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LETTER

Testing the Janzen-Connell mechanism: pathogens cause overcompensating density dependence in a tropical tree

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Abstract

The Janzen-Connell hypothesis is a leading explanation for plant-species diversity in tropical forests. It suggests that specialized natural enemies decrease offspring survival at high densities beneath parents, giving locally rarer species an advantage. This mechanism, in its original form, assumes that density dependence is overcompensating: mortality must be disproportionately high at the highest densities, with few offspring recruiting below their parents. We tested this assumption using parallel shadehouse and field density-series experiments on seedlings of a tropical tree, *Pleradenophora longicuspis*. We found strong, overcompensating mortality driven by fungal pathogens, causing 90% (shadehouse) or 100% (field) mortality within 4 weeks of germination, and generating a negative relationship between initial and final seedling densities. Fungicide treatment led to much lower, density-independent, mortality. Overcompensating mortality was extremely rapid, and could be missed without detailed monitoring. Such dynamics may prevent dead trees from being replaced by conspecifics, promoting coexistence as envisioned by the Janzen-Connell hypothesis.

Keywords

Host–pathogen interactions, nonlinear dynamics, plant diversity, population dynamics, species coexistence, tropical forests.

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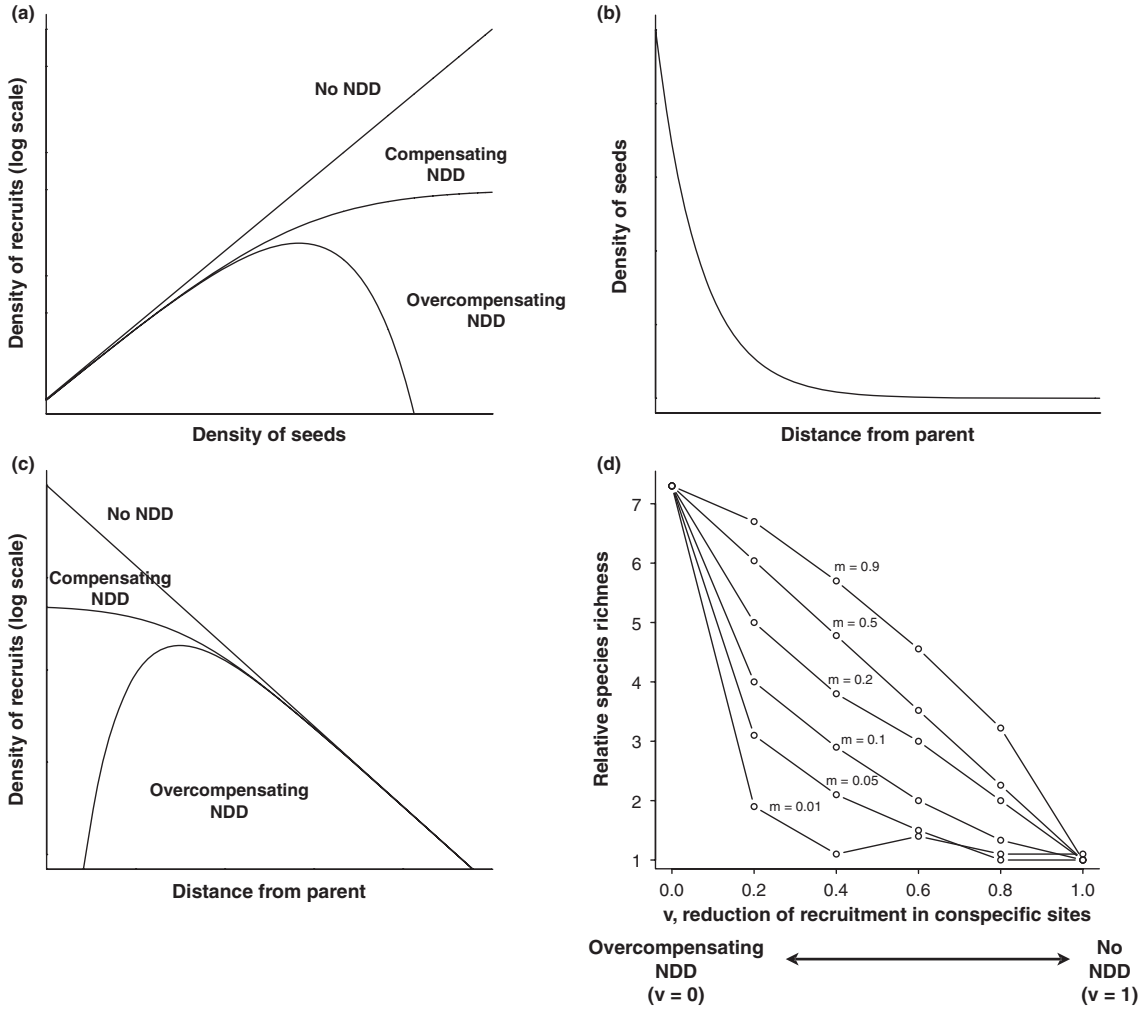
INTRODUCTION

