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Incorporating density dependence into the directed-dispersal hypothesis

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Abstract. The directed-dispersal (DrD) hypothesis, one of the main explanations for the adaptive value of seed dispersal, asserts that enhanced (nonrandom) arrival to favorable establishment sites is advantageous for plant fitness. However, as anticipated by the ideal free distribution theory, enhanced seed deposition may impair site suitability by increasing density-dependent mortality, thus negating the advantage postulated by the DrD hypothesis. Although the role of density effects is thoroughly discussed in the seed-dispersal literature, this DrD paradox remains largely overlooked. The paradox, however, may be particularly pronounced in animal-mediated dispersal systems, in which DrD is relatively common, because animals tend to generate local seed aggregations due to their nonrandom movements.

To investigate possible solutions to the DrD paradox, we first introduce a simple analytical model that calculates the optimal DrD level at which seed arrival to favorable establishment sites yields maximal fitness gain in comparison to a null model of random arrival. This model predicts intermediate optimal DrD levels that correspond to various attributes of the plants, the dispersers, and the habitat. We then use a simulation model to explore the temporal dynamics of the invasion process of the DrD strategy in a randomly dispersed population, and the resistance of a DrD population against invasion of other dispersal strategies. This model demonstrates that some properties of the invasion process (e.g., mutant persistence ratio in the population and generations until initial establishment) are facilitated by high DrD levels, and not by intermediate levels as expected from the analytical model.

These results highlight the need to revise the DrD hypothesis to include the countering effects of density-dependent mortality inherently imposed by enhanced arrival of seeds to specific sites. We illustrate how the revised hypothesis can elucidate previous results from empirical studies reporting little or no support for the DrD hypothesis, and we suggest its incorporation in designing empirical studies of plant recruitment and in management practices.

Key words: adaptive models; frugivory; heterogeneous environments; ideal free distribution; nonrandom dispersal; safe sites; seed dispersal; seed survival.

INTRODUCTION

Seed dispersal is one of the key processes determining the spatial structure and the dynamics of plant populations and communities (Howe and Smallwood 1982, Schupp and Fuentes 1995, Nathan and Muller-Landau 2000). The "directed dispersal" (abbreviated DrD to avoid confusion with density dependence) hypothesis has been proposed to explain the adaptive value of dispersal in spatially heterogeneous environments where habitats differ in their suitability for plant establishment (Howe and Smallwood 1982, Wenny 2001, Vander Wall and Longland 2004). According to the broadly accepted definition of the DrD hypothesis, two fundamental independent components—seed arrival and seed-to-adult survival—delineate two necessary conditions for DrD to bear a selective advantage. DrD requires that (1) diaspores (i.e., dispersal units, hereafter simply seeds) preferentially arrive at sites that are (2) particularly favorable for establishment (Howe and Smallwood 1982, Wenny 2001). The spatial scale of favorable establishment sites can vary from a few centimeters of a specific branch diameter of a mistletoe host plant (Reid 1989, Sargent 1995) to a few kilometers in the case of lakes that are particularly suitable for freshwater plants (Husband and Barrett 1998, Figuerola and Green 2005). Nearly all rigorous examples of DrD are cases of animal-mediated dispersal (Wenny 2001), presumably because animal movements are more fine structured than the flow of abiotic dispersal vectors, and animals tend to select habitats in a predictable manner (e.g., Graham 2001; see also Wenny 2001, Aukema and del Rio 2002).

DrD has been considered to be a strong selective force for the evolution of morphological adaptations to facilitate dispersal, as compared with the weaker

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selection suggested by the colonization hypothesis of arrival to randomly available establishment sites (Howe and Smallwood 1982). Although plant–animal coevolutionary dispersal interactions are in general rather diffusive (Bascompte et al. 2006), some DrD examples, such as the interaction between plants with elaiosome-equipped seeds and ants, or between large-seeded fleshy fruited plants and monkeys, suggest strong selection for a specific suite of dispersers (Herrera 1998, Lord et al. 2002, Lord 2004, Vander Wall and Longland 2004, Bascompte et al. 2006). Correspondingly, the concept of "disperser effectiveness" emphasizes cases of DrD in which selection favors the most effective dispersers that maximize plant recruitment by disproportionately depositing seeds in favorable establishment sites (Schupp 1993, Wenny 2001, Calviño-Cancela 2002).

According to its current definition, the DrD hypothesis emphasizes the suitability of the local characteristics of the site at which seeds are deposited irrespective of the deposition pattern. Yet, as postulated by the ideal free-distribution theory, patch quality is determined not only by the quality and the amount of resources it contains, but also by the number of consumers within it (Fretwell and Lucas 1969). Put into the context of plant dispersal, this idea suggests that site suitability for establishment also depends on the seed deposition itself. High deposition rate leads to higher seed densities and thereby may lead to higher mortality of seeds and seedlings. The importance of density-dependent processes such as seed predation, seedling competition and pathogen herbivory (Janzen 1970, Donohue 1997, Nathan and Muller-Landau 2000, Mari et al. 2008) is amplified in heterogeneous environments, where favorable habitats are typically limited in space, or under contagious dispersal, where dispersal vectors generate local seed aggregations (Kwit et al. 2004, 2007). Local seed aggregations are commonly generated by spatiotemporal variation in the activity of the dispersal vectors (Schupp et al. 2002, Clark et al. 2004), especially vertebrates (Schupp and Fuentes 1995, Schupp et al. 2002, Russo and Augspurger 2004, Kwit et al. 2007).

