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REVIEW AND SYNTHESIS

Bias in the detection of negative density dependence in plant communities

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Abstract

Regression dilution is a statistical inference bias that causes underestimation of the strength of dependency between two variables when the predictors are error-prone proxies (EPPs). EPPs are widely used in plant community studies focused on negative density-dependence (NDD) to quantify competitive interactions. Because of the nature of the bias, conspecific NDD is often overestimated in recruitment analyses, and in some cases, can be erroneously detected when absent. In contrast, for survival analyses, EPPs typically cause NDD to be underestimated, but underestimation is more severe for abundant species and for heterospecific effects, thereby generating spurious negative relationships between the strength of NDD and the abundances of con- and heterospecifics. This can explain why many studies observed rare species to suffer more severely from conspecific NDD, and heterospecific effects to be disproportionally smaller than conspecific effects. In general, such species-dependent bias is often related to traits associated with likely mechanisms of NDD, which creates false patterns and complicates the ecological interpretation of the analyses. Classic examples taken from literature and simulations demonstrate that this bias has been pervasive, which calls into question the emerging paradigm that intraspecific competition has been demonstrated by direct field measurements to be generally stronger than interspecific competition.

Keywords

Errors in predictors, measurement error, negative density dependence, plant community, regression dilution.

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INTRODUCTION

Understanding and quantifying density-dependent population regulation is essential to design effective measures to preserve biodiversity and manage the planet's ecosystems (Tilman 2000). The stable maintenance of local species diversity requires conspecific negative density dependence (CNDD), in which each species' population growth rate declines as its abundance increases (Chesson 2000). Because measures of CNDD characterise deterministic forces that keep each species from extinction when at low density, detecting and quantifying CNDD have been principal goals of basic and applied ecologists. The strength of CNDD has profound practical implications in conservation, from the sizes of ecological reserves to the design of interventions intended to rescue critically endangered species. The alternative hypothesis that species abundances drift randomly relative to one another without any CNDD (Hubbell 2009), implies a different set of conservation measures. For example, in the presence of strong CNDD, reserves can be much smaller than in the absence of CNDD. Conversely, in presence of stochastic drift, human intervention would be required to stop the drift to extinction of rare species, even in pristine locations.

Over the last two decades, plant ecologists have developed a large body of evidence purporting to detect CNDD in many forest and grassland plant species and to quantify patterns in the strength of CNDD and the relative strength of heterospecific negative density dependence (HNDD) among species and ecosystems (Adler *et al.* 2018). The body of work on forest tree species is particularly comprehensive. CNDD is reported to be nearly ubiquitous in tropical forests (Harms *et al.* 2000), to be generally stronger in tropical than in temperate forests (LaManna *et al.* 2017b), and within forests, to be stronger for rare than common species (Comita *et al.* 2010). These three papers are part of a growing literature that takes advantage of the network of permanent forest census plots to measure CNDD and HNDD for many species within each community and across communities. Here, we focus on these community-scale studies and the striking results that emerge from their statistical examination of many within- and between-species density-dependent interactions.

Collectively, the results in Harms *et al.* (2000), Comita *et al.* (2010) and LaManna *et al.* (2017b), hereafter H2000, C2010 and L2017, are consistent with the hypothesis that specialist natural enemies maintain forest diversity (the Janzen-Connell Hypothesis, Janzen, 1970), and maintain the tropical–temperate gradient in tree species diversity. CNDD is also reported to be generally stronger than HNDD in forests and grasslands, which would facilitate coexistence (Adler *et al.* 2018). The growing prominence and maturation of this work has now led to the first call to develop forest management practices that take advantage of CNDD (Cordonnier *et al.* 2018).

This development is reminiscent of a previous episode in ecology that deserves more widespread visibility. Prior to the publication of Walters & Ludwig (1981), CNDD was thought to be strong and easily quantified in fish communities.

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Perhaps the most important relationship used to manage commercial fisheries – the stock–recruitment curve – often showed strong evidence of CNDD, like the red curves rather than the black line in Fig. 1b,c. The black lines in Fig. 1 show no CNDD because the number of seedlings or saplings (recruits) is simply proportional to the number of seeds and parents (stock). In contrast, the flatness of the red curves exhibits strong CNDD because the number of recruits per unit stock decreases as the amount of stock increases.

However, Walters & Ludwig (1981) argued that many published examples of CNDD in stock–recruitment curves were statistical artifacts, produced by errors in the predictor (the stock) when used with regression methods that assume errors only in the dependent variable (recruits). This 'regression dilution' problem was first explained more than a century ago (Spearman 1904). Errors in the predictor of a generalised linear model (GLM) regression cause the estimated slope to be too small, and induce curvilinearity if the intercept is forced to be zero or the regression is performed after log-transforming the abundances (see below). Indeed, the points in Fig. 1b,c were obtained by introducing measurement errors in the conspecific density (number of seeds or adults), and the red curve was estimated from these points. Regression dilution caused repeated crashes of fisheries around the world, by convincing managers of the mistaken idea that they could harvest the stock down to low levels and still maintain high recruitment (Mason 2002). A similar problem has been recognised in animal ecology (Shenk *et al.* 1998; Dennis *et al.* 2006; Freckleton *et al.* 2006).

In this paper, we argue that also many of the most prominent results in the plant CNDD literature are likely to be affected by statistical artifacts produced because of a wide variety of errors in the predictors.

Note that the residuals in Figure 1c would be highly unbalanced if one were to fit a line with a zero intercept to the data. These patterns, therefore, typically cause researchers to fit a curvilinear relationship, often by log-transforming the data before linear regression. For example, suppose that the number of seedlings is constant times the number of seeds raised to the power b:

$$seedlings = a \times seeds^b.$$

If $b = 1$, the germination probability is the constant a and so there is no CNDD. But, if $b < 1$, the fraction surviving decreases as seeds increases, which is CNDD. Power functions are often fitted using the equivalent linear log–log relationship: $\log(\text{seedlings}) = log(a) + blog(\text{seeds})$. Thus, a test for the presence of CNDD is that the slope of a linear regression of log(seedlings) against log(seeds) is less than one. Because regression dilution flattens slopes, estimates of b in situations with no CNDD are biased beneath the correct value of one, which falsely implies the presence of CNDD.

Detection of CNDD and Error-Prone Proxies (EPPs)

A lot more is known about the biases created in regressions by errors in predictors than when Walters & Ludwig (1981) was written (Stefansky & Carroll 1985; Frost & Thompson 2000; Carroll *et al.* 2006; Buonaccorsi 2010; Grace 2017). The subject is considerably more complex than simple regression dilution and is also necessarily mathematical (see the typology of measurement errors in Box). Box summarises the most common statistical methods to detect CNDD from observational data. When one or more of the covariates in eqn 1 contains errors, parameter estimation can be biased leading to biased or apparent CNDD (false detections), depending on the type of data and model used. In general, we can identify two sources of error: measurement errors, and errors induced by choosing the wrong scale or the wrong metric (e.g. the number of individuals as a proxy for below-ground biomass). The first source of error can be accounted for by including repeated measurements in the sampling. The other sources of

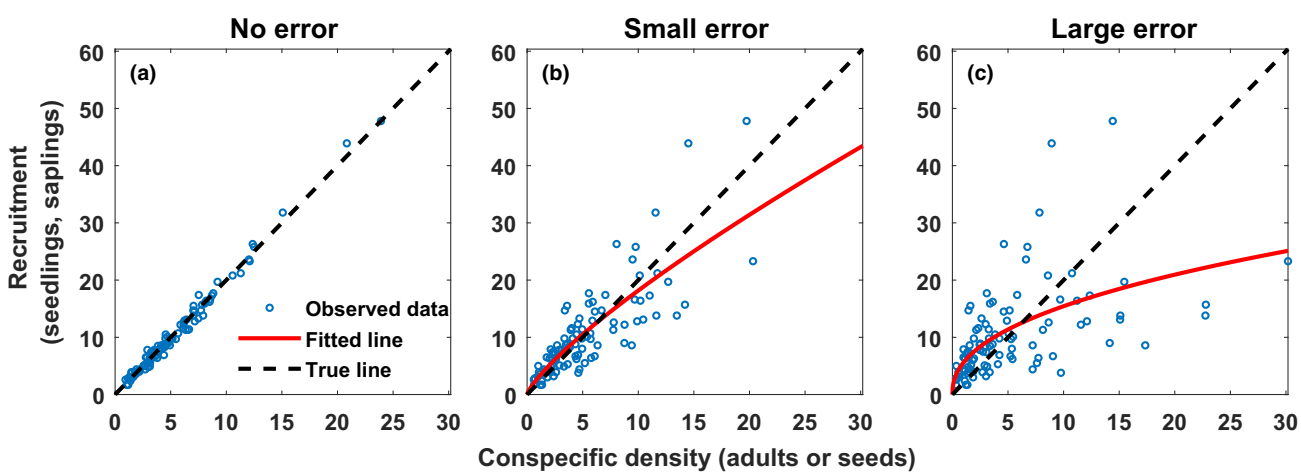

Figure 1 Examples of apparent CNDD produced by errors in the independent variable (conspecific density). In all examples recruitment is drawn from a Poisson distribution proportional to conspecific density, that is, no CNDD. (a) Conspecific density is observed without error. (b) Conspecific density is observed with a small multiplicative error $x_{obs} = xe^u$, where u is a random variable with variance $\sigma_u^2 < \sigma_{log(x)}^2$. (c) Conspecific density is observed with a large multiplicative error, $\sigma_u^2 \approx \sigma_{log(x)}^2$. The black line is the true relationship between conspecific density and recruitment, the red line is fitted with the model $y = ax^b$.