Despite 40 years of intensive research, the mechanisms allowing species coexistence remain poorly understood, especially in species-rich communities such as tropical forests (Hubbell 2001; Wright 2002). One of the leading explanations for plant-species coexistence is the Janzen-Connell mechanism: when seeds or seedlings occur at high density or close to adult conspecifics they are more vulnerable to attack from specialized natural enemies such as pathogens and insect herbivores (Janzen 1970; Connell 1971). The Janzen-Connell hypothesis predicts that, as a consequence, the probability of a dead tree being replaced by a conspecific is low. This local negative density dependence (NDD) will promote diversity by ensuring that there is turnover in local species composition at the patch level. An accumulation of empirical evidence points to a role for this mechanism in species coexistence in plant communities in tropical and temperate regions (Harms *et al.* 2000; Petermann *et al.* 2008) through the action of both fungal pathogens (Bever 1994; Packer & Clay 2000; Klironomos 2002; Bell *et al.* 2006; Mangan *et al.* 2010) and insect herbivores (Sullivan 2003; Norghauer *et al.* 2006). Recent research suggests that local NDD may strongly influence the abundances of species and promote coexistence in tropical forest tree communities (Comita *et al.* 2010; Mangan *et al.* 2010).

In spite of this increasing evidence for NDD, the extent to which it contributes to species coexistence depends crucially on its functional form (Freckleton & Lewis 2006; Box 1). NDD simply means that *per capita* mortality increases with density, but the form of the relationship can be compensating, undercompensating or overcompensating (Fig. Box 1a). If NDD is undercompensating, the number of recruits increases with initial density, despite an increase in the proportion of individuals dying. Compensating NDD represents a special case where the number of recruits tends to an asymptote as initial density increases. When NDD is overcompensating, high initial seed and seedling densities lead to disproportionate increases in mortality and consequently, the resulting density of recruits is extremely low (Bellows 1981; Fig. Box 1c). Thus, the number of recruits will actually decline as initial density increases.

Box 1 The Janzen-Connell effect and its assumptions

The Janzen-Connell mechanism relies on assumptions about the relationship between seed (or seedling) density and the probability of individual recruitment, and the relationship between seed dispersal and distance from the parent plant. As shown in (a) the relationship between recruitment and density may be density independent, compensating or overcompensating. Seed dispersal is typically leptokurtic (b), so that most seeds land immediately beneath the parent tree. When this leptokurtic dispersal is combined with the density responses in (a), the net density of recruits depends on the form of density dependence as shown in (c). The Janzen-Connell mechanism is based on the prediction that when density dependence is overcompensating, recruitment fails beneath a parent tree. On the other hand, if density dependence is compensating, absent or undercompensating (intermediate between compensating or absent) then the highest densities of recruits occur immediately beneath the adults (c). The Janzen-Connell effect is a powerful mechanism: to illustrate this (d) shows the effect in operation in a simple model (described by Pacala 1997). In this model, there is a series of sites, each of which is occupied by an adult tree. Following the death of an adult, an empty site is occupied by another tree, the new occupier being determined by a simple lottery in which the probability of any species occupying being proportional to the number of seeds it disperses. To mimic the Janzen-Connell effect, the probability of recruitment of species i into a site formerly occupied by species i is reduced by a factor v. $v = 1$ corresponds to no density dependence (equivalent to a neutral model), whereas when $v = 0$ density dependence is completely overcompensating. The model assumes that a fraction of seed (m) disperses globally (varied between 0.01 and 0.9, indicated by the numbers above the lines in d), whereas the rest stays in the natal site. As shown, in (d) diversity is always maximized when density dependence is overcompensating. Moreover, when most seed lands in the parental site (global dispersal is 0.5 or less) the relationship is nonlinear so that the increases in diversity as a consequence of density dependence are greatest when v is small. This emphasizes that overcompensating density dependence is potentially an extremely powerful force, particularly when dispersal is low, exactly as envisaged in the original model (models were run for 1000 patches, with simulations lasting 5×10^5 generations. 30% of individuals died in each generation and the model was initiated with 50 identical species at time 0).



The Janzen-Connell mechanism, in its original formulation, depends on the creation of a 'death zone' beneath the canopy of the parent tree (Janzen 1970; Connell 1971) and this requires overcompensating NDD (Freckleton & Lewis 2006). As seed dispersal kernels are typically leptokurtic (Hubbell 1980), the highest initial densities of seeds and seedlings are generally found under parent trees. If NDD is overcompensating, these high initial densities will be subject to very high mortality and few conspecific survivors will remain under parent trees. Consequently, the gap created when a tree dies is unlikely to be filled by individuals of the same species. Without overcompensation, the density of recruits will always be highest beneath the parent (Hubbell 1980). Compensating and undercompensating NDD could contribute to coexistence (Muller-Landau & Adler 2007), but the mechanism would then differ from that envisaged by the Janzen-Connell hypothesis. The difference between these mechanisms is more than academic. Overcompensating NDD is much more effective at maintaining species coexistence, and this is especially true when dispersal is local (Fig. Box 1d).