Overall, animal-mediated dispersal increases the spatial variation of post-dispersal seed density, thus generating hotspots of high density-dependent mortality that might negate the advantage of favorable site characteristics (Schupp and Fuentes 1995). This reinforces an underappreciated conflict inherent to the most basic mechanism underlying the DrD hypothesis. On the one hand, DrD selects for traits attracting dispersers that disproportionately deposit seeds in sites of favorable local conditions. On the other hand, enhanced seed deposition can impair site suitability since high density-dependent mortality can make the same sites unfavorable for recruitment. This *DrD paradox* is essentially equivalent to the basic trade-off underlying the ideal free distribution model (Fretwell and Lucas 1969), as mentioned above. The importance of this paradox in the context of seed dispersal has been acknowledged (e.g., Wenny 2000, Kwit et al. 2007) but, to the best of our knowledge, it has not as yet been directly and thoroughly investigated.

We hypothesize that the DrD paradox can shape the evolution of plant dispersal traits. To examine this hypothesis, we define the *DrD level* as the ratio between the proportion of seeds expected to arrive at favorable sites under DrD to the expected proportion under random dispersal (RD). We consider both DrD and RD to be independent of distance from the source plant, acknowledging that localized (distance-declining) dispersal is a much more realistic assumption (Nathan and Muller-Landau 2000). We find this simplification instrumental, and even mandatory, to clearly convey our take-home messages in this first treatment of the DrD paradox. We further explain our reasoning, and discuss the implications of this assumption for the interpretation of the results, in the *Discussion*, below. We predict (1) an intermediate optimal DrD level, which maximizes plant fitness under the two opposing forces of habitat suitability and density-dependent mortality. We also hypothesize that several properties of the plant and of the habitat influence this optimal level. Specifically, we predict that (2) the optimal level of DrD that maximizes offspring survival should be higher when the difference in suitability between habitats increases (i.e., higher establishment probability in the favorable habitat as compared with the unfavorable habitat). This is expected to occur because the favorable habitat, compared with the unfavorable one, is characterized by either a lower density-independent mortality, a lower density-dependent mortality response (to increasing seed density), or both. We also predict that (3) DrD level influences the invasion success of a "mutant DrD" (differing from the RD wild type only in allocating more seeds than randomly expected into the favorable habitat) into a randomly dispersed population and the resistance of a DrD population to invasion of RD and DrD mutants. Optimal DrD level will facilitate colonization of favorable sites and resistance against invasion of RD and other DrD strategies. Finally, because variation in DrD levels around a given mean value may increase the conflict with density-dependent mortality on the one hand, and limit the DrD advantage on the other hand, we predict that (4) variance in DrD levels among mutants will negatively affect the DrD mutant invasion process.

In this paper, we combine two modeling approaches to test these four predictions. First, we present a simple analytical model to assess the adaptive value (expected net fitness gain) of the DrD vs. the RD strategy in relation to several plant and habitat properties: the plant fecundity, the proportion that the favorable habitat occupies in the region, and its suitability for establishment. Second, we use a simulation model to investigate the invasion process of a DrD mutant in a finite population of RD individuals and vice versa. This enables us to explore the temporal dynamics of the

invasion process, and to assess its sensitivity to variation in DrD levels. We focus on the role of a few important components to keep our models tractable (Levin 1992). Altogether, our models highlight the DrD paradox and the critical importance of incorporating density-dependent mortality in any consideration of the DrD hypothesis.

Methods and Results

An analytical model for the adaptive value of directed dispersal

In order to explore whether the trade-off between DrD (directed dispersal) and density-dependent mortality leads to optimal intermediate levels of DrD, and to evaluate the effect of system properties on this optimal DrD level, we compare the expected fitness of DrD to the expected fitness of the null model of random dispersal (RD; Levins 1969). For simplicity, we consider a population of a single plant species in a region composed of two homogeneous habitats that differ in their negative response of the per capita seed-to-adult survival probability to increasing seed density in the habitat. Each individual can employ only one of the two possible dispersal strategies (RD or DrD), yet the number of individuals is not specified since all individuals exhibiting a certain strategy are assumed to be identical. To assess the adaptive value of DrD relative to RD (i.e., the expected net fitness gain, measured as the difference in the expected number of offspring), we subtract the total expected fitness of the RD strategy in both habitats from that of the DrD strategy in both habitats. Thus, the expected per capita net fitness gain of the DrD over the RD strategy, ΔF, is

$$\Delta F = (F_{\mathrm{DrD}_1} + F_{\mathrm{DrD}_2}) - (F_{\mathrm{RD}_1} + F_{\mathrm{RD}_2}) \quad (1)$$

where F_{DrD} and F_{RD} are, respectively, the expected per capita fitness of individuals exhibiting the DrD and RD strategies, and subscripts 1 and 2 denote, throughout this paper, the favorable and unfavorable habitats. Eq. 1 can be solved analytically by incorporating the fraction of seeds dispersed to each habitat by each strategy (resulting in a specific seed density for each habitat) and the distinct habitat response to increasing density, a process determining the seed-to-adult survival probability. To be compatible with the original work of Fretwell and Lucas (1969), we consider first a linear negative effect of seed density on seed survival, that can simply be expressed as

$$\omega = \beta - \alpha \times \delta \quad (2)$$

where ω is the proportion of surviving seeds, δ is seed density in the habitat, and α and β are two distinct properties of the habitat affecting seed survival. The parameter β corresponds to habitat basic suitability (Fretwell and Lucas 1969), when density effects are negligible and thus, following common practice, is hereafter referred to as "density-independent suitability." The parameter α determines the rate by which habitat suitability deteriorates in response to increasing seed density (hereafter "density-dependent suitability"). Eq. 2 is restricted to the biologically relevant parameter range of $0 < \omega < 1$, $0 < \alpha < 1/\delta$ and $0 < \beta < 1$. Note the opposing trends of the parameters, with larger β and smaller α values representing a better habitat. We consider two homogenous habitats that may differ either in α, β, or both, due to various biotic and abiotic factors. Because habitats that are favorable in terms of per capita (seed-to-adult) survival may also be favorable in terms of higher per capita fecundity, our model may conservatively underestimate the difference in suitability between favorable and unfavorable habitats. Nevertheless, we show that the simplifying assumption of habitat-independent fecundity does not alter our conclusions about the importance of density-dependent effects for DrD.