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Box 1. Measurement error typology

Regression dilution is a problem in statistical inference first identified by Spearman (1904) which causes the underestimation of the strength of dependency among variables when the predictors (independent variables) contain errors. To understand the problem, consider the OLS estimator of the slope in a linear regression model $y = a + bx$, given by the ratio between the covariance of x and y and the variance of x:

$$b_{\mathrm{OLS}} = \frac{cov(x, y)}{\sigma_x^2}. \tag{1}$$

Errors in the observed response variable ($y_{obs} = y + err$) increase the variability in the slope estimation without introducing bias, as the errors are by nature uncorrelated with x and do not affect the covariance. For this reason, classical measuring errors (additive and uncorrelated with the covariates) in the response variables are generally ignored in regression analysis as they are absorbed into the error residuals (Abrevaya & Hausman 2004). However, errors in the observed predictor variable ($x_{obs} = x + u$) cause an inflation of the denominator in the ratio of eqn 1, so the magnitude of the slope is biased towards zero.

$$b_{\mathrm{OLS}} = \frac{cov(x, y)}{\sigma_x^2 + \sigma_u^2}. \tag{2}$$

In general, the magnitude and the direction of the effect of measurement error depend on the model under consideration and on the joint distribution of the measurement error and the other variables. The theory of measuring error distinguishes between three types of error: classic, Berkson and differential (Carroll *et al.* 2006). OLS regression provides a tractable conceptual framework for understanding the origin of the bias for each type of error. For classic additive errors, the true value x is observed as $x_{obs} = x + u$, where u represents random noise with mean zero and variance σ_u^2. The naïve OLS estimator b_{OLS} can be expressed as

$$b_{\mathrm{OLS}} = b\frac{\sigma_x^2}{\sigma_x^2 + \sigma_u^2}, \tag{3}$$

From eqn 3 is clear that b_{OLS} will always be underestimated, and the larger the error in the predictor (σ_u^2) the larger the bias. Equation 3 also provides a means to correct for the bias if both, σ_x^2 and σ_u^2 are independently estimated. A similar problem occurs with multiplicative errors, that is, $x_{obs} = xe^u$, where $u \sim N(-\sigma_u^2/2, \sigma_u^2)$. Although a log-transformation will make the error additive, some researchers prefer to perform the analyses in the original scale. The naïve OLS estimator is given by (Hwang 1986, eqn 2.12):

$$b_{\mathrm{OLS}} = b\frac{\sigma_x^2}{\sigma_x^2 + \left(\mu_x^2 + \sigma_x^2\right)\left(exp(\sigma_u^2) - 1\right)}, \tag{4}$$

which is qualitatively similar to eqn 3, except that the bias depends also on the mean of x.

In contrast, Berkson error assumes that $x = x_{obs} + u$ and $x = x_{obs}e^u$, for the additive and multiplicative error respectively, so the error is independent of x_{obs} but not of x (note that the positions of x and x_{obs} are reversed relative to classical additive case). Such error occurs, for example, when a continuous variable is forced into discrete categories, when random replicates of a manipulative experiment are assigned to the same treatment or when an allometric relationship is used instead of a true size. Berkson error always decreases the variance of the observed variables, whereas the opposite is true for the classic errors. Berkson error reduces statistical power without causing a bias in the OLS estimator. However, in nonlinear models, e.g. logistic regression, Berkson errors produce bias (Heid *et al.* 2002).

In general, measuring errors will contain both, classic (u) and Berkson (v) components. The mixture of classic and Berkson additive error is represented as:

$$x = z + v, \tag{5a}$$
$$x_{obs} = z + u, \tag{5b}$$

where u and v are independent random noises and z a common random variable. Note that $v = 0$ corresponds to the classic error and $u = 0$ to the Berkson error. The naïve OLS estimator can be expressed as

$$b_{\mathrm{OLS}} = b\frac{\sigma_z^2}{\sigma_z^2 + \sigma_u^2}. \tag{6}$$

Differential error is defined as an error whose magnitude or direction depends on the response variable. In this case u can be correlated with the error residual ε, that is, $\mathrm{cov}(u,\varepsilon) \neq 0$. This can occur, for example, when individuals are divided in two groups and one group is used as the response and the other group is used as the predictor in a regression analysis. Any classification error will result in an overestimation of one variable and underestimation of the other, and vice versa, that is, $cov(u, \varepsilon) < 0$. For differential errors, the OLS estimator is given by (Carroll *et al.* 2006):

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$$b_{\mathrm{OLS}} = \frac{b\sigma_x^2 + cov(u, \varepsilon)}{\sigma_x^2 + \sigma_u^2}, \tag{7}$$

so the bias can be positive or negative and it depends not only on the magnitude of the error in x, σ_u^2, but also on the magnitude and sign of the $cov(u,\varepsilon)$.

GLMs and nonparametric estimators also are affected by measuring errors (Armstrong 1985; Stefansky & Carroll 1985; Fan & Young 1993). As the likelihood maximisation does not have a closed solution, the mathematical formulation of the bias is not always available. (see Fig. S2–S3 for additional examples).

error can only be resolved by mechanistic understanding of the effects of conspecifics, in particular the nature and the range of the interactions, including maternity when assessing adult–recruitment relationships.

To get a flavour of the issues involved without mathematical formalism, consider the following three simple numerical examples (Fig. 2), which qualitatively correspond to three of the most prominent papers in the forest CNDD literature:

