the capacity of host species to keep pace with changes in abiotic and biotic conditions. The combination of increasingly active pathogens and increased clearing and fragmentation may prove a potent threat to forest ecosystems.

Conclusions

Pathogens are now starting to be considered in models of extinction risk for animals (Gerber *et al*. 2005), and interactions between climate change and plant pathogens are becoming a research priority (Garrett *et al*. 2006). Research has identified changes in the geographical range of infection risk (Brasier 1996; Bergot *et al*. 2004) and also in terms of the severity of epidemics (Garrett *et al*. 2006; Evans *et al*. 2008). It is now clear that climate change also has the potential to impact the abundance, virulence and density dependence of fungal pathogens attacking *I. deltoidea* seed and seedlings. The impact of these changes will be decreased seedling survival and reduced recruitment with potential ramifications at an ecosystem-wide level. The magnitude of the predicted shifts appears to be strongly dependent on the specific fungal epidemiology, the details of which are still unclear for many important host–pathogen relationships.

Critically, and in contrast to the predictions of carbon- and water-mediated 'Amazon Collapse', the timescales associated with the exacerbation of fungal pathogenicity are likely to be very rapid: in the experiments reported here, fungi responded immediately to changes in ambient temperature. While reductions in seedling recruitment are unlikely to result in community-scale changes on such rapid timescales, recovery in disturbed or fragmented environments is likely to be immediately impacted. Consequently, the potential for climatic effects to increase the severity of fungal infections must be considered as an additional threat to the integrity of Amazonian ecosystems, acting synergistically with clearing, temperature and water stress. As ongoing research elucidates further information regarding pathogen temperature response and fungal epidemiology, the approaches developed here can be used to continue the quantification and assessment of this risk.

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References

Agrios, G. (2005) *Plant Pathology*. Elsevier-Academic Press, San Diego, CA, USA.

Alvarez-Loayza, P. (2009) *The role of pathogens in determining plant recruitment and distribution patterns in a western Amazonian floodplain*. PhD Thesis, Rutgers, New Brunswick.

Arnold, E., Maynard, Z., Gilbert, G., Coley, P.D. & Kursar, T.A. (2000) Are tropical fungal endophytes hyperdiverse? *Ecology Letters*, **3**, 267–274.

Asner, G.P., Townsend, A.R. & Braswell, B.H. (2000) Satellite observation of El Nino effects on Amazon forest phenology and productivity. *Geophysical Research Letters*, **27**, 981–984.

Asner, G.P., Nepstad, D., Cardinot, G. & Ray, D. (2004) Drought stress and carbon uptake in an Amazon forest measured with spaceborne imaging spectroscopy. *Proceedings of the National Academy of Sciences of the United States of America*, **101**, 6039–6044.

Augspurger, C.K. & Kelly, C.K. (1984) Pathogen mortality of tropical tree seedlings – experimental studies of the effects of dispersal distance, seedling density, and light conditions. *Oecologia*, **61**, 211–217.

Bell, T., Freckleton, R.P. & Lewis, O.T. (2006) Plant pathogens drive density-dependent seedling mortality in a tropical tree. *Ecology Letters*, **9**, 569–574.

Benitez-Malvido, J. & Lemus-Albor, A. (2005) The seedling community of tropical rain forest edges and its interaction with herbivores and pathogens. *Biotropica*, **37**, 301–313.

Bergot, M., Cloppet, E., Perarnaud, V., Deque, M., Marcais, B. & Desprez-Loustau, M.L. (2004) Simulation of potential range expansion of oak disease caused by *Phytophthora cinnamomi* under climate change. *Global Change Biology*, **10**, 1539–1552.

Betts, R.A., Malhi, Y. & Roberts, J.T. (2008) The future of the Amazon: new perspectives from climate, ecosystem and social sciences. *Philosophical Transactions of the Royal Society B-Biological Sciences*, **363**, 1729–1735.