In the two-habitat region, the favorable habitat occupies a relatively small proportion H_1 of the total area (R), while the unfavorable habitat occupies the majority of the region (i.e., $H_2 = 1 - H_1$, $H_2 > 0.5$). This proportion, together with the quantity of seeds dispersed to each habitat, determines seed density in the two habitats. While the proportion of seeds expected to arrive at the favorable habitat under RD is equal to H_1 (Levins 1969), a higher proportion is expected under the DrD. The ratio of the proportion of the seeds arriving under a particular DrD compared with an RD strategy is represented by Ω. For instance, if the favorable habitat covers 10% of the area, nonrandom arrival of 30% of the seeds to this habitat compared with the 10% expected under RD implies a DrD level of $\Omega = 3$. Biologically, Ω is defined for the range $1 \leq \Omega \leq 1/H_1$; that is, between the RD situation ($\Omega_{\min} = 1$) and the highest possible DrD level ($\Omega_{\max} = 1/H_1$). The Ω index is similar in essence to the combination of the Φ and γ indices of Purves and Dushoff (2005), replacing their qualitative approach of patch occupancy with our quantitative approach of patch density. For simplicity, we consider the total habitat area R as one spatial unit, and provide the general model implementing R in Appendix A. This simplification implies calculating seed numbers (n) instead of seed densities (δ). The allocation of seeds by the DrD strategy (n_{DrD}) to the two habitats is thus

$$n_{\mathrm{DrD}_1} = N \times \Omega \times H_1 \quad (3)$$

$$n_{\mathrm{DrD}_2} = N(1 - \Omega \times H_1) \quad (4)$$

where N is the total fecundity of all plants of the two dispersal strategies combined; that is, fecundity is assumed to be constant across dispersal strategies, individuals and habitats. Seed allocation by the RD strategy can be extracted from Eqs. 3 and 4 by setting $\Omega = 1$.

Finally, it can be shown (see Appendix A) that by substituting Eqs. 2, 3, and 4 into Eq.1, the net fitness

gain is a second-order polynomial function of Ω with one vertex point and two solutions (Ω_a, Ω_b). As the coefficient of Ω^2 is negative the function vertex is a maximum, reflecting a maximum possible fitness gain for the optimal DrD level. The value of Ω at the vertex point (Ω^*) is

$$\Omega^* = \frac{(\beta_1 - H_1 \times \beta_1 + 2N \times \alpha_2 - \beta_2 + H_1 \times \beta_2)}{2N(\alpha_1 - H_1 \times \alpha_1 + H_1 \times \alpha_2)}. \quad (5)$$

In addition to Ω^*, the function has two solutions, where DrD levels leads to no fitness gain ($\Delta F = 0$). The first solution $\Omega_a = 1$ represents the biologically meaningless case where the particular DrD is actually RD. The second solution,

$$\Omega_b = \frac{\{(H_1 + 1)(\beta_1 - \beta_2) + N[\alpha_1 - H_1(\alpha_1 - \alpha_2) - 2\alpha_2]\}}{N[H_1(\alpha_1 - \alpha_2) - \alpha_1]} \quad (6)$$

represents the case where the particular DrD level is sufficiently biased toward the favorable habitat so that seed survival probability in the favorable habitat is reduced by the high densities to the level expected in the unfavorable habitat for RD strategy. For higher DrD levels than Ω_b (if applicable, i.e., if $\Omega_b < \Omega_{max}$) the DrD strategy is thus adaptively inferior to the RD strategy. Additionally, seed shortage decreases seed density in the unfavorable habitat and thus minimizes its disadvantage for establishment. Therefore, high Ω values counteract the fitness advantage of DrD over the RD.

Eqs. 5 and 6 suggest that properties of the habitat (H_1, α_1, α_2, β_1, β_2) and the plant (N) determine both the optimal value for Ω (Ω^*) and its maximal value with a nonnegative fitness gain (Ω_b). We emphasize that these properties include habitat-independent plant properties (N) and plant-dependent habitat properties (all others) that are determined by the interaction between the plants and their environment.

The habitats in our model can differ along two axes—their ratio of density-dependent suitability (i.e., α ratio, α_2/α_1) and their ratio of density-independent suitability (i.e., β ratio, β_1/β_2). Note the opposing order of habitats in the two ratios, allowing straightforward (positive) interpretation of higher ratio values representing higher suitability of the favorable habitat. For instance, a larger ratio of density-dependent suitability implies large α_2 and therefore a faster deterioration of the unfavorable habitat in response to increasing density.

While the linear function of habitat density response allows a simple analytical solution to the model, nonlinear functions can also be used to express habitat response. Although more complicated response functions might better represent reality, they are harder to solve analytically. In Appendix B, we demonstrate an analytical solution for the model using a quadratic function, $\omega = \beta - \alpha\delta^2$, reflecting situations in which the negative effect of density-dependent mortality amplifies as density grows. The results of this model are similar to the following results of the linear model, suggesting that the DrD paradox is not an artificial result of the linear function used to describe the habitat response.