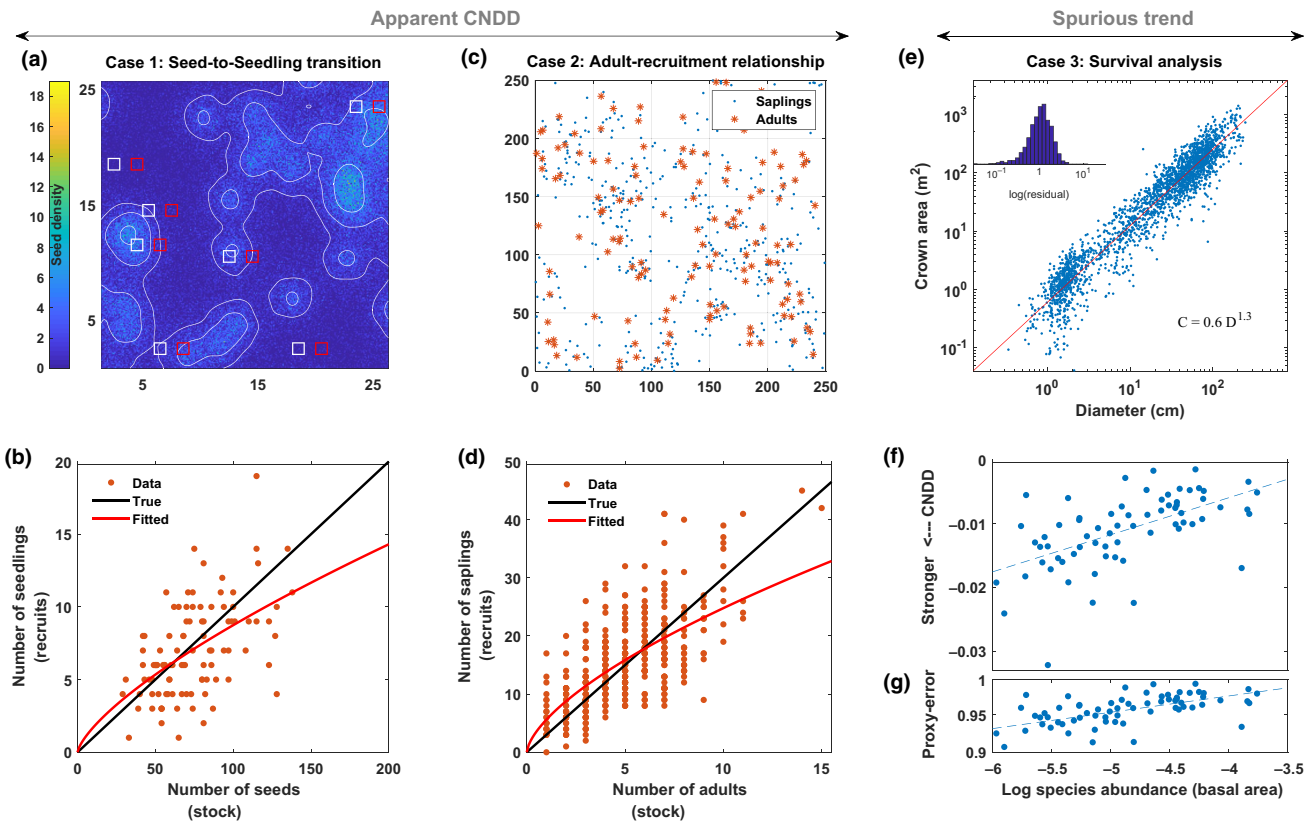

Figure 2 Error-prone proxies can cause apparent CNDD or spurious trend. *Case 1:* Apparent CNDD in a simulated study of a system with random seed germination. Colors in (a) give the intensity of the seed shadow. The seeds and seedlings are censused in paired 1m^2 seed traps and seedling plots (white and red quadrats, respectively, in (a) located 1 m from one another. The number of seeds before germination in each seedling plot is estimated with error, causing an apparent curvilinear relationship between number of seeds and number of seedlings (red line in b). *Case 2*: Apparent CNDD in a simulated study of a system in which saplings are randomly produced by adults. Recruits (blue points in c) are randomly produced by adults (red symbols) and dispersed using a fat-tail dispersal kernel with mean dispersal equal to 15 m. The number of juveniles and adults are counted in 50x50 m quadrats. Because some juveniles have parents outside their quadrat, the parents are measured with errors causing an apparent curvilinear relationship between numbers of adults and juveniles in the quadrats (red line in d). *Case 3:* Apparent trend between species abundance and the strength of CNDD, in simulated study in which the strength of CNDD is the same for all species. Density-dependent survival is simulated for saplings of 68 species that span a large range of abundances on a 50 ha plot on Barro Colorado Island (Panama), using the actual locations and diameters of saplings (≤ 10 cm diameter) and adults (> 10 cm) and local conspecific density given by the sum of the crown areas of conspecifics within 5 m. Each individual is assigned a 'true' crown area using an empirical allometric equation for trees measured in central Panama (e, Cano et al., 2019) times a lognormal random variable with mean one and variance equal to the residual variance from the allometric regression (insert in e). But, the measure of sum of local basal areas rather than sum of crown areas is then used in the logistic regressions that estimate the strength of CNDD for each species. The error in the proxy, $var(Z_{proxy} - Z_{true})/var(Z_{proxy})$, caused by using basal area instead of the true crowns increases with the abundance of the species (g). Because of this, regression dilution increases with a species' abundance, and causes a false trend in which the estimated strength of CNDD increases as abundance decreases (f).

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Box 2. The estimation of CNDD

CNDD affecting transitions between life stages can be detected by fitting a demographic model to field data that describes the transition from one stage to the next (e.g., from seed to seedling, from an initial size to a later size, etc.), of the form

$$N_1 = p(N_0, Z_1, Z_2, \ldots, Z_m)N_0, \tag{8}$$

where N_0 and N_1 are the abundance of individuals (or seeds) in the initial and subsequent stage, respectively, in specified areas (not necessarly the same). The function p describes probabilities of moving from the initial to the subsequent stage (or per capita fecundity). $Z_1, Z_2, .., Z_m$ are additional covariates that can include the densities of conspecifics in the various stages, corresponding densities of heterospecifics and environmental factors. CNDD is detected if p is a decreasing function of the density of conspecifics (N_0 or any Z representing conspecific densities). The functional form of p depends on the precise nature of the interaction (Pacala 1986; Freckleton & Lewis 2006), which is usually unknown so that phenomenological models are used. For example, one of the simplest and widely used is the power law model (e.g. Wright *et al.* 2005; Kellner & Hubbell 2018):

$$N_1 = aN_0^b, \tag{9}$$

where a is the transition probability when $N_0 = 1$ and b is a parameter that indicates CNDD when $b < 1$. A commonly fitted version of this model is the offset-power model (Harms *et al.,* 2000), which uses ordinary linear least squares (OLS) on log-transformed variables, after adding one to handle the zeros:

$$\log(N_1 + 1) = log(a) + blog(N_0 + 1), \tag{10}$$

We immediately noticed that eqns 9 and 10 are not equivalent. Log transformation and OLS, are known to perform poorly on count data and should be replaced by methods based on likelihood maximisation (O'Hara & Kotze 2010), such as generalised linear models (GLMs). In a GLM, the response variable is assumed to be generated by a particular distribution in the exponential family. The mean response depends on the predictors through a link function g as follows:

$$E(p) = g^{-1}(\beta_0 + \beta_1 N_0 + \beta_2 Z_1 + \ldots + \beta_{m+1} Z_m). \tag{11}$$

GLMs commonly used for the analyses of CNDD are listed in Table 1. For example, logistic regressions are appropriate to model the survival probability of seedlings between census intervals. Logistic regressions assume a binomial distribution and a logit link function (other link functions can also be used) :

$$log\left(\frac{p}{1-p}\right) = \beta_0 + \beta_1 N_0 + \beta_2 Z_1 + \ldots + \beta_{m+1} Z_m. \tag{12}$$

Ecological count data are often overdispersed, that is, they exhibit greater variation than predicted by the model, which can result in erroneous model selection and too liberal hypothesis testing (Richards 2008). In order to take into account such variation, it is possible to use compound distributions, such as the beta-binomial for binary count data (e.g. survival) and the negative binomial for Poisson count data (Wright *et al.* 2005; Richards 2008; Gripenberg *et al.* 2014). For example, a log-linear model and negative binomial distribution has been used to fit adult reproductive ratio as:

$$log(p) = \beta_0 + \beta_1 N_0 + \beta_2 Z_1 + \ldots + \beta_{m+1} Z_m. \tag{13}$$

It is easy to show that, excluding the Z covariates, the log-linear model is equivalent to the Ricker population model (Brännström & Sumpter 2005).

$$N_1 = N_0 e^{\beta_0 + \beta_1 N_0}. \tag{14}$$

GLMs are fitted using maximum likelihood (Table S1).

A more sophisticated approach assumes that replicate observations of the predictor, N_0, are available, $N_0^{'}$, and that N_0 can be treated as a latent variable (Muff *et al.* 2015). The parameters are fitted by coupling to the regression model, eqn 11, two additional models, named the error and exposure (predictor) models (e.g. Bagchi *et al.,* 2014):

$$N_0^{'} x007C; N_0 \sim NB(N_0, k), \tag{15a}$$
$$N_0 \sim lognorm(\mu, \sigma^2), \tag{15b}$$

where k is an overdispersion parameter in the negative binomial distribution (NB). The parameters are then fitted using approximate Bayesian inference for latent variables (Rue *et al.* 2009). All the above methods can be extended to include random effects as species, treatments, locations and periods. For examples, generalised linear mixed models (GLMMs) are formulated as:

$$E(p_{ij}) = g^{-1}(\beta_0 + \beta_1 N_{0,i} + \beta_2 Z_{1,i} \ldots + \beta_1 Z_{m,i} + \phi_i + \phi_j), \tag{16}$$

where ϕ are normally distributed random intercepts for species i and location j. Another widely used class of models are hierarchical models (Gelman 2006). In these models the parameters can vary at more than one level as follows:

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$$E(p_i) = g^{-1}(\beta_{0i} + \beta_{1i}N_{0,i} + \beta_{2i}Z_{1,i} \ldots + \beta_{1i}Z_{m,i}), \quad (17a)$$
$$\beta_{ki} = \gamma_{k0} + \gamma_{k1}W_{1i} + \gamma_{k2}W_{2i} + \ldots + \gamma_{kn}W_{ni}, \quad (17b)$$

where W are n species traits or other species-level properties, e.g. relative abundance (e.g. Comita *et al.* 2010; Lebrija-Trejos *et al.* 2016). These models can also include other random effects. The parameters can be estimated with the Bayesian or likelihood method. A List of GLMs used in the detection of CNDD is presented in Table 1.

H2000, C2010 and L2017 respectively. These examples are provided as accessible cartoons, and are explicitly not intended to substitute for the detailed modelling and analysis that forms the bulk of this paper.

H2000 analysed seed-to-seedling transitions using census plots that were spatially distributed in a tropical forest in Central Panama. To detect CNDD, they regressed the seedling abundances from a set of seedling census plots against the seed abundances from a different set of collocated seed-census plots. Similarly, the setup in Fig. 2a has paired seed-census (white squares) and seedling-census (red squares) 1 m^2 plots. Seeds were stochastically dispersed from randomly located parent trees using a fat-tailed dispersal kernel (the colors in Fig. 2a depict the intensity of the seed rain). Seeds in the seedling-census plots were then converted into seedlings with stochastic coin tosses (probability 0.1). Thus, there was absolutely no CNDD in the simulation.

A regression of seedling numbers from the seedling plots against the seed numbers from the collocated seed traps produces a curvilinear relationship (red line in Fig. 2b) that falsely implies the presence of CNDD (the black line is the true relationship). The concave-down red curve falsely implies CNDD, because it indicates that the probability of surviving from seed to seedling falls as seed abundance increases. This is classical regression dilution, exactly analogous to the stock–recruitment problem. We get the red curve rather than the black line, because of error in the predictor. The number of seeds counted in a seed census plot is an error prone proxy (EPP) for the number of seeds that actually fell into the paired seedling plot.

The main result in H2000, is that all species showed statistically significant CNDD. This led ecologists to believe that CNDD is nearly ubiquitous among tropical plant species (Silvertown & Charlesworth 2001; Terborgh 2012), causing the paper to be cited more than 900 times and paving the way to a long series of similar studies (e.g. Hille Ris Lambers *et al.* 2002; Bagchi *et al.* 2014; Umana *et al.* 2016; Krishnadas *et al.* 2018).

The recent study by L2017 (LaManna, 2016, 2017b) relied on a single mapped census for each of many plots. They imposed a spatial grid on each plot, assumed a size threshold to separate 'saplings' from 'adults', and regressed the saplings in each grid square against the adults in the same square. The idea was to determine if and how the number of saplings produced per adult decreases with local abundances of conspecifics and heterospecifics.