Existing theoretical models demonstrating that NDD can contribute to species coexistence (Becker *et al.* 1985; Armstrong 1989) implicitly rely on overcompensation. More recent modelling has shown explicitly that overcompensating NDD can contribute to species coexistence (Kuang & Chesson 2008; Münkemüller *et al.* 2009). However, overcompensation is thought to be rare in plants (Rees & Crawley 1989, 1991; Freckleton & Watkinson 2002) and there are very few studies that show overcompensating density dependence in plant populations (Thrall *et al.* 1989; Buckley *et al.* 2001). In particular, the literature on tropical forests includes many examples of NDD (Harms *et al.* 2000; Hubbell *et al.* 2001; Bell *et al.* 2006; Comita *et al.* 2010), but to our knowledge, no previous study in natural systems has investigated the form of the density dependence or shown that NDD can result in 'death zones' in areas of initially high seed or seedling density.

We investigated the form of NDD caused by fungal pathogens of *Pleradenophora longicuspis* (Euphorbiaceae) seedlings in a tropical forest in Belize, Central America. Specifically, we addressed two hypotheses: (1) survival of *P. longicuspis* seedlings decreases disproportionately with initial seedling density and (2) fungal pathogens are the cause of this overcompensating NDD. We took a dual approach, using parallel experiments manipulating *P. longicuspis* density and pathogen presence both in a shadehouse and in the field. This allowed us to combine the precision of a controlled environment experiment with the ecological relevance of working with a natural population. Our results show that fungal pathogens caused strong and rapid overcompensating NDD in both experiments, consistent with the mechanism assumed by the Janzen-Connell hypothesis.

MATERIAL AND METHODS

Site description and study species

This study was conducted near the Las Cuevas Research Station within the Chiquibul Forest Reserve, Belize. Forests in this area have historically been subject to hurricanes and light selective logging, and are classified as Deciduous Seasonal Forest and Deciduous Semi-Evergreen Seasonal Forest (Wright *et al.* 1959). Annual rainfall is typically 1500 mm, with a dry season between February and May, and a humid season from June to January (Johnson & Chaffey 1973).

Pleradenophora longicuspis (Standl.) Esser (Euphorbiaceae) is among the most common tree species at this site (Bird 1998). Its seeds are dispersed ballistically in the late dry season (mid-May) and germination begins with the onset of the first heavy rains in early June. The resulting seedling carpets (up to 1650 seedlings m^{-2}) have been shown to undergo NDD survivorship as the result of fungal pathogens (Bell *et al.* 2006). Note that Bell *et al.* (2006) incorrectly refer to *P. longicuspis* as '*Sebastiana longicuspis*'.

Ongoing work at this site suggests that several pathogens isolated from *P. longicuspis* are able to infect other plant species. However, *P. longicuspis* seedlings are more likely than other species to become infected and die when experimentally exposed to certain strains isolated from *P. longicuspis* (R. Gallery, unpublished data). Other tropical forest studies of host–pathogen interactions have also found that while most fungi are polyphagous and only moderately pathogenic, certain strains demonstrate strong host-specific infectivity and/or pathogenicity (e.g. Gallery *et al.* 2007; Gilbert & Webb 2007).

Shadehouse experiment

Prior to seed dispersal, the upper *c.* 10 cm of soil and humic matter from four areas of high *P. longicuspis* adult density were used to fill seed trays (36 cm L × 24 cm W × 5 cm D). Soil was sieved to remove large stones, roots and seeds. The trays were placed in a shadehouse, covered with rain-permeable shade netting, in a small forest gap. Seedling densities were selected using a power-series with eight densities. The highest density (d_{max}; 4272 seeds m^{-2}) was four times the maximum seedling density previously recorded in the field (Bell *et al.* 2006). Within each block, the *i*th density was selected as a uniform random draw from the interval $\{d_{max}^{i/8}, d_{max}^{i+0.5/8}\}$.

Freshly dispersed *P. longicuspis* seeds were collected in seed traps. Seeds that were obviously unviable were discarded. Seeds were planted on a uniform lattice at the appropriate densities (but at least 20 mm from the tray edge) and covered by *c.* 5 mm of soil and a thin covering of homogenized, seed-free leaf litter from the same locations

as the corresponding soil. Fungicide (see below for details) and control treatments (application of water) were randomly allocated and initiated on 31 May 2008. Positions of trays within the shadehouse were randomized weekly. Seedling survival was recorded approximately every 5 days for 7 weeks.