Betts, R.A., Cox, P.M., Collins, M., Harris, P.P., Huntingford, C. & Jones, C.D. (2004) *The Role of Ecosystem-Atmosphere Interactions in Simulated Amazonian Precipitation Decrease and Forest Dieback Under Global Climate Warming*, pp. 157–175. Springer, Wien.

Boland, G.J., Melzer, M.S., Hopkin, A., Higgins, V. & Nassuth, A. (2004) Climate change and plant diseases in Ontario. *Canadian Journal of Plant Pathology-Revue Canadienne De Phytopathologie*, **26**, 335–350.

Brasier, C.M. (1996) Phytophthora cinnamomi and oak decline in southern Europe Environmental constraints including climate change. *Annales Des Sciences Forestieres*, **53**, 347–358.

Broadbent, E.N., Asner, G.P., Keller, M., Knapp, D.E., Oliveira, P.J.C. & Silva, J.N. (2008) Forest fragmentation and edge effects from deforestation and selective logging in the Brazilian Amazon. *Biological Conservation*, **141**, 1745–1757.

Brown, G.E. (1970) Pycnidial release and survival of *Diplodia natalensis* spores. *Phytopathology*, **61**, 559–561.

Cavaleri, M.A., Oberbauer, S.F. & Ryan, M.G. (2008) Foliar and ecosystem respiration in an old-growth tropical rain forest. *Plant, Cell and Environment*, **31**, 473–483.

Chakraborty, S., Tiedemann, A.V. & Teng, P.S. (2000) Climate change: potential impact on plant diseases. *Environmental Pollution*, **108**, 317–326.

Clark, D.B., Palmer, M.W. & Clark, D.A. (1999) Edaphic factors and the landscape-scale distributions of tropical rain forest trees. *Ecology*, **80**, 2662–2675.

Clarke, B.B., White, J.F., Hurley, R.H., Torres, M.S., Sun, S. & Huff, D.R. (2006) Endophyte-mediated suppression of dollar spot disease in fine fescues. *Plant Disease*, **90**, 994–998.

Coakley, S.M., Scherm, H. & Chakraborty, S. (1999) Climate change and plant disease management. *Annual Review of Phytopathology*, **37**, 399–426.

Cochrane, M.A. & Barber, C.P. (2009) Climate change, human land use and future fires in the Amazon. *Global Change Biology*, **15**, 601–612.

Copes, W.E. & Hendrix, F.F. (2004) Effects of temperature on sporulation of *Botryosphaeria dothidea*, *B. obtusa* and *B. rhodina*. *Plant Disease*, **88**, 292–296.

Coppola, E. & Giorgi, F. (2005) Climate change in tropical regions from high-resolution time-slice AGCM experiments. *Quarterly Journal of the Royal Meteorological Society*, **131**, 3123–3145.

De Wet, J., Slippers, B., Preisig, O., Wingfield, B.D. & Wingfield, M.J. (2008) Phylogeny of the Botryosphaeriaceae reveals patterns of host association. *Molecular Phylogenetics and Evolution*, **46**, 116–126.

Evans, N., Baierl, A., Semenov, M.A., Gladders, P. & Fitt, B.D.L. (2008) Range and severity of a plant disease increased by global warming. *Journal of the Royal Society Interface*, **5**, 525–531.

Farr, D.F. & Rossman, A.Y. (2010) *Fungal Database:* Diplodia mutila *and Synonyms*. Systematic Mycology and Microbiology Laboratory, ARS, USDA, Beltsville, MD, USA.

Gallery, R.E., Dalling, J.W. & Arnold, A.E. (2007) Diversity, host affinity, and distribution of seed-infecting fungi: a case study with *Cecropia*. *Ecology*, **88**, 582–588.

Garrett, K.A., Dendy, S.P., Frank, E.E., Rouse, M.N. & Travers, S.E. (2006) Climate change effects on plant disease: genomes to ecosystems. *Annual Review of Phytopathology*, **44**, 489–509.

Gentry, A.H. (1990) *Four Neotropical Rainforests*. Yale University Press, New Haven, CT, USA.