The simpler but analogous simulation in Fig. 2c shows adults (blue dots) that stochastically produced an average of three juveniles (red dots), which then randomly dispersed according to a fat-tailed dispersal kernel. As before, there is no CNDD in this simulation, simply because sapling production by the adults is unaffected by abundance. However, the curvilinear relationship (red line Fig. 2d) produced by a regression of the number of juveniles in 50 m × 50 m quadrats, against the number of adults in the same quadrats falsely implies CNDD, because the number of adults in a quadrat is a highly error prone proxy (EPP) of the parents that produced the juveniles in the quadrat.

This example may seem analogous to the one in Fig. 2a and b, but it is actually much more problematic. In the previous example, the count in a seed census plot is an EPP, but one knows what it is a proxy for: the actual number of seeds in the paired seedling census plot before germination. This means that one should be able to fix the problem with replicate seed traps that would allow one to measuring the proxy error and compensate for it in the statistical analysis (Muff *et al.* 2015 and see examples below). However, there is no additional census of quadrats that would quantify the conspecific proxy error that biases the detection of CNDD in the

Table 1 List of generalised linear models (GLMs) used in the detection of CNDD and demographic variables and applications for which each GLM has been used. A GLM is expressed as $E(p) = g^{-1}(\eta)$, where $p = N_1/N_0$ and $\eta = \beta_0 + \beta_1 N_0 + \beta_2 Z_1 + \ldots + \beta_{m+1}Z_m$ is a linear function of the model predictors. The estimation of the parameters can be biased if there are errors in the predictors (N_0 and/or Z)

Model	Distributions (exponential family)	Link function $g(p) = \eta$	Mean function $E(p) = g^{-1}(\eta)$	Applications
Linear	Normal	p	η	Relative and absolute growth
Logistic	BinomialBeta-Binomial	$\log(p/(1-p))$	$(1+e^{-\eta})^{-1}$	Seedling survival, seed germination
Log-linear(Ricker model)	Poisson, Binomial,Negative Binomial	$\log(p)$	e^{η}	Seedling recruitment, seed germination, seedling survival
Complementary log-log	BinomialBeta-Binomial	$\log(-\log(1-p))$	$1-e^{-\exp(\eta)}$	Seedling survival, seed germination
Power-law†	PoissonNegative Binomial	$\log(p)$	$e^{\tilde{\eta}}$	Sapling recruitment, seed germination, adult recruitment

*The power law model is made linear by log-transformation: $\tilde{\eta} = \beta_0 + \beta_1 \log(N_0) + \beta_2 \log(Z_1) + \ldots$

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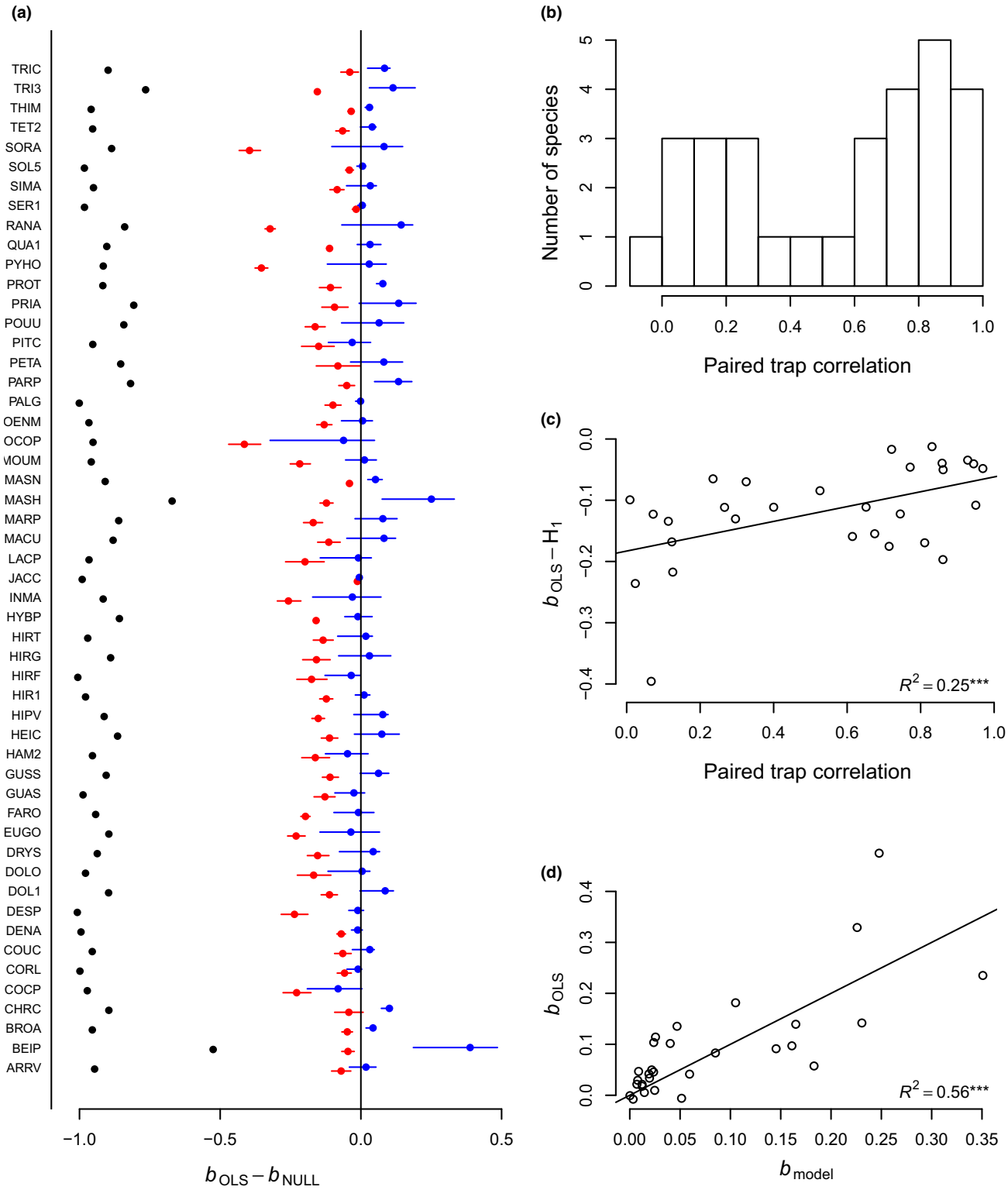

Figure 3 (a) Original estimates of the slope b in H2000 of the model: $log(N_{seedling} + 1) = a + blog(N_{seed} + 1)$, minus the null hypotheses H_0 (●): $b_{OLS} = 1$; H_1 (red, ●): b_{OLS} is obtained assuming constant species-specific germination probability (constant with location), but without considering collocation error; H_2 (blue, ●): b_{OLS} is obtained assuming constant species-specific germination probability and accounting for collocation error (Appendix A in supplementary material). Horizontal bars represent 95% confidence intervals, negative deviations indicate stronger CNDD. b) Histogram of the observed spatial correlation among paired traps. c) Relationship between the spatial correlations among paired traps and the deviance from null hypothesis H_1. d) Relationship between the original estimates of the slope parameter b_{OLS} and an approximate analytical model which assumes no CNDD and accounts for colocation error, eqn 18. 1:1 shown for reference.

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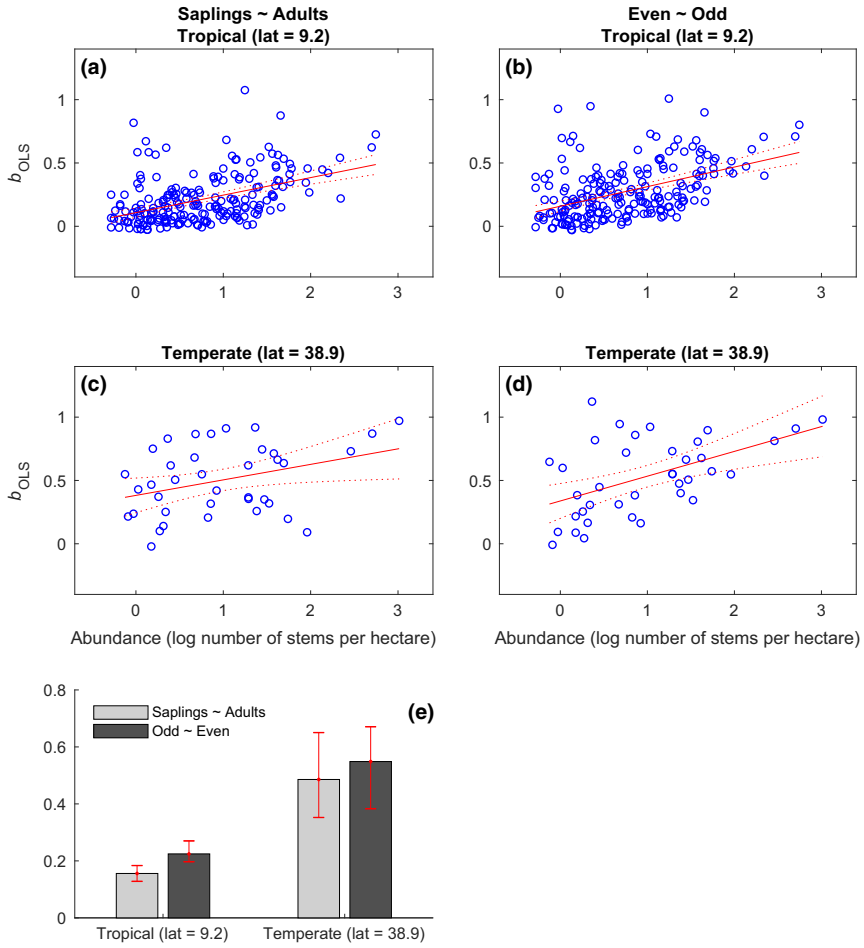