Field experiment

Germination of *P. longicuspis* seedlings began in early June 2008. Four blocks, separated by 1–20 m, each comprising ten 0.25 m^2 plots were established within an area of high *P. longicuspis* adult and seedling density, allowing for a large range in initial seedling density. Within each block, plots were randomly assigned to density (1–5) and fungicide (water control or fungicide) treatments. We tagged and counted all *P. longicuspis* seedlings in each plot. The number of seedlings for each of the five density treatments was thinned to a number determined at random from within the five quintiles (1–20, 21–40, …, 81–100%) of the maximum observed density in the block. If the assigned number of seedlings exceeded that observed, the initial density was set to the observed density.

Survivors and recruits were censused weekly for 5 weeks. For the analyses of overall mortality in the field experiment, the density of seedlings (N_0) was calculated as the sum of the thinned density and the number of subsequent recruits.

Fungicide manipulations

In both experiments, fungicides were applied to one replicate of each density in each block to isolate pathogen-induced mortality from other processes. Ridomil Gold® and Amistar® (Syngenta Ltd, Basel, Switzerland) were used to provide broad-spectrum protection against fungi and oomycetes. These pesticides were effective against pathogens of *P. longicuspis* in previous work at this site (Bell *et al.* 2006). They were applied weekly, as a spray, in accordance with the manufacturer's guidelines: 0.25 g m^{-2} of Ridomil® and 0.005 g m^{-2} of Amistar®, each dissolved in 100 mL m^{-2} (shadehouse) or 50 mL m^{-2} (field) of water. Control trays and plots were sprayed with the volume of water used to dissolve the fungicides (200 and 100 mL m^{-2} in the shadehouse and field respectively). Screens were used to prevent spray drift between replicates.

Statistical analysis

The same statistical methodology was used for the analysis of both experiments. Initial analyses indicated that the density responses could not be modelled by a single density function. We therefore took a flexible approach, using generalized additive models (GAMs) to generate smoothed curves for the density responses (Fewster *et al.* 2000), and analysed these smoothed curves to determine the nature of the NDD in the different treatments.

The number of survivors at the end of the experiment was modelled as a function of fungicide treatment (F) and initial density (N_0) as

$$N_t|N_0,F = N_0 \cdot (1+\exp(-(\beta_0+\beta_1 F+f_0(N_0)+f_1(N_0)F)))^{-1}, \quad (1)$$

where β_0 represents the proportion of control seedlings surviving at low density and β_1 the fungicide treatment effect at low density. The functions, $f_i(N_0)$, represent cubic regression splines describing the effects of initial density on survival probability; f_0 represents the function for control seedlings and f_1 the difference between treatments. Regression splines were restricted to a maximum of three knots, identified by cross-validation, to prevent under-smoothing (Wood 2006). Models were fitted separately to the shadehouse and field data using GAMs (Hastie & Tibshirani 1990) assuming a quasi-binomial error distribution. We excluded one replicate from the fungicide treatment of both experiments because many seedlings were infected despite fungicide application.

Confidence envelopes were estimated using nonparametric bootstrapping with 1000 iterations (Fewster *et al.* 2000). At each iteration, we randomly selected M trays (or plots), with replacement, where M is the total number of trays (or plots) in the experiment. Sub-samples were stratified by fungicide and initial density treatments. Therefore, each sub-sample contained the same number of trays (or plots) from each fungicide treatment and density quantile as the original data. The GAM was refitted to these data. This model was used to predict the survival probabilities of seedlings over the range of initial densities in the sub-sample of the data to which it was fitted. The 95% quantiles of these predictions were estimated at each initial density within the range of the complete data set.

To determine the form of NDD, we numerically differentiated the GAM functions describing the relationship between N_0 and N_1 for both fungicide and control treatments $g(N_0,F)$ with respect to N_0. Without NDD, this differentiated function, g', is independent of initial density. A negative relationship between g' and N_0 indicates NDD. The form of NDD can be inferred from the value of g' at high densities, with $g' > 0$ for undercompensation and $g' < 0$ for overcompensation. We numerically differentiated the GAM functions to estimate their slopes along the range of initial densities at intervals of 10. This smoothed out small irregularities in the model fits, yet retained a resolution suitable for later inferences. To estimate confidence intervals for g', we numerically differentiated