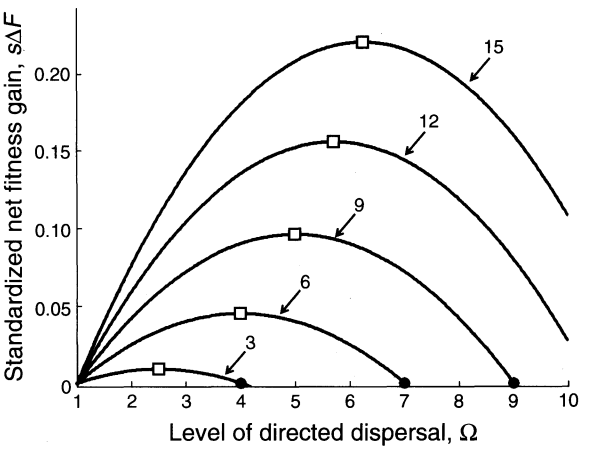

FIG. 1. Results of the analytical model describing the effect of directed-dispersal (DrD) level, Ω, on expected per capita fitness gain of the DrD strategy in comparison with random dispersal ($s\Delta F$; standardized by fecundity). Each line represents a model solution using a different ratio of density-dependent suitability between the two simulated habitats (i.e., α_2/α_1 ratio; values noted by arrows). A higher ratio represents a stronger difference between habitats, and hence higher fitness gain for a given Ω, along with a wider range of positive fitness-gain values. Points of maximum fitness gain, Ω^*, and zero fitness gain (Ω_a, Ω_b) are marked (open squares and solid circles, respectively). Other parameters were set as follows: N (total fecundity of all plants in the two dispersal strategies) = 1000; H_1 (favorable habitat) = 0.1; α_1 (density-dependent suitability) = 3×10^{-5}; β_1 (density-independent suitability, favorable habitat) = 1, β_2 (density-independent suitability, unfavorable habitat) = 1.

Results of the analytical model for the adaptive value of directed dispersal

To illustrate the per capita net fitness gain (ΔF) as a function of the DrD level, Ω, we use an arbitrarily chosen set of parameter values. For each parameter set, the fitness gain rises to a maximum at Ω^*, and then drops off as Ω increases (Fig. 1). A larger ratio of density-dependent suitability (α ratio, i.e., a greater difference between the habitats in their suitability) entails a higher fitness gain for a given Ω, a higher value of Ω^* and a wider range of DrD levels having a positive fitness gain. For high ratios of density-dependent suitability Ω_b is equal to or greater than $1/H_1$, thus all DrD levels will have a positive effect on fitness gain compared to RD.

The combined effects of the density-dependent suitability ratio (α ratio) and the density-independent suitability ratio (β ratio) on Ω^* are shown (Fig. 2). Both ratios have a positive effect on Ω^* since a stronger difference between habitats implies a higher benefit of directing more seeds to the favorable habitat and thus higher Ω^*. The effect of the β ratio is significantly stronger that of α ratio as a small increase in the former leads Ω^* to its maximum ($1/H_1 = 10$; β ratio > 4 in this

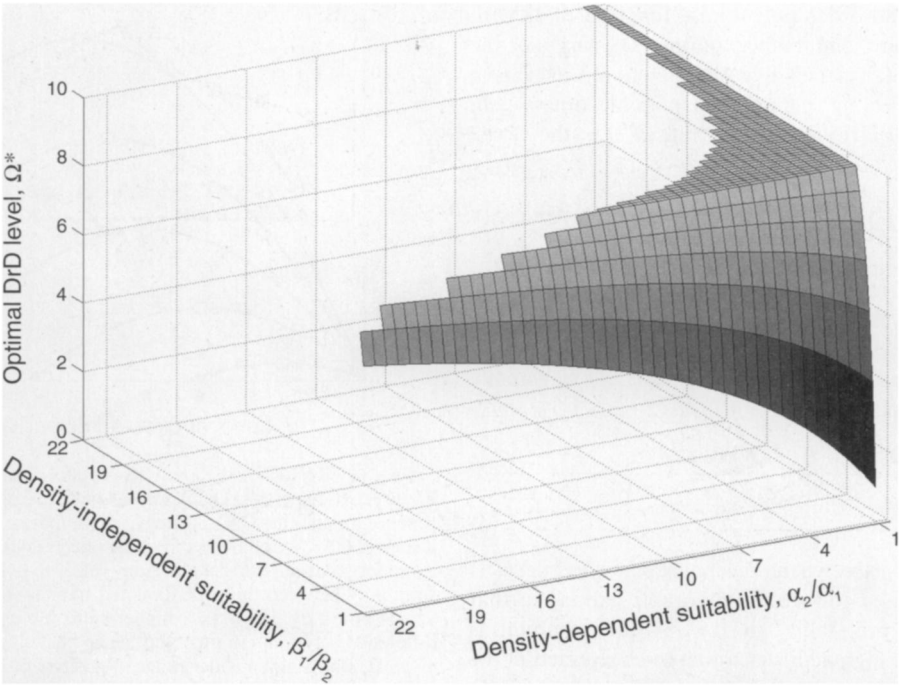

FIG. 2. The effect of differences in habitat suitability on optimal DrD levels (Ω^*) maximizing the net per capita fitness gain. Increasing values in both horizontal axes indicate increasing differences between habitats, either in density-independent suitability (β ratio, β_1/β_2) or in their density-dependent suitability (α ratio, α_2/α_1). The Ω^* value rises with differences between habitats, and finally levels up along both axes. The effect of the ratio of density-independent suitability diminishes as the ratio of density-dependent suitability increases. Other parameters were set as follows: $N = 1000$, $H_1 = 0.1$, $\alpha_1 = 3 \times 10^{-5}$, $\beta_1 = 1$.

specific case). For low β ratio values, the α ratio effect weakens toward its high values. See Appendix A for further discussion of the asymptotes of Ω^*.

A simulation model of a DrD mutant invasion in a RD population

While the stationary analytical model is suitable for testing our first and second predictions, a dynamic simulation model is required to test the third prediction on the temporal dynamics of the invasion process of a particular DrD strategy in a RD population and vice versa. We thus formulated a spatially implicit simulation model of this invasion process over successive nonoverlapping generations (Appendix C). We also incorporated variation in the level of Ω to test our fourth prediction that variance in Ω levels will negatively affect the invasion process of a DrD mutant (Appendix D). This model more realistically represents dispersal systems where the DrD level is averaged over all dispersal vectors and across spatial and temporal scales.