Figure 4 The species-specific quantification of CNDD between individuals with odd and even tag number in 20x20 quadrats (b and d) resembles CNDD between juveniles and adults (a and c) and varies in the same way with a species' abundance. Regression lines and 95% confidence bounds are shown for reference in red. b_{OLS} is the OLS estimator of the slope of the model $log(N_{sapling}+1) = a + blog(N_{adult}+1)$, where $N_{sapling}$ and N_{adult} are respectively the numbers of small and large individuals in the same grid square. (e) The median slope of the OLS estimator is lower for a high-diversity tropical forest compared to a low-diversity temperate forest. Bootstrap 95% confidence intervals are shown as red error bars. The area of the tropical plot is 50 ha and the analysis includes 208 species. The area of the temperate plot is 16 ha and the analysis includes 40 species. Species with less than 0.5 individuals per hectare were excluded. See Appendix B in supplementary material for further details on the analysis.

sapling–adult regressions. Instead, one would need the mechanistic understanding provided by genetic analyses that identify each sapling's mother and consider that some of the mothers might be dead at the time of the inventory. Without a way to eliminate or measure conspecific proxy error, there is no way to design a reliable statistical test to detect CNDD from a single census.

This problem is just one of several related problems in the L2017 study that are examined below in the body of the paper. Other problems have been pointed out by published critiques of L2017 (see Damgaard & Weiner 2017b; Chisholm & Fung 2018; Hülsmann & Harting 2018).

Our third example, concerns the quantification rather than the detection of CNDD. C2010 studied the survivorship of rainforest tree seedlings with repeated censuses of permanently tagged individuals. They used logistic regressions to fit the probability of seedling survival against distance-weighted measures of the local abundances of conspecific and heterospecific neighbors. They reported the amazing result that CNDD increases as a species' abundance decreases, which is commonly taken as evidence that rare species are rare because they suffer strong CNDD. The simpler but analogous simulation in Fig. 2e and f, shows how a relationship like the one in C2010 could also have been a false signal, caused by a seemingly benign predictor error. In this case, the false pattern is caused by systematic errors in the measure of local abundance, but the general point is that EPP's can do more than imply CNDD where none exists. They can also create false patterns.

The simulation behind Fig. 2e and f uses the actual spatial coordinates and diameters of all saplings and adults of 68 species on the 50 hectare plot in Barro Colorado Island Panama (Condit *et al.* 2019). We assumed that a sapling's probability of survival, p, decreases as a logistic function:

$$p = \left(1 + e^{-a-bZ}\right)^{-1}.$$

for all species, where b governs the strength of CNDD (-0.5 in this example), a sets the survivorship with no conspecifics in the neighbourhood (set to 1.4 in this example),

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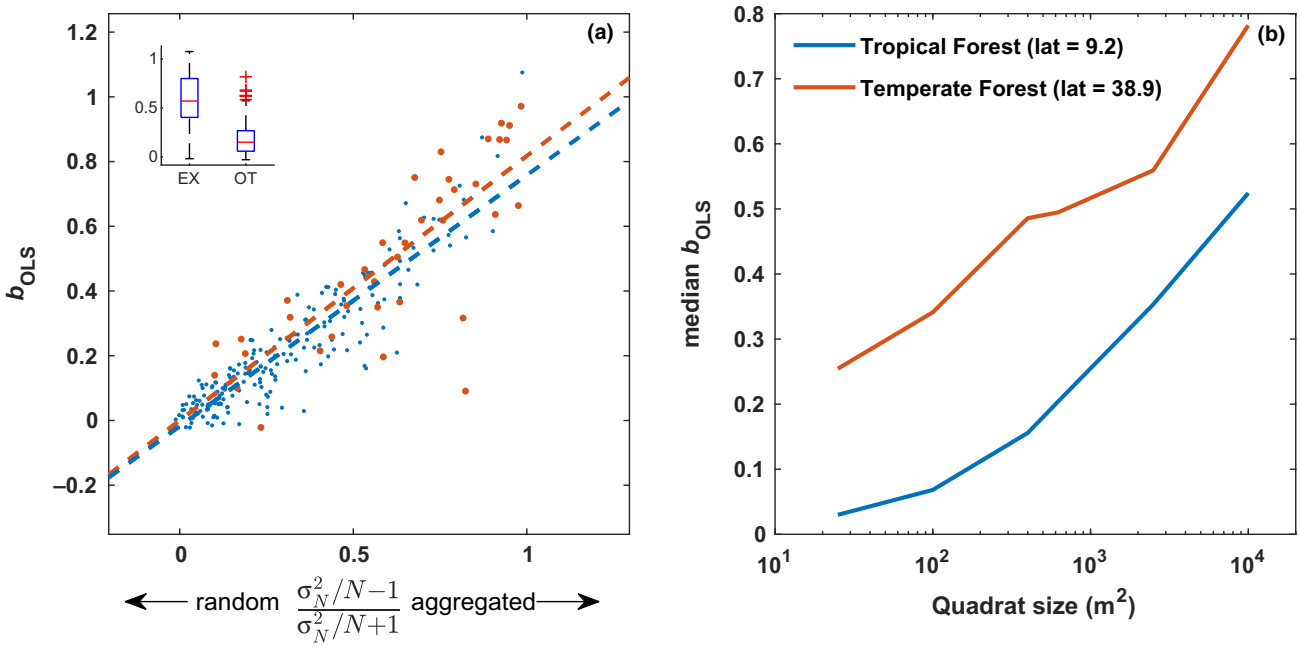

Figure 5 (a) The aggregation of individuals explains interspecific variation in the fitted slope parameter b_{OLS} for the relationship between juveniles and adults ($R^2 = 0.84$). $\bar{N}$ and σ_N^2 are the mean and variance of individuals across quadrats. Linear regression lines (dashed) are shown for reference. In the inset, the boxplot of b_{OLS} for species with explosive dispersal (EX) and species with other dispersal syndrome (OT) for BCI, showing that explosively dispersed species have statistically higher mean (one-way ANOVA, $P < 1e{-}7$). (b) The median b_{OLS} across species decreases for small quadrat size, suggesting that CNDD is stronger for small quadrat size. Panel A is for 20 m by 20 m quadrat size.

and Z is the sum of the crown areas of conspecifics within 5 m of each sapling. Note that CNDD, as measured by b, is equally strong for all species. We assigned each sapling a crown area using the mean from an empirical allometric equation with diameter and exponent 1.3 (Fig. 2e, Cano *et al.* 2019), times a lognormal random number estimated from the regression's residuals (Fig 2e, insert).

As the true Z is unknown in reality, we included predictor error in the logistic regressions by calculating Z using the basal areas of neighbors, as done for example in C2010 and others, which is equivalent to assuming an allometry with exponent 2 and no residual variation. We then estimated values of a and b for each species with logistic regressions using the survival probability computed with the true Z. Because the variance of a sum of independent random variables is the sum of the variances, and because each Z is a sum over conspecific neighbours, the predictor error is larger for abundant species, which tend to have many conspecific neighbours, than for rare species, which tend to have few neighbours (Fig. 2g). This, in turn, dilutes regressions of common species more than rare ones, and causes the analysis to produce a false inverse relationship between the strength of CNDD and abundance (Fig. 2f), like the trend reported in C2010.

The simulations producing Fig. 2f contain both good news and bad news. The good news is that studies that estimate vital rates by following individuals through time are not biased to detect CNDD where there is none, unlike H2000 or L2017. This is because longitudinal data allows the regression of observed individual performance (i.e. survival, growth, or fecundity), against local densities, in which the null hypothesis of no density dependence is that the slopes should be zero. Errors in the predictors flatten estimated slopes, and thus bias the detection of CNDD in the direction of a type 2 error whenever the null hypothesis implies zero slope. The implication is that C2010's analysis is conservative when detecting NDD.

The corresponding bad news is that regressions with longitudinal data are still biased by errors in the predictors, and these biases may produce exciting but false patterns, like the false relationship between the strength of CNDD and abundance in Fig. 2f, produced only because the regressions used basal area as a proxy for actual crown area. Although one could compensate for predictor errors by measuring them and then including them in the statistical analysis, this presupposes that they are understood well enough to be measured. Is basal area in C2010 a proxy for fine root mass, foliar mass or something else? Is the distance weighting in C2010 appropriate for competition for light, water or nutrients, or for the risk of infection by pathogens or herbivores infesting neighbours? How much error is caused by assuming that the distance weighting is the same for all focal and neighbouring species? How much error is built into estimates of the local abundance of heterospecifics by the practice of summing the basal areas of all heterospecific species? Note that summing all heterospecific neighbors generally means that the local abundances of heterospecifics will greatly exceed the local abundance of conspecific neighbours.