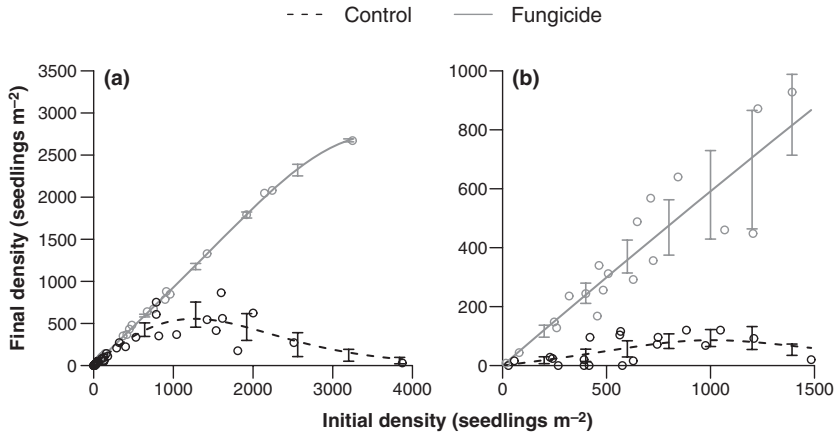


Figure 1 The relationship between initial and final density of *Pleradenophora longicuspis* seedlings in (a) the shadehouse and (b) the field. Survival was compared between seedlings sprayed with water (control) and seedlings treated with systemic fungicides. The initial number of seedlings was manipulated in both experiments and the number of survivors monitored over seven (shadehouse) or five (field) weeks. Error bars represent 95% confidence intervals and the lines are the predictions of the fitted models described by eqn 1.

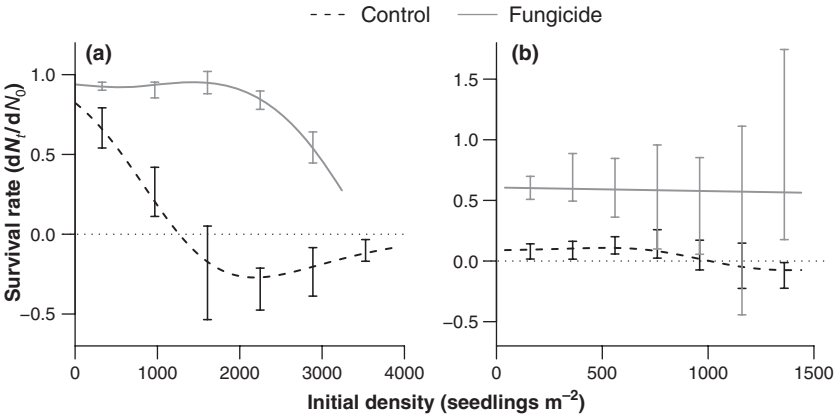


Figure 2 The form of density dependence in *Pleradenophora longicuspis* seedlings in (a) the shadehouse and (b) the field. Negative relationships between dN_t/dN_0 and initial density indicate negative density dependence. Overcompensation is inferred when $dN_t/dN_0 < 0$. The form of the density response was compared between seedlings sprayed with water (control) and seedlings treated with systemic fungicides. Error bars represent 95% confidence intervals.

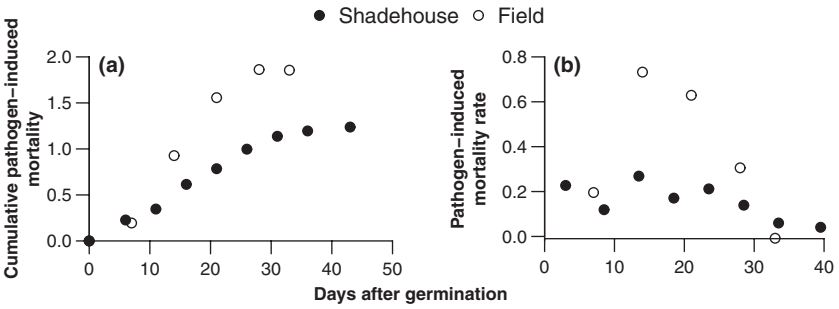


Figure 3 The time course of mortality from fungal pathogens in *Pleradenophora longicuspis* seedlings in the shadehouse and field experiments. This is expressed as (a) the log ratio of survivors between the fungicide and control treatments at each census (D_t) and (b) the differential of D_t: the rate of pathogen-induced mortality (k_t).

the functions from the bootstrapping procedure in the same way. The 95% confidence intervals were estimated as the 95% quantiles of the slopes at each interval (Fewster *et al.* 2000).

Finally, we explored the temporal patterns of pathogen-induced mortality by estimating the difference in surviving seedlings between the fungicide and control treatments. Specifically, we used GAMs to model the number of survivors at each census (N_t) as functions of initial density and fungicide treatment. We then numerically integrated the functions for the fungicide ($N_{t,F}$) and control ($N_{t,C}$) treatments over the range of initial densities. We calculated the cumulative effect of pathogens on mortality to time t as $D_t = \log(N_{t,F}/N_{t,C})$. Numerically differentiating the relationship between D_t and time gives a measure of the intensity of pathogen-caused mortality in each census interval, k_t, effectively the k-value (Varley & Gradwell 1960; Haldane 1992).

RESULTS

Seedling survival was dramatically lower in the control (no fungicide) treatments of both experiments (Fig. 1). Furthermore, high initial densities resulted in very low numbers of recruits in the control treatments of both experiments, indicating overcompensating NDD (Fig. 1). Strong NDD in the control treatments of both experiments was confirmed by the clear negative relationship between the slope of the density response and initial density (Fig. 2). Specifically, the slope of the density response dropped below zero at high densities in both experiments, demonstrating overcompensation. Fungicide treatment removed the density response in the field experiment, suggesting that fungal pathogens were wholly responsible for the NDD. In the shadehouse, slight (undercompensating) NDD was observed in the fungicide treatment at the highest densities, perhaps because seedlings reached a density high enough to generate intraspecific resource competition.