Gerber, L.R., McCallum, H., Lafferty, K.D., Sabo, J.L. & Dobson, A. (2005) Exposing extinction risk analysis to pathogens: is disease just another form of density dependence? *Ecological Applications*, **15**, 1402–1414.

Givnish, T.J. (1999) On the causes of gradients in tropical tree diversity. *Journal of Ecology*, **87**, 193–210.

Holling, C.S. (1959) Some characteristics of simple types of predation and parasitism. *Canadian Entomologist*, **91**, 385–398.

Huntingford, C., Fisher, R.A., Mercado, L., Booth, B.B.B., Sitch, S., Harris, P.P. *et al.* (2008) Towards quantifying uncertainty in predictions of Amazon 'dieback'. *Philosophical Transactions of the Royal Society B-Biological Sciences*, **363**, 1857–1864.

Ibanez, I., Clark, J.S., Dietze, M.C., Feeley, K., Hersh, M., LaDeau, S., McBride, A., Welch, N.E. & Wolosin, M.S. (2006) Predicting biodiversity change: outside the climate envelope, beyond the species-area curve. *Ecology*, **87**, 1896–1906.

Ingram, D.S. (1999) Biodiversity, plant pathogens and conservation. *Plant Pathology*, **48**, 433–442.

Jacobs, K.A. & Rehner, S.A. (1998) Comparison of cultural and morphological characters and ITS sequences in anamorphs of *Botryosphaeria* and related taxa. *Mycologia*, **90**, 10.

Janzen, D.H. (1970) Herbivores and number of tree species in tropical forests. *American Naturalist*, **104**, 501.

Katul, G.G., Porporato, A., Nathan, R., Siqueira, M., Soons, M.B., Poggi, D., Horn, H.S. & Levin, S.A. (2005) Mechanistic analytical models for long-distance seed dispersal by wind. *American Naturalist*, **166**, 368–381.

Lloyd, J. & Farquhar, G.D. (2008) Effects of rising temperatures and [CO_2] on the physiology of tropical forest trees. *Philosophical Transactions of the Royal Society B-Biological Sciences*, **363**, 1811–1817.

Lo, J.Y. & Clark, C.A. (1988) Source of inoculum and infection courst of *Diplodia gossypina* on sweet potato. *Phytopathology*, **61**, 1442–1446.

Losos, E. (1995) Habitat specificity of 2 palm species – experimental transplantation in Amazonian successional forests. *Ecology*, **76**, 2595–2606.

Macia, M.J. & Svenning, J.C. (2005) Oligarchic dominance in western Amazonian plant communities. *Journal of Tropical Ecology*, **21**, 613–626.

Mari, L., Casagrandi, R., Gatto, M., Avgar, T. & Nathan, R. (2008) Movement strategies of seed predators as determinants of plant recruitment patterns. *American Naturalist*, **172**, 694–711.

Martinez-Ramos, M., Anten, N.P.R. & Ackerly, D.D. (2009) Defoliation and ENSO effects on vital rates of an understorey tropical rain forest palm. *Journal of Ecology*, **97**, 1050–1061.

Maxwell, D.L., Kruger, E.L. & Stanosz, G.R. (1997) Effects of water stress on colonization of poplar stems and excised leaf disks by *Septoria musiva*. *Phytopathology*, **87**, 381–388.

Mayek-Perez, N., Acosta-Gallegos, J.A. & Lopez-Castaneda, C. (2002) Relationship between early vigor of common bean (*Phaseolus vulgaris* L.) and resistance to water stress and to *Macrophomina phaseolina* (Tassi) *Goidanich*. *Revista Mexicana de Fitopatologia*, **20**, 133–140.

Mittler, R. (2006) Abiotic stress, the field environment and stress combination. *Trends in Plant Science*, **11**, 15–19.

Nathan, R. & Casagrandi, R. (2004) A simple mechanistic model of seed dispersal, predation and plant establishment: Janzen-Connell and beyond. *Journal of Ecology*, **92**, 733–746.