In general, the results of the simulation model agree with those of the analytical model, further highlighting the trade-off depicted by the DrD paradox. Yet, the simulation shows that some properties of the establishment process are actually facilitated by high DrD levels and not by intermediate levels as expected and sheds light on the temporal changes during this process (Fig. 3, Appendix C).

According to the simulation model, intermediate levels of DrD are likely to maximize fitness. Increasing DrD level adds to mutant population at the lower range of Ω but has an opposite effect at the higher range (Fig. 3A for α ratio = 6, 8 and also Fig. 3B for $\Omega = 1$, 10). These negative effects of high levels of DrD arise mainly from seed shortage in the unfavorable habitat that is not compensated by a higher success in the favorable habitat. However, the time required to establish (for the model runs during which mutants did not become extinct) shows a different pattern in which the invasion process is faster for the higher DrD levels of the invading mutant and not for intermediate DrD levels (Fig. 3C). In addition, mutant persistence ratio in the population increases with Ω (Fig. 3E). When inter-habitat differences increase, DrD is expected to be more beneficial because mutant advantage is maximized. Indeed, for a given Ω value, increasing α ratio facilitates invasion, increases the final mutant population size (Fig. 3A), and minimizes both time till establishment (Fig. 3C) and the chances for the mutant to go extinct (Fig. 3E).

The DrD level of the invaded population also influences its persistence and the invasion dynamics. An invaded population with an optimal DrD level ($\Omega^* = 5$) is completely resistant to invasion by DrD mutant of any Ω, including a RD mutant (hence the line $\Omega = 5$ is missing in the right-hand panels of Fig. 3). When the