In the field experiment, almost all pathogen-related mortality occurred during the first 4 weeks (Fig. 3a). In the control (no fungicide) treatment, the mortality rate peaked in the second week and declined thereafter (Fig. 3b). There were very few deaths in the control treatment after the fourth week. Similarly, 90% of the observed deaths in the shadehouse experiment occurred during the first 4 weeks (Fig. 3a) and the mortality rate declined thereafter (Fig. 3b).

DISCUSSION

Our results demonstrate rapid, overcompensating density-dependent mortality in seedlings of *P. longicuspis*, driven by pathogens. To our knowledge, our data are the first to reveal the functional form of density dependence resulting from pathogen-induced mortality and show that it is consistent with the Janzen-Connell mechanism in a natural system. Moreover, our data represent one of only a few examples to date of overcompensating density responses in plant populations (Thrall *et al.* 1989; Crone & Taylor 1996; Buckley *et al.* 2001). Although most previous studies have not enumerated the exact form of density dependence, it is very possible that many do indeed exhibit overcompensation. If overcompensating density dependence is widespread for tree species in this and other tropical forests, it may represent a powerful demographic mechanism promoting local diversity.

As argued above, the form of local density dependence should have consequences for the dynamics of turnover of gap occupancy. Overcompensating density dependence should generate cyclical dynamics of gap occupancy: a space currently occupied by one species should, following the death of the occupant, become occupied by a different species. Replacement by the same species will be unlikely, as the survival of seedlings in the vicinity of the parent will be low. This is a macroecological prediction that should be testable using long-term survey data.

While the Janzen-Connell effect is a leading hypothesis for explaining species coexistence in tropical forests through density-dependent attack by natural enemies, alternative mechanisms have also been proposed. For example, local density dependence of whatever form (overcompensating or not) can generate population-level density-dependent recruitment that limits population sizes and thus enables multiple species to coexist (Chesson 2000; Adler & Muller-Landau 2005; Muller-Landau & Adler 2007). Such a mechanism is different and does not rely on the local restriction of recruitment that is inherent in the Janzen-Connell mechanism. In contrast, the Janzen-Connell hypothesis proposes that coexistence is driven by local recruitment effects and their consequences for species replacement at individual sites. Our results indicate that the magnitude of pathogen-induced mortality is very sensitive to local densities, perhaps because infected seedlings act as sources of further infection. If this mechanism is general, then models need to account for the details of how local transmission and density dependence translate into population-level effects (e.g. Adler & Muller-Landau 2005; Muller-Landau & Adler 2007).

Overcompensating density dependence is considered to be rare in plant populations (Rees & Crawley 1989, 1991; Freckleton & Watkinson 2002). This is because, conventionally, plant populations are thought to be regulated by density dependence resulting from resource competition affecting growth and fecundity (Watkinson 1980). Such density dependence is typically compensating: for example, the 'law of constant final yield' is a compensating density-dependent response common in plants (Kira *et al.* 1956). However, the lack of examples of overcompensating density dependence in the literature may also partly result from the relative paucity of studies that have characterized density responses for aspects of performance other than yield. Density dependence at other stages in the life-cycle has been found to be overcompensating. Other studies have found evidence of density-dependent survival of seedlings and greatly reduced survival beneath adult crowns, consistent with our findings (Silva Matos *et al.* 1999). Delayed flowering (Buckley *et al.* 2001) and reduced seed viability (Thrall *et al.* 1989) at high densities have been demonstrated to lead to overcompensating density dependence in some annual plant populations. Additionally, both natural enemies and maternal effects have been implicated in causing nonlinear population dynamics in plants (Crone & Taylor 1996). The effects of natural enemies have often been excluded in studies estimating the form of density dependence in plants. In this study, pathogens clearly drove the

overcompensation we observed. We only considered a single stage in the life-cycle, and therefore our conclusions must be cautious. However, previous work has shown that strong density-dependent regulation at an early stage can cause density dependence of overall population dynamics (Silva Matos *et al.* 1999). Perhaps the action of natural enemies means that overcompensation in natural plant populations may be more common than suggested by the evidence currently available.

The search for coexistence mechanisms has been largely driven by the suggestion that the number of stably coexisting species cannot exceed the number of limiting resources (Hutchinson 1961; Macarthur & Levins 1964). However, Levins (1979) demonstrated theoretically that in a fluctuating environment this limit can be expanded to include the number of resources plus the number of distinct nonlinearities. Several further studies have demonstrated that species-specific nonlinear responses of population growth to limiting factors and predation, including overcompensating density dependence, can contribute to species coexistence (Crone 1997; Chesson 2000; Kuang & Chesson 2008; Münkemüller *et al.* 2009). If the species occupying a site shows overcompensating dynamics, it increases the likelihood of invasion by another, less competitive species. *Pleradenophora longicuspis* has a clustered spatial distribution in our study area and occurs locally at high densities. These aggregations are likely established after hurricanes, which affect the area at regular intervals. Making conclusions about coexistence from studies of one species would be premature, but our results do suggest that the nonlinear dynamics of *P. longicuspis* seedlings may potentially permit replacement by other species.