In what follows, we perform new analyses of three data sets that extend H2000, L2017 and C2010, and in each section provide new formulae for the biases caused by the use of EPPs. These sections assume some mathematical knowledge, but the most technical material is relegated to Appendices. We then describe a series of modelling studies aimed at

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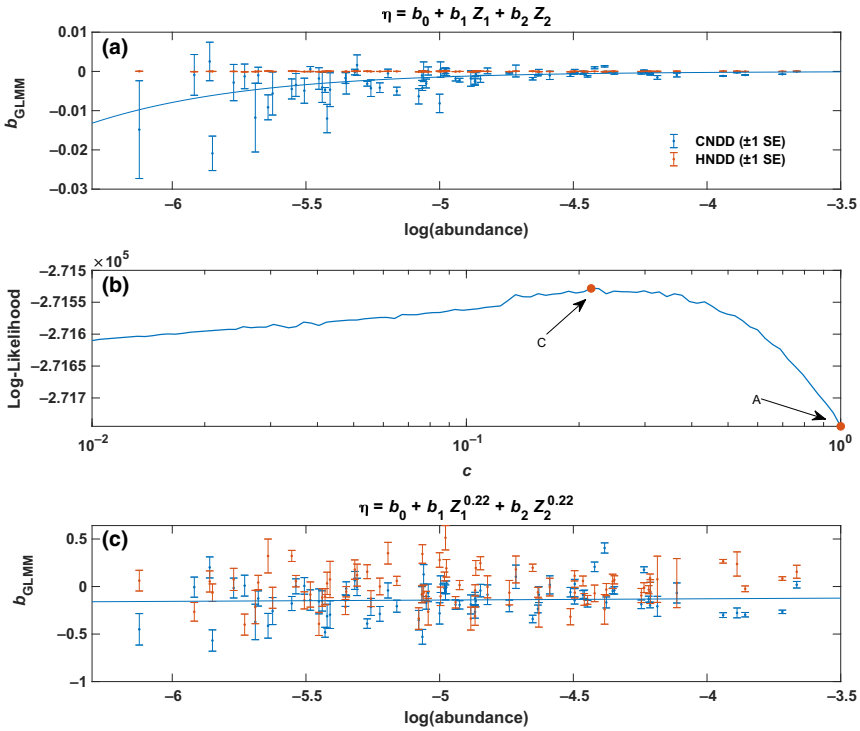

Figure 6 The density dependence of sapling (individuals with diameter at breast height $DBH \leq 10$ cm) survival evaluated for eight censuses (1982–2015) of the BCI 50 ha plot. The analysis includes 76 species with $DBH_{max} > 20$ cm that average more than 200 individuals in the 50 ha plot over the eight censuses. CNDD and HNDD were estimated for each species using GLMMs with a logistic regression: $\log\left(\frac{p_i}{1-p_i}\right) = b_0 + b_1 Z_{1i}^c + b_2 Z_{2i}^c + \phi_i$, where p_i is the individual survival probability in the ith census interval, Z_1 and Z_2 are distance-weighted sums of basal area of conspecifics and heterospecifics respectively, and ϕ_i a random effect for census interval. The exponent c regulates the functional shape of the local interactions and was varied between 0.01 and 1. Individuals in a 10 m buffer zone were excluded to minimise edge effects. (a) Estimated parameters b_1 and b_2 as function of plot level species basal area for $c = 1$. (b) Log-likelihood as a function of the exponent c. The log-likelihood is the sum of the log-likelihoods of each individual species. The red dots indicate the maximum value of the log-likelihood (labelled C) and the log-likelihood for $c = 1$ (labelled A), where the labels A and C correspond to panels in this figure. (c) Same as A, but for $c = 0.22$, the value corresponding to the maximum log-likelihood. Trend lines for CNDD are also shown for reference (blue lines).

quantifying impacts of the most likely sources of predictor error in the above studies and the many similar studies.

Our analysis shows that predictor errors in these studies are likely to be large enough to account for most, but not all, of their results. The problems we study are generic to the vast majority of studies reviewed by Adler *et al.* (2018), and this brings into question the emerging paradigm that CNDD is generally stronger than HNDD in plant communities and makes it unwise to assume strong CNDD when designing conservation measures. Although sobering, these results also point to a path forward, which we offer in the Discussion.

RESULTS

Seedling–seed relationships

H2000 quantified CNDD by estimating the slope parameter b_{OLS} of the model: $\log(seedlings + 1) = a + b \log(seeds + 1)$, hereafter referred as offset-power model, which is an inexact linearisation of the power law model (inexact because 1 is added to the abundances to avoid having to take the log of zero). Here, we reanalyse the same seed-to-seedling transition data from Barro Colorado Island in Central Panama (BCI), after first obtaining estimates of the errors between traps and plots from new field data (Appendix A in supplementary material). We show that the estimates H2000 were biased and that the primary result that CNDD is pervasive among tropical species disappears when a more conservative test is applied.

There were two sources of error in the paper (Appendix A in supplementary material), bias caused by adding one to the abundances in the offset-power model (Hille Ris Lambers *et al.* 2002), and bias caused by a colocation error – using the abundances of seeds in one place (a seed trap) as a proxy for the seeds that dispersed into another (a nearby seedling census plot as in Fig. 2a). In order to test the significance of the estimated parameter b_{OLS}, we used three null models, H_0, H_1 and H_2. Null model H_0 contains both sources of error (as does the original paper); H_1 removes the error caused by adding one to the abundances in the offset-power model and H_2 removes both sources of error. All null models assume that $b = 1$, which is the expectation under no CNDD, whereas a negative deviation ($b < 1$) implies CNDD.

The deviations from the null model H_1 are much closer to zero than the deviations from the null model H_0 (compare black and red dots in Fig. 3a). However, deviations from the null model H_1 were still significantly negative, which implies CNDD, for all species. The null model H_2 takes into account

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the collocation error by including the observed spatial correlation between two paired seed traps in randomisations (Appendix A in supplementary material). These paired seed-trap data, which were not available at the time of H2000, revealed large interspecific variation in spatial correlation among paired traps. The correlation is sometimes very low, implying that seeds observed in traps are poor proxies for unobserved seeds in collocated seedling plots (Fig. 3b). As a consequence, the deviations from H_1 reduced with increasing spatial correlation ρ ($R^2 = 0.29$), indicating that bias in CNDD increased as correlation among paired traps decreased (Fig. 3c). Consistent with this test, the original estimates in H2000 were well described by an approximate statistical model that accounts for the degree of correlation, and derived under the assumption of no CNDD (Fig. 2d, $R^2 = 0.57$, see supplementary material Appendix A for the derivation of the approximate solution). Thus, the apparent CNDD obtained under H_1 is an artifact caused by colocation error. With both sources of error accounted for, there is no species left with statistically significant CNDD according to H_2.

The following equation provides a simple solution for b_{OLS} in absence of CNDD and when seeds and seedlings are censused in separate plots (Appendix A in supplementary material):

$$b_{\mathrm{OLS}} \cong \frac{\bar{N}_{seed} + 1}{\bar{N}_{seed} + \bar{p}^{-1}} \rho, \tag{18}$$

where $\overline{p} = \overline{N}_{seedling}/\overline{N}_{seed}$, is the mean germination probability, $\overline{N}_{seed}$ the mean seed density and ρ the correlation between the seeds observed in the seed traps and the (usually unknown) true number of seeds that dispersed into each seedling census plot. Larger collocation errors are associated with smaller values of ρ and larger underestimation of b_{OLS} (Fig. 3c).

Adult–juvenile relationships

The analysis in this section is similar to the one above, but targets CNDD affecting the numbers of juveniles produced by adults. Like L2017 (LaManna *et al.* 2016, 2017a), we use stem maps of forest plots to study the spatial relationship between juvenile and adult trees. There are many sources of error inherent in using individuals larger than a threshold size in a grid square as a proxy for the parents of those smaller than the threshold in the same square. Mothers of the plants in the small-sized group could be located outside the square, could have died, or could even be other individuals beneath the size threshold in the same square. In this section, we show that the surprising results in L2017 are obtained no matter how we divide the individuals in each square into two classes – for example, assigning individuals with odd census tags as juveniles and even census tags as adults. We show that the patterns reported in L2017 are simply caused by patterns of species aggregation.

In this analysis, the number of juveniles (defined as individuals below an arbitrary size threshold) in a specified area (usually 10 × 10 or 20 × 20 quadrats) are regressed against the number of adults (defined as individuals above the same threshold) using the offset-power and other models (as in L2017). Figure 4a and c show that OLS estimates of the slope, b_{OLS}, are smaller than one for almost all of the species analysed, as in L2017, which appears to imply CNDD. The OLS estimates of the slope of the offset-power model also reproduce two other patterns in L2017. Slopes were inversely correlated with the abundance of the species (strongest CNDD for rare species) and the median slope was significantly lower in the tropical forest plot than in the temperate plot (Fig. 4e), which indicates stronger CNDD in the tropics. The same results was obtained if the median was calculated only for rare species (species with basal area < 0.1 m^2 ha^{-1}), that is, when results are compared on a common abundance axis.

When we applied the same method to the same forest data, but instead of splitting trees into the biologically meaningful but error-prone categories, we divided trees into biologically meaningless categories: for example even tag versus odd census tag, the same patterns were obtained (Fig. 4b and d). As before, the spurious CNDD decreased with abundance and the median slope was lower for the tropical than for the temperate plot. Similar results were obtained by splitting the individuals with other meaningless criteria and by fitting different models, such as the Ricker model (Fig. 3 and 4).