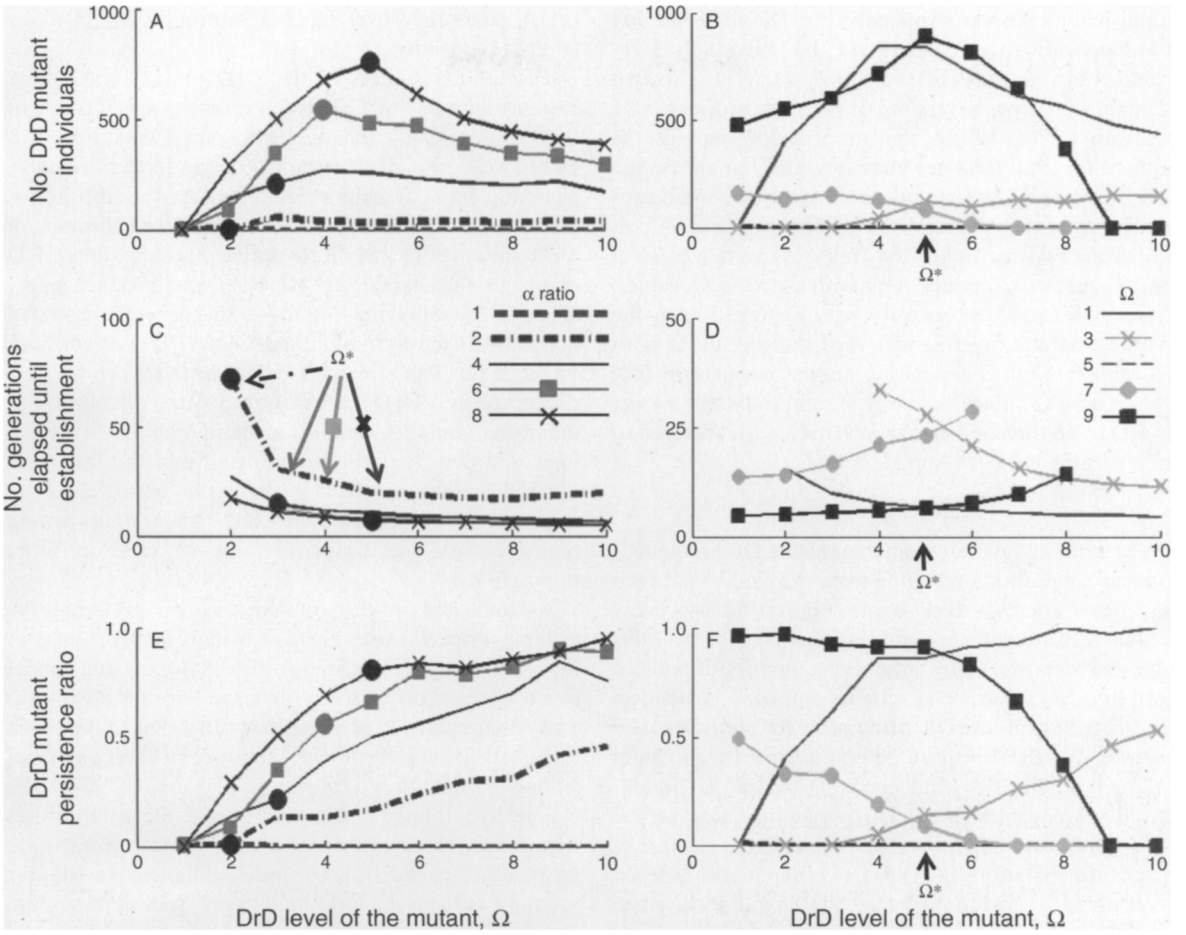

FIG. 3. Results of a simulation model (Appendix C) showing the effects of the DrD level (Ω) on the invasion process of the DrD mutants. The left-hand panels (A, C, E) show a single DrD mutant invading a randomly dispersed (RD) population under different values of habitat density-dependent suitability (α ratio; see key on figure). The right-hand panels (B, D, F) show a single DrD mutant invading a DrD population with different DrD levels (Ω values of the invaded population; see key). Three indices of invasion success are presented: (A, B) mutant final population size (average of number of DrD mutants in the last generation over all model runs; (C, D) the number of generations elapsed until establishment (defined as the mutant being at least 10% of the population); and (E, F) the proportion of model runs in which the mutant population persisted throughout the simulation. In the left-hand panels (A, C, E) the solid circles refer to the value of Ω^* for each line (i.e., they represent rounded values of analytically expected optimal Ω for each α ratio). In the right-hand panels (B, D, F) α ratio = 9 was used, implying a value of $\Omega^* = 5$, as indicated by the arrows on the *x*-axis. Other parameters were set as follows: $H = 0.1$, β ratio = 1, no. model runs = 100, no. generations = 100.

invaded population has a DrD value close to optimal it is invaded by fewer mutants (Fig. 3B), invasion takes a longer time (Fig. 3D) and occurs in a smaller proportion of cases (Fig. 3F). Moreover, an invaded population with a high DrD level can be easily invaded by a DrD mutant with low Ω, which directs enough seeds to establish in the nonfavorable habitat. Similarly, populations with low DrD levels are easily invaded by mutants with high DrD levels. This mechanism of unbalanced seed allocation between habitats at extreme DrD levels contributes also to shorter establishment times of the mutant invading the population (Fig. 3D), and to a higher probability of persistence (Fig. 3F).

To conclude, the results of the simulation model support the prediction that the DrD level plays an important role in determining the temporal dynamics of the invasion process. As predicted, the final number of DrD mutants invading a RD population, and the resistance of a DrD population to invading RD individuals, are maximized in *intermediate* DrD levels, suggesting an evolutionary stable strategy (ESS). Nevertheless, *high* DrD levels can facilitate both the likelihood and the duration of a DrD mutant establishment process in a RD population. This result was not expected intuitively from the analytical model, and arises from a rapid establishment in the favorable habitat first, and then in the entire population.

As anticipated from the hump-shaped fitness function described in the analytical model (Fig. 1), increasing variation in the DrD level negatively affects the

establishment process (Appendix D). The final number of mutant individuals decreases in 100 individuals as σ_Ω increases in one unit ($R^2 = 0.98$, $P = 0.001$). The two other indices show a very weak response to increasing variation in Ω. While the number of generations required for establishment increases, and mutant persistence decreases, the amplitudes of these significant trends are weak (Appendix D). The negative effects arise from two reasons. First, high σ_Ω values entail a high frequency of model runs with extreme Ω values. Those high Ω values intensify seed aggregation in the favorable habitat together with seed shortage in the nonfavorable habitat, thus limiting mutant population size. Second, low Ω values minimize mutant advantage over the RD in all three indices, as fewer seeds are directed to the favorable habitat (Appendix D).

Discussion

We highlight a paradox inherent to the DrD (directed-dispersal) hypothesis, resulting from the conflict between the positive effects of DrD by placing seeds in especially suitable recruitment sites, and the negative effects of this enhanced deposition by amplifying density-dependent mortality. In essence, we argue for applying basic ideal free distribution (IFD) principles to seed-dispersal research. We thus propose adding a third fundamental condition to the two (independent) traditional fundamental prerequisites for DrD (disproportionate arrival to disproportionately favorable establishment sites). The current formulation of the DrD hypothesis should therefore be revised to posit that "DrD will occur when a disperser disproportionately deposits seeds in exceptionally favorable sites, but without generating densities that can reduce the per capita survival of seeds below the level of randomly dispersed seeds." DrD is thus a frequency-dependent process that can be considered as an evolutionary stable strategy (ESS) at some intermediate levels, favorable in terms of fitness and resistance to invasions by other mutants (Fig. 3B). The dynamics and stability of the invasion process of DrD mutants in general, and the optimal DrD level in particular, correspond to the disperser attributes, to plant fecundity, and to the suitability and cover of the habitat.

Habitat, plant and vector properties constrain the optimal level of DrD and its adaptive value

The DrD paradox affects both the optimal level of DrD and its adaptive value for a given level. These two variables (Ω [the ratio of seeds arriving under DrD to seeds per RD] and ΔF [per capita net fitness gain of the DrD strategy over the RD strategy]) respond to the plant life-history traits (e.g., fecundity that is conservatively habitat independent in our model) as well as to habitat properties (e.g., habitat area and suitability for establishment; see Eq. 5 and Fig. 2). In general, the optimal level of DrD depends more on differences between habitats in their density-independent suitability (β ratio) than on their density-dependent suitability (α ratio), since the latter reaches saturation in Ω^* faster (Fig. 2; Appendix A).

The limited success of the DrD mutant with either extreme high or low DrD levels reflects a specific case of the "source–sink" dynamics (Pulliam 1988). For high DrD levels, biased allocation of seeds to the favorable (spatially limited) habitat leads to rapid establishment and facilitates mutant persistence. Yet, seed shortage of DrD mutants in the unfavorable habitat allows RD plants in this habitat to establish and produce many seeds. In the following generation this generates a spatial mass effect (sensu Shmida and Ellner 1984) of constant RD seed flux from the unfavorable to the favorable one, outcompeting DrD seeds in the latter habitat and impairing mutants establishment. Combined with the increased density-dependent mortality in the favorable habitat, these mass effects intensify the costs of the DrD strategy in comparison to RD, suggesting strong limitation on the adaptive value of DrD in finite populations.