Similar experiments on other species are now needed to determine the prevalence of overcompensating NDD in tropical plant communities. Experimental approaches of the sort we describe will be necessary, both to rule out intraspecific competition as the cause of NDD, and to control for the fact that natural variations in seedling density may be confounded with pathogen incidence and environmental variables that affect mortality from pathogens. Furthermore, our results highlight the importance of targeting studies at very young seedlings. We have shown that in *P. longicuspis* pathogen-driven NDD occurs in a short period immediately after germination. Other studies have also highlighted this period as crucial for density dependence and the maintenance of species diversity (Harms *et al.* 2000; Hille Ris Lambers *et al.* 2002). While important, studies of seedlings and saplings in older age classes (Hubbell *et al.* 2001; Comita *et al.* 2010) may thus overlook a key stage in plant population dynamics, leading to an underestimation of the role of overcompensating density dependence in seedling demographics and the maintenance of diversity (Harms *et al.* 2000).

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REFERENCES

Adler, F.R. & Muller-Landau, H.C. (2005). When do localized natural enemies increase species richness? *Ecol. Lett.*, 8, 438–447.

Armstrong, R.A. (1989). Competition, seed predation and species coexistence. *J. Theor. Biol.*, 141, 191–195.

Becker, P., Lee, L.W., Rothman, E.D. & Hamilton, W.D. (1985). Seed predation and the coexistence of tree species: Hubbell's models revisited. *Oikos*, 44, 382–390.

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

Bellows, T.S. (1981). The descriptive properties of some models for density dependence. *J. Anim. Ecol.*, 50, 139–156.

Bever, J.D. (1994). Feedback between plants and their soil communities in an old field community. *Ecology*, 75, 1965–1977.

Bird, N.M. (1998). *Sustaining the Yield. Improved Timber Harvesting Practices in Belize, 1992-1998*. Natural Resources Institute, Chatham.

Buckley, Y.M., Hinz, H.L., Matthies, D. & Rees, M. (2001). Interactions between density-dependent processes population dynamics and control of an invasive plant species, *Tripleurospermum perforatum* (scentless chamomile). *Ecol. Lett.*, 4, 551–558.

Chesson, P. (2000). Mechanisms of maintenance of species diversity. *Annu. Rev. Ecol. Syst.*, 31, 343–366.

Comita, L.S., Muller-Landau, H.C., Aguilar, S. & Hubbell, S.P. (2010) Asymmetric density dependence shapes species abundances in a tropical tree community. *Science,* 329, 330–332.

Connell, J.H. (1971). On the role of natural enemies in preventing competitive exclusion in some marine animals and in rain forest trees. In: *Dynamics of Numbers in Populations* (eds den Boer, P.J. & Gradwell, G.R.). PUDOC, Wageningen, The Netherlands, pp. 298–312.

Crone, E.E. (1997). Delayed density dependence and the stability of interacting populations and subpopulations. *Theor. Popul. Biol.*, 51, 67–76.

Crone, E.E. & Taylor, D.R. (1996). Complex dynamics in experimental populations of an annual plant, *Cardamine pensylvanica*. *Ecology*, 77, 289–299.

Fewster, R.M., Buckland, S.T., Siriwardena, S.M., Baillie, S.R. & Wilson, J.D. (2000). Population trends for farmland birds using generalized additive models. *Ecology*, 81, 1970–1984.

Freckleton, R.P. & Lewis, O.T. (2006). Pathogens, density dependence and the coexistence of tropical trees. *Proc. R. Soc. Lond. B*, 273, 2909–2916.

Freckleton, R.P. & Watkinson, A.R. (2002). Are weed population dynamics chaotic? *J. Appl. Ecol.*, 39, 699–707.

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.

Gilbert, G.S. & Webb, C.O. (2007). Phylogenetic signal in plant pathogen-host range. *Proc. Natl. Acad. Sci. USA*, 104, 4979–4983.

Haldane, J.B.S. (1992). Disease and Evolution (Reprinted from La Ricerca Scientifica Supplemento, Vol. 19, Pg 1–11, 1949). *Curr. Sci.*, 63, 599–604.

Harms, K.E., Wright, S.J., Calderón, O., Hernández, A. & Herre, E.A. (2000). Pervasive density-dependent recruitment enhances seedling diversity in a tropical forest. *Nature*, 404, 493–495.

Hastie, T.J. & Tibshirani, R. (1990). *Generalized Additive Models*. Chapman and Hall, London.