This result that any arbitrary grouping of the individuals (a.k.a. random labelling) produced the same patterns, challenges the ecological interpretation of the mechanisms underlying such patterns. In particular, assuming that the slope is one as a null hypothesis for the absence of CNDD is not totally justified. For instance, using random labelling, we derived an expression for b_{OLS} which is independent of how individuals are classified, and it depends only on how individuals are distributed in space (Appendix B in supplementary material):

$$b_{\mathrm{OLS}} \cong \frac{\sigma_N^2/\bar{N} - 1}{\sigma_N^2/\bar{N} + 1}, \tag{19}$$

where σ_N^2 and $\bar{N}$ are the variance and the mean number of individuals per quadrat. Equation 19 shows that any process that affects the spatial variability among individuals can generate $b_{\mathrm{OLS}} < 1$. Note that for a complete spatially random process (e.g. Poisson), $\sigma_N^2/\bar{N} = 1$ and $b_{\mathrm{OLS}} = 0$.

Importantly, $\sigma_N^2/\bar{N}$ is not necessarily constant with abundance, and lower values of b_{OLS} for rare or common species are expected if $\sigma_N^2/\bar{N}$ increases or decreases with abundance. Taking $\sigma_N^2/\bar{N}$ as a measure of species aggregation, eqn 19 indicates that less aggregated species, according to this metric, would indeed result in lower b_{OLS}. Therefore, everything else being equal, a relationship is expected between apparent CNDD and spatial aggregation. Many biological processes affect spatial aggregation, including dispersal strategies and habitat heterogeneity as well as CNDD. For this reason, the analysis of L2017 is an inappropriate test for CNDD.

The trends in Fig. 4 are expected according to eqn 19, because $\sigma_N^2/\bar{N}$ increased with abundance in both forests (Spearman's $r = 0.50$, $P < 10^{-9}$), and thus any arbitrary grouping of the data will inevitably find stronger CNDD in rare species. Equation 19 explains a large proportion of the variation in b_{OLS} for both sites (Fig. 5, 2a $R^2 = 0.84$). Importantly, the relation between b_{OLS} and $\frac{\sigma_N^2/\bar{N}-1}{\sigma_N^2/\bar{N}+1}$ does not differ

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among forest types (Fig. 5a), which indicates that aggregation alone is responsible for variability in CNDD, within and among forests. Dispersal limitation is one of the main factors causing aggregation. For example, some of the shrubs on BCI have limited dispersal strategies (e.g. explosive capsule) and display strong clumping (Clark *et al.* 2017), which might result in higher values of b_{OLS} (apparent weak CNDD). A one-way ANOVA revealed that explosively dispersed species have significantly higher b_{OLS} than species with other dispersal syndromes ($P < 1e{-}7$, insert in Fig. 5a).

Furthermore, b_{OLS} decreased with the size of the quadrat (Fig. 5b). Small quadrats increase the error in quantifying the number of parents, because many offspring will be dispersed outside their mother's quadrat, and consequently aggravate the regression dilution problem. However, a lower value of b_{OLS} indicates stronger apparent CNDD, so the results might be erroneously interpreted as support of the hypothesis that CNDD operates over small distances.

Survival analyses

In this last case, we evaluate NDD by analysing the survival of focal juveniles as a function of local density of conspecifics and heterospecifics similar to C2010, and others (e.g. Johnson *et al.* 2017; Zhu *et al.* 2018) as in Fig. 2e and f, using saplings from periodic censuses of mapped individuals in the same BCI forest. Also, in contrast to the previous two cases, this analysis cannot generate NDD when absent. However, the choice of the functional form of local interactions can bias the estimation of the parameter b, and the bias can be more severe for heterospecific negative density dependence (HNDD) than for CNDD and more severe for abundant species than rare species. This can generate spurious interspecific trends and give the appearance that HNDD is negligible compared to CNDD.

This analysis classifies as saplings individuals with diameter at breast height ≤ 10 cm, which were mapped and censused eight times from 1982 to 2015 in the BCI 50 ha plot. It includes 76 species with at least one individual with a diameter > 20 cm and with at least 200 individuals on average per census. CNDD and HNDD were estimated for each species using logistic regressions as in C2010:

$$log\left(\frac{p_i}{1-p_i}\right) = b_0 + b_1 Z_{1i}^c + b_2 Z_{2i}^c + \phi_i, \tag{20}$$

where p_i is the individual survival probability in the ith census interval, Z_1 and Z_2 are distance-weighted sums of basal areas of conspecifics and heterospecifics respectively, and ϕ_i a random effect for census interval. The exponent c regulates the functional shape of the local interactions; for $c = 1$ local interactions increase linearly with Z, as in the original analysis of C2010, for $c < 1$ the marginal effect decreases as Z increases. The model was fitted for different values of c and the model with maximum likelihood was selected.

For $c = 1$, estimated CNDD was consistently greater than HNDD (by an order of magnitude) and stronger for rare species than for common (Fig. 6a). These results mirror the seedling analysis in C2010, suggesting a dominant role for CNDD across life stages for rare species. However, the likelihood was maximised for $c = 0.22$ (Fig. 6b). 10-fold cross-validation confirmed that the model with $c = 0.22$ outperformed $c = 1$ (Fig. C3). With $c = 0.22$, the trend with abundance disappeared, demonstrating that the trend was highly dependent on the nonlinearity of the density metric used (Fig. 6c). Furthermore, the differences in magnitude between b_1 and b_2 decreased. Note that the exponent c can only account for nonlinearity in the functional shape of the interactions, but not for measurement errors, which might still be responsible for differences between CNDD and HNDD. Furthermore, the optimal c might differ across species and life stages.

The potential problems in this analysis appear to be caused by a different mechanism that the problems in H2000 and L2017 – the use of an inappropriate functional form rather than the presence of random errors in the predictors. However, this distinction is less than sharp. Here, density was quantified by a relatively complex function (sums of distance-weighted basal areas), that is likely to include both systematic and random errors. For example, the case in Fig. 2e–g includes systematic error because the fitted model assumes a different allometric exponent in the density metric than the exponent used to generate the data (Fig. 2e). This example also includes random errors, because random residuals from the allometric regression were added to the 'actual' crown areas when generating the data, whereas the fitted model simply summed distance-weighted basal areas. Both systematic and random errors in density metrics can create false patterns involving the strength of density dependence (Appendix C in supplementary material). If C2010 had used a saturating density metric with properties similar to Z_1^c instead of Z_1, then the analysis might not have detected the reported relationship between abundance and the strength of CNDD or reported the result that CNDD is generally much stronger than HNDD. The point here is that the distinction between a systematic error in the functional form in a regression and systematic and random errors in the density measure used for predictors is blurred once the density metric becomes complex.

DISCUSSION

False or biased detection of CNDD has been recognised as a major problem in ecological population studies with serious practical implications for species conservation practices (Walters & Ludwig 1981; Shenk *et al.* 1998; Mason 2002; Freckleton *et al.* 2006). Accurate estimates of CNDD rely on precise model formulations and error-free predictors, conditions that have never been carefully assessed in plant studies (Freckleton & Lewis 2006), despite the large body of work conducted on this subject in plant community ecology (Adler *et al.* 2018). As this body of work claims to have empirically demonstrated a basic tenet of modern coexistence theory - that 'intraspecific competition is stronger than interspecific competition', which paves the road to applications in ecosystem and reserve management (Cordonnier *et al.* 2018) – the time is ripe for a critical review.

The estimation of CNDD in plants from observational data is challenging, as it requires evaluating individual level performance as a function of conspecific interactions in the

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neighborhood of focal individuals. So, quantifying CNDD ideally requires a metric of conspecific density measured at an appropriate spatial scale which depends on the precise nature of the interaction (Gripenberg *et al.* 2014; Damgaard & Weiner 2017a). In the absence of such data, proxies can be used, but these are generally error-prone, requiring special statistical attention.

Error-prone proxies (EPPs)

In ecological studies, errors in the measured variables (e.g. miscounting, species misidentification and errors in digitalisation) are ubiquitous and often unavoidable (Chen *et al.* 2013; Morrison 2016; Groom & Whild 2017). Measurement error in the predictors is what caused the false CNDD in stock-recruitment relationships. However, in ecological field studies, it is more common to use a measured error-prone proxy in the place of the quantity that we are actually interested in. For example, because dispersal is highly stochastic in both time and space, the number of seeds observed at two different locations, no matter how close, will always differ. Similarly, seed traps elevated from the ground cannot account for secondary dispersal by scatter-hoarding vertebrates (Jansen *et al.* 2012), which can contribute to the mismatch between observed and true numbers of seeds. This type of error is common to all situations where the independent and response variables cannot be measured in the exact same location or time.

Smart experimental designs and robust statistical methods can minimise the bias caused by such errors. In practice, this requires repeated measurements which enable inclusion of error uncertainty when fitting models (Carroll *et al.* 2006; Muff *et al.* 2015). For example, Bagchi *et al.* (2014) used a multi-trap design to quantify CNDD affecting the seed-to-seedling transition. The results in Bagchi *et al.* (2014) dramatically differ from the results in H2000 and subsequent analyses (Wright *et al.* 2005; Fricke & Wright 2017), as the former detected CNDD in few species only, whereas the latter reported CNDD in all the species. This can be explained by the multi-trap design's ability to correct for bias, which is consistent with our re-analysis of the data set used by H2000 informed by a newly available multi-trap estimate of collocation error (Fig. 2a) and by numerical simulations (Appendix A in supplementary material). However, our simulations demonstrated also that the bias is not eliminated completely with multi-trap designs. In the study of Bagchi *et al.* (2014), strong CNDD was observed for *Terminalia amazonia*, a species with a high density of small wind-dispersed seeds, attributes that our simulations indicate cause overestimation of CNDD (Fig. 7c, Appendix A in supplementary material). Bagchi *et al.* (2014) also showed that applying pesticides increased germination probability and reduced CNDD. According to our simulations, increasing germination probability should increase the bias (Fig. 7f), thus the result in Bagchi might indicate a real biological effect. Unfortunately the decrease in CNDD with pesticides for *T. amazonia* was not confirmed by a simultaneous manipulation experiment conducted in the same forest with the same treatment (Gripenberg *et al.* 2014).