As indicated in the *Introduction*, we preferred the random dispersal over the much more realistic case of localized (distance-declining) dispersal as a null model due to three main reasons. First, the selective advantage and disadvantage of localized dispersal have been discussed intensively in the literature in the context of sibling competition (Hamilton and May 1977), the escape hypothesis (Janzen 1970), and the colonization hypothesis (Howe and Smallwood 1982). Implementing localized dispersal in our models would have led our focus away from the DrD hypothesis. Second, assuming localized dispersal dictates additional assumptions about the shape of the dispersal kernel and the structure of the favorable habitat (Satterthwaite 2007), thereby considerably complicating our introductory treatment of the DrD paradox. Third, localized and distance-independent dispersal differ strongly only under the specific scenario combining no safe-site limitation and strong dispersal limitation (Satterthwaite 2007); that is, when arrival to empty suitable sites is limited by the dispersal distances. In the more general case of nearly all other scenarios, for which the DrD hypothesis is certainly relevant also, the qualitative results of the two alternative null models should converge. In fact, as explained in the following section, combining DrD with localized dispersal is expected to amplify the importance of considering the DrD paradox.

The DrD paradox explains apparent inconsistency between past empirical studies of DrD

The importance of the DrD paradox is probably underestimated by our assumptions (e.g., equal fecundity in favorable and unfavorable habitats, constant population size, and, especially, uniform exploitation of sites within each habitat). It is unlikely that favorable sites within a given habitat are all utilized equally by the dispersal vectors. Animals tend to produce clumped seed shadows with seeds aggregated at various density

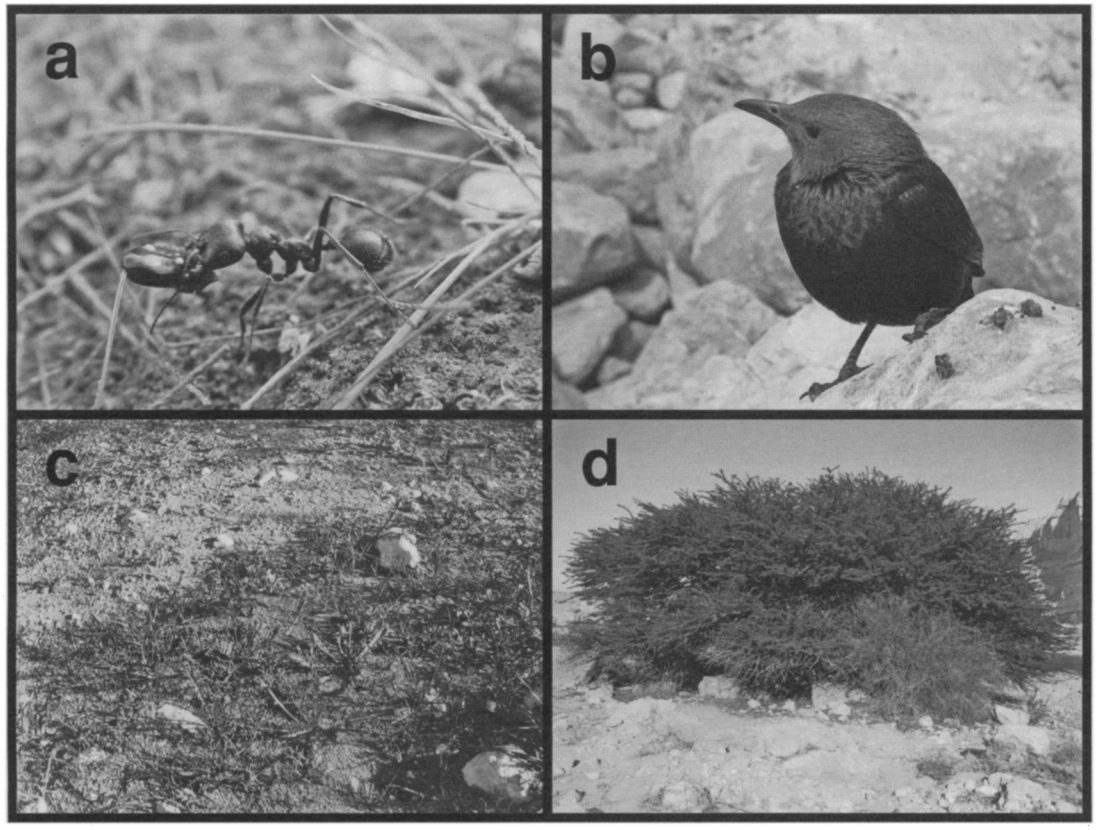

PLATE 1. Dispersal vectors and seed deposition sites customarily considered as examples for directed dispersal into favorable establishment sites, yet possibly representing dispersers that generate seed aggregations enhancing density-dependent mortality. (a) Harvester ant (*Messor arenarius*) carring a seed; (b) Tristam's grackle (*Onychognathus tristramii*) with feces containing *Ochradenus baccatus* seeds. Both species occasionally generate high seed aggregations at otherwise preferrred establishment sites at (c) ant nests and (d) under acacia trees, respectively. Photo credits: panel (a) Ofir Altstein; panels (b–d) O. Spiegel.

hotspots along their movement path, such as at roosting sites, feeding sites, and ant nests (Schupp et al. 2002, Clark et al. 2004, Giladi 2006, Russo et al. 2006; see also Plate 1). Also the leptokurtic nature of most dispersal kernels (Nathan and Muller-Landau 2000), where most seeds are deposited beneath or very close to the mother plant, probably contributes to the unequal utilization of sites.