Hille Ris Lambers, J., Clark, J.S. & Beckage, B. (2002). Density-dependent mortality and the latitudinal gradient in species diversity. *Nature*, 417, 732–735.

Hubbell, S.P. (1980). Seed predation and the coexistence of tree species in tropical forests. *Oikos*, 35, 214–229.

Hubbell, S.P. (2001). *The Unified Neutral Theory of Biodiversity and Biogeography*. Princeton University Press, Princeton.

Hubbell, S.P., Ahumada, J.A., Condit, R. & Foster, R.B. (2001). Local neighborhood effects on long-term survival of individual trees in a neotropical forest. *Ecol. Res.*, 16, 859–875.

Hutchinson, G.E. (1961). The Paradox of the plankton. *Am. Nat.*, 95, 137–145.

Janzen, D.H. (1970). Herbivores and the number of tree species in tropical forests. *Am. Nat.*, 104, 501–528.

Johnson, M.S. & Chaffey, D.R. (1973). *An Inventory of the Chiquibul Forest Reserve, Belize*. Overseas Development Administration, Surrey, UK.

Kira, T., Ogawa, H., Hozumi, K., Koyama, H. & Yoda, K. (1956). Intraspecific competition among higher plants V. Supplementary notes on the C-D effect. *J. Inst. Polytech. Osaka City Univ. Ser. D Biol.*, 7, 1–14.

Klironomos, J.N. (2002). Feedback with soil biota contributes to plant rarity and invasiveness in communities. *Nature*, 417, 67–70.

Kuang, J.J. & Chesson, P. (2008). Predation-competition interactions for seasonally recruiting species. *Am. Nat.*, 171, E119–E133.

Levins, R. (1979). Coexistence in a variable environment. *Am. Nat.*, 114, 765–183.

Macarthur, R. & Levins, R. (1964). Competition, habitat selection and character selection in a patchy environment. *Proc. Natl. Acad. Sci. USA*, 51, 1207–1210.

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

Muller-Landau, H.C. & Adler, F.R. (2007). How seed dispersal affects interactions with specialized natural enemies and their contribution to the maintenance of diversity. In: *Seed Dispersal: Theory and its Application in a Changing World* (eds Dennis, A.J., Schupp, E.W., Green, R.J. & Westcott, D.A.). CABI Wallingford, UK, pp. 407–426.

Münkemüller, T., Bugmann, H. & Johst, K. (2009). Hutchinson revisited: patterns of density regulation and the coexistence of strong competitors. *J. Theor. Biol.*, 259, 109–117.

Norghauer, J.M., Malcolm, J.R. & Zimmerman, B.L. (2006). Juvenile mortality and attacks by a specialist herbivore increase with conspecific adult basal area of Amazonian *Swietenia macrophylla* (Meliaceae). *J. Trop. Ecol.*, 22, 451–460.

Pacala, S.W. (1997). Dynamics of plant communities. In: *Plant Ecology* (ed. Crawley, M.J.). Blackwell Science, Oxford, pp. 532–555.

Packer, A. & Clay, K. (2000). Soil pathogens and spatial patterns of seedling mortality in a temperate tree. *Nature*, 404, 278–281.

Petermann, J.S., Fergus, A.J.F., Turnbull, L.A. & Schmid, B. (2008). Janzen-Connell effects are widespread and strong enough to maintain diversity in grasslands. *Ecology*, 89, 2399–2406.

Rees, M. & Crawley, M.J. (1989). Growth, reproduction and population dynamics. *Funct. Ecol.*, 3, 645–653.

Rees, M. & Crawley, M.J. (1991). Do plant-populations cycle. *Funct. Ecol.*, 5, 580–582.

Silva Matos, D.M., Freckleton, R.P. & Watkinson, A.R. (1999). The role of density dependence in the population dynamics of a tropical palm. *Ecology*, 80, 2635–2650.

Sullivan, J.J. (2003). Density-dependent shoot-borer herbivory increases the age of first reproduction and mortality of neotropical tree saplings. *Oecologia*, 136, 96–106.

Thrall, P.H., Pacala, S.W. & Silander, J.A. (1989). Oscillatory dynamics in populations of an annual weed species *Abutilon theophrasti*. *J. Ecol.*, 77, 1135–1149.

Varley, G.C. & Gradwell, G.R. (1960). Key factors in population studies. *J. Anim. Ecol.*, 29, 399–401.

Watkinson, A.R. (1980). Density-dependence in single-species populations of plants. *J. Theor. Biol.*, 83, 345–357.

Wood, S.N. (2006). *Generalized Additive Models: An Introduction With R*. Chapman and Hall/CRC, Boca Raton, FL, USA.

Wright, S.J. (2002). Plant diversity in tropical forests: a review of mechanisms of species coexistence. *Oecologia*, 130, 1–14.

Wright, A.C.S., Romney, D.H., Arbuckle, R.H. & Vial, V.E. (1959). *Land in British Honduras*. H.M. Stationery Office, London.

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