Nepstad, D.C., Moutinho, P., Dias, M.B., Davidson, E., Cardinot, G., Markewitz, D. *et al.* (2002) The effects of partial throughfall exclusion on canopy processes, aboveground production, and biogeochemistry of an Amazon forest. *Journal of Geophysical Research-Atmospheres*, **107**, doi: 10.1029/2001JD000360.

Olatinwo, R., Cardwell, K., Menkir, A., Deadman, M. & Julian, A. (1999) Inheritance of resistance to *Stenocarpella macrospora* (Earle) ear rot of maize in the mid-altitude zone of Nigeria. *European Journal of Plant Pathology*, **105**, 535–543.

Pennypacker, B.W., Leath, K.T. & Hill, R.R.J. (1991) Impact of drought stress on the expression of resistance to *Verticillium-Albo-Atrum* in Alfalfa. *Phytopathology*, **81**, 1151.

Petrini, O. (1986) Taxonomy of endophytic fungi of aerial plant tissues. *Microbiology of the Phyllosphere* (eds N.J. Fokkema & J. van den Heuve). pp. 175–187. Cambridge University Press, Cambridge, UK.

Pitman, N.C. (2009) *An Overview of the Los Amigos Watershed, Madre de Dios, Southeastern Peru*. ACCA, Puerto Maldonado, Peru.

Pitman, N.C.A., Terborgh, J.W., Silman, M.R., Nunez, P., Neill, D.A., Ceron, C.E., Palacios, W.A. & Aulestia, M. (2001) Dominance and distribution of tree species in upper Amazonian terra firme forests. *Ecology*, **82**, 2101–2117.

Ragazzi, A., Moricca, S. & Dellavalle, I. (1999) Water stress and the development of cankers by *Diplodia mutila* on *Quercus robur*. *Journal of Phytopathology-Phytopathologische Zeitschrift*, **147**, 425–428.

Rodriguez, R.J., Redman, R.S. & Henson, J.M. (2004) The role of fungal symbioses in the adaptation of plants to high stress environments. *Mitigation and Adaptation Strategies for Global Change*, **9**, 261–272.

Sanchez, M.E., Venegas, J., Romero, M.A., Phillips, A.J.L. & Trapero, A. (2003) Botryosphaeria and related taxa causing oak canker in southwestern Spain. *Plant Disease*, **87**, 1515–1521.

Schulz, B. & Boyle, C. (2005) The endophytic continuum. *Mycological Research*, **109**, 661–686.

Seth, A., Rauscher, S.A., Camargo, S.J., Qian, J.H. & Pal, J.S. (2007) RegCM3 regional climatologies for South America using reanalysis and ECHAM global model driving fields. *Climate Dynamics*, **28**, 461–480.

Sezen, U.U., Chazdon, R.L. & Holsinger, K.E. (2007) Multigenerational genetic analysis of tropical secondary regeneration in a canopy palm. *Ecology*, **88**, 3065–3075.

Srivastava, D.N. (1972) Epidemiology and prevention of Diplodia stem-end rot of ripe mango fruits. *Acta Horticultura*, **24**, 235–236.

Tapsoba, H. & Wilson, J.P. (1997) Effects of temperature and light on germination of urediniospores of the pearl millet rust pathogen, *Puccinia substriata* var. *indica*. *Plant Disease*, **81**, 1049–1052.

Terborgh, J. (1983) *Five New World Primates: A Study in Comparative Ecology*. Princeton University Press, Princeton, NJ, USA.

Wattenberg, I. & Breckle, S.W. (1995) Tree species diversity of a premontane rain forest in the Cordillera de Tilaran, Costa Rica. *Ecotropica*, **1**, 21–30.

Waugh, M.M., Kim, D.H., Ferrin, D.M. & Stanghellini, M.E. (2003) Reproductive potential of *Monosporascus cannonballus*. *Plant Disease*, **87**, 45–50.

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