Often, we lack understanding of the negative interactions among plants that we need to design an EPP with as little error as possible. Although we know a significant amount about shading, we know little for most plant species about root deployment and thus about neighborhood competition for water and nutrients (which is also affected by symbionts). The problem is perhaps most acute for negative effects of natural enemies. For example, it is common to use the number of individuals, or basal area of conspecific neighboring plants as a proxy for pathogen load and transmission risk (Liang *et al.,* 2016; Forrister *et al.* 2019). However, many of the natural enemies of plants are unknown, as are the distances over which many pathogens are transmitted, and we should expect that transmission risk will generally vary among plant and enemy species and with plant size (Uriarte *et al.* 2010).

EPPs have particularly severe effects in studies that attempt to detect CNDD from the spatial relationship between conspecific juvenile and adult trees (Johnson *et al.* 2012; LaManna *et al.* 2016, 2017b; Bennett *et al.* 2017) because both the metric for the competitive interaction and the number of parents are EPPs. Assuming that in the absence of CNDD the recruitment of juveniles in a quadrat is linearly proportional to the number of large trees is unjustified. First, seed dispersal displaces offspring from parents, and hence the number of juveniles in a quadrat is not expected to be proportional to the number of adults. Second, juveniles can be found in areas where conspecific adults are completely absent, as they require gaps or other environmental conditions to establish, or simply, because parents are dead at the time of the inventory. For example, *Tachigali versicolor*, a common monocarpic tree in the lowland tropical forests of Central America, reproduces only once in its lifetime and dies within a year of flowering (Foster 1977). So, the saplings are never found in proximity of their parents, and even when dispersed close to other conspecific adults, their numbers are unlikely to be proportional to the number of adults. Third, because the reproductive status and age of the individuals is usually not recorded, dividing individuals based on a fixed size threshold introduces errors, such that individuals can be classified as adults even though they are not reproductive and, vice versa. Individuals close to but on opposite sides of the threshold size are characterised as being very different rather than very similar (Altman & Royston 2006). Here, we expect that the number of adults to be a highly EPP which strongly biases the estimation of CNDD (Appendix B in supplementary material). So, as in the case of H2000, CNDDs reported in L2017 are certainly overestimated because the problem of errors in the predictors has not been controlled for appropriately. Therefore, the patterns generated by such analyses should be taken with extreme caution.

Static spatial patterns

The analyses of static data contains another serious drawback. Here, we showed that the ecological interpretation of strength of apparent CNDD with abundance or among forests is questionable as the same relationships are obtained regardless of dividing individuals according to juveniles-adults or ecologically meaningless criteria (parity of tree tag). In point pattern analysis, this problem is explored by performing a random labelling, a type of randomisation to evaluate interactions between two spatial processes (Goreaud & Pélissier 2003;

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Wiegand & Moloney 2004). The random labelling does not hold information about adults and juveniles, but only about the spatial distribution of individuals, as it retains species aggregation properties. Thus, the patterns reported by L2017 are interesting because they reveal biological trends in species aggregation as done in previous studies (Condit *et al.* 2000; Gilbert *et al.* 2010; Detto & Muller-Landau 2016; Umaña *et al.* 2017).

One interpretation for such trends, which is consistent with the interpretation in L2017, is that aggregation is weaker in species which suffer high CNDD. However, interspecific variation in aggregation is a result of multiple processes, including seed dispersal (Seidler & Plotkin 2006; Clark *et al.* 2017) and habitat heterogeneity. Dispersal strategies are quite variable between species and among biomes (Clark *et al.* 1999; Muller-Landau *et al.* 2008). Habitat filtering is also a strong driver of species aggregation and varies systematically between temperate and tropical forests (Myers *et al.* 2013). Therefore, any static spatial analysis that does not control explicitly for species-specific and forest-specific dispersal, and habitat heterogeneity (Zhu *et al.* 2010; Yao *et al.* 2016) can erroneously interpret these important factors as CNDD.

These statistical issues are symptomatic of the challenges inherent in inferring dynamic processes from static observational data. Although such inferences are possible, they are usually weak and require an extra degree of caution relative to inferences from dynamic experimental studies (Damgaard & Weiner 2017a) and appropriate statistical techniques (e.g. Bagchi *et al.* 2011; Yao *et al.* 2016). A major problem is that different processes and different combinations of processes can generate similar spatial patterns, which makes it difficult to distinguish between them (Detto & Muller-Landau 2013; Damgaard & Weiner 2017b; Hülsmann & Harting 2018).

Characterisation of CNDD

Analyses conducted with longitudinal data, such as survival and growth rates are, in this regard, more robust than analyses of static data. However, they still rely on arbitrary density metrics and proxies of competitive interactions. Although these analyses do not generate false detection of CNDD, they bias the quantification of both CNDD and HNDD by introducing systematic errors that produce spurious interspecific patterns. For example, logistic regressions, a common tool for survival analysis, are quite sensitive to measuring errors and nonlinearity (Stefansky & Carroll 1985; Heid *et al.* 2002; Bergtold *et al.* 2018). A logistic regression assumes that survival approaches zero for high values of local density. This assumption is not biologically consistent with transmission of infectious diseases where mortality reaches an asymptote equal to the sum of base mortality and lethality when all the individuals are infected (Anderson & May 1982). Thus, fitting a logistic regression when survival flattens at high densities produces a bias that causes CNDD to appear to weaken as species become increasingly abundant.

Unfortunately, in complex fitting algorithms such as GLMMs and hierarchical Bayesian models, the direction and magnitude of bias caused by EPP's are often not easy to predict, and different fitting algorithms can produce different results. For example, Johnson *et al.* (2012) and Zhu *et al.* (2015) produced opposite relationships between CNDD and abundance from the same Forest Inventory and Analysis dataset.

Furthermore, in natural settings, species appear to interact weakly because they are often confined to their realised niches by competition, which makes much of HNDD undetectable (Tuck *et al.* 2018).

Recommendations and Conclusions

Although the results presented in this study seem discouraging, they also offer clear directions for resolving some of the issues in detecting CNDD. In the cases where the density metric is known (e.g. number of seeds), but it is measured with errors, a quantification and characterisation of the errors, for example with repeated measurements, is often sufficient to correct for the bias. A large literature on how to account for independent-variable errors in statistical models already exists and is available in commonly used computational platforms (e.g. Martins *et al.* 2013; Bürkner 2017; Yenni *et al.* 2017).

However, there are many cases where CNDD is inferred from simple proxies of density, because these are readily available from forest inventories, remote sensing products and other counting measurements (Kellner & Hubbell 2018). In all these cases, a mechanistic understanding of the interactions may be required. Distinguishing effects of pathogens from competition for resources (e.g. Forrister *et al.* 2019) is essential to choose the appropriate functional form and spatial scale for the predictors and ultimately to evaluate their uncertainties.

In contrast, analyses of spatial relationships between adults and juveniles contain too many uncertainties and are based on strong assumptions that are often difficult to verify. The information necessary to reliably characterise CNDD includes not only the metrics of density, but also the metrics of fecundity which are specific to each species and to each individual and need to be integrated across multiple cohorts. These studies need to be redesigned, for example using genetic analyses, or abandoned.

Currently, CNDD trends reported in a large number of studies of transitions among stages and static spatial data are not robust because they arise naturally in null models and are biased by errors in predictor variables. This does not imply that CNDD is absent, unimportant, or invariant with abundance or latitude; it simply shows that better experimental design and more robust analyses are needed to properly quantify CNDD. For instance, as we generally expect less error in manipulation studies, errors in predictors could explain why Adler *et al.* (2018) found a larger difference between CNDD and HNDD in observational compared to manipulation studies.

We hope this contribution will help familiarise researchers with measurement error problems and potential solutions, and to stimulate a new generation of studies that ultimately resolve these issues, by focusing on the mechanisms behind negative interactions and with reduced reliance on phenomenological models of density dependence.

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AUTHORSHIP

MD and MV, designed the study and performed statistical analyses, SJW collected data, SWP contributed to the theoretic modelling. MD wrote the first draft of the manuscript and all authors contributed substantially to writing and revisions.

DATA ACCESSIBILITY STATEMENT

All data and codes used for this study can be downloaded at https://forestgeo.si.edu/explore-data, https://oneshare.cdlib.org/stash/dataset/https://doi.org/10.15146/5xcp-0d46 and https://github.com/mdetto/Bias-in-the-detection-of-negative-density-dependence.

Data DOI: BCI 50 ha plot data (8 censuses, 1982-2015) are freely available at https://oneshare.cdlib.org/stash/dataset/doi:10.15146/5xcp-0d46 SERC 16ha plot data can be obtained via ForestGeo Data Portal http://ctfs.si.edu/datarequest/ The BCI paired traps data are freely available at https://doi.org/10.25570/stri/10088/98254. Codes are available at https://github.com/mdetto/Bias-in-the-detection-of-negative-density-dependence

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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