Clumped seed shadows are expected to amplify density effects and favor lower DrD levels than would have been predicted by the "classical" DrD hypothesis since the effective size of the favorable habitat is smaller than its real size, and the local densities are much higher. For instance, myrmecochory (ant-mediated dispersal) is often thought of as an illustrative example of DrD, yet more than a half of the studies directly testing DrD found no support for the hypothesis (Giladi 2006). Similarly, dispersal of large tropical fruits by nesting hornbills reduces seed survival due to increased density-dependent mortality (Kitamura et al. 2004). We can speculate that many equally suitable potential establishment sites are not in use, thus diminishing the effective habitat size. Other dispersal systems where the DrD hypothesis was tested and rejected, presumably due to the same effect, include the dispersal of acorns by wood mice (Iida 2006) and the dispersal of the desert shrub *Ochradenus baccatus* by two frugivorous bird species (Spiegel and Nathan 2007; see Plate 1). In the latter dispersal system, birds concentrated their activity in specific habitats, resulting in increased density-dependent mortality in these otherwise-favorable establishment habitats (O. Spiegel and R. Nathan, *unpublished data*). This may lead to publication bias, since a failure to find empirical support to the DrD hypothesis is less likely to be published in a DrD context.

One of the classical examples of the DrD hypothesis is the elegant work by Wenny and Levey (1998), where male bellbirds preferably disperse seeds into canopy gaps under perching points. While considering habitat suitability for establishment as constrained by factors that do not respond to seedling density (e.g., fungal pathogens that are limited by light intolerance), the authors concluded that male bellbirds provide DrD to the *Ocotea endresiana* seeds. Further investigation of density-dependent factors that influence seed and seedling mortality of this species in the same ecosystem (e.g., predation by

rodents) had altered the conclusion and led to the statement that "Whether habitual perches represent foci of seedling recruitment or lead to density-dependent seed and seedling mortality, needs to be examined in more detail" (Wenny 2000:345). Similarly, previous studies hypothesized that roost sites and leks may function as a focus for DrD (Wenny 2001). Nevertheless, consideration of the increased density-dependent mortality associated with enhanced arrival may lead to reexamination of these ideas and to a refinement of DrD predictions and more appropriate experimental design for testing these predictions.

Another classic example of DrD by birds is the dispersal of the mistletoe by small passerines to favorable establishment locations on host branches. Contrary to the previous examples and to the common situation we explore in our models, where density has a negative effect on seed survival, increasing parasite numbers on a given tree weakens the host resistance and facilitates mistletoe establishment (Aukema and del Rio 2002), though in extreme cases it may eventually lead to host death. In this example, the positive effects of DrD are not negated by negative density effects (at least until some very high limit), and high DrD levels are likely to be selected for. Also DrD provided by granivores who cache seeds in safe sites (often as secondary dispersal) is less likely to be constrained by the negative density effect due to the scatter nature of the caching sites (Briggs et al. 2009).

Evolutionary implications of the revised DrD hypothesis

The classic formulation of the DrD hypothesis implies no constraints on potential "DrD traits," such as fruit size and pulp-to-seed ratio, which might contribute to plant fitness by attracting dispersers that efficiently direct seeds to favorable establishment sites. By highlighting a fundamental trade-off inherently caused by DrD traits, the revised DrD hypothesis implies that selection should promote traits leading to intermediate levels of DrD, for example by introducing within-plant variation in potential DrD traits, or a combination of DrD and contra-DrD traits. However, this pattern may be obscured by the fact that for many dispersal systems only a fraction of the crop is dispersed, and other selective forces (e.g., seed predation and post-dispersal survival) may have countering effects on these traits (García et al. 2005).

The DrD paradox is intensified by the intrinsic stochasticity of natural ecosystems, where both habitat suitability, and, even more so, vector activity, are expected to vary in time and space. This variation presents an additional challenge to the invasion process of the DrD strategy in a RD wild-type population (Appendix D), suggesting that the invasion may be strongly influenced by the consistency (amount of variation around the mean) of the vector.

More specifically, we have shown that differences between dispersal vectors in their variation around the mean DrD level, and not only the mean DrD level itself, may strongly impact disperser effectiveness, suggesting that both the identity and the variety of dispersal vectors might affect plant success. If a single vector could constantly fulfill the desired optimal DrD level (Ω^*) across large spatiotemporal scales, this might encourage selection of seeds adapted for this specific vector, and the emergence of species-specific dispersal interactions. We propose, however, that due to fluctuation in vector abundance and/or behavior across large spatiotemporal scales (e.g., Herrera 1998), a single vector is unlikely to constantly provide the desirable DrD level (Ω^*) across these scales. Additionally, a single vector may intensify seed aggregations because of its specific nonuniform use of the sites within a given habitat, thus increasing fluctuations in DrD level between sites or across time intervals. The importance of disperser consistency suggests that a multi-vector dispersal strategy may be more beneficial for a plant. The multi-vector strategy, probably the rule rather than the exception (Nathan et al. 2008), may lessen the fluctuations around the mean level of DrD, preventing a situation where a single supposedly effective disperser aggregates seeds to high densities, leading to disproportional mortality. This conclusion agrees with the current paradigm suggesting weak co-evolutionary plant–disperser linkage even for classic DrD cases (Bascompte et al. 2006).

Implications of the revised DrD hypothesis for management and future research

Restoration of human-disrupted habitats, where DrD is pronounced (Wenny 2001), may benefit from considering the effect of the assemblage of the disperser community and the effect of the environment on their movement pattern and not only the contribution of a single or multiple disperser (Wunderle 1997, Au et al. 2006, Buckley et al. 2006, Nathan et al. 2008). Additionally, our results stress the importance of the consistency of the disperser (predictability of its dispersal service over time and uniformity of habitat use) for assessing its actual effectiveness in an applied ecological context. Thus, we call to integrate disperser consistency into the qualitative component of the "disperser effectiveness" framework proposed by Schupp (1993).

The integration of density effect into meta-population models accounting for nonrandom dispersal (e.g., Purves and Dushoff 2005) will provide more accurate evaluation of patch persistence. In addition to other widely acknowledged effects, habitat loss can encourage high DrD levels in the remaining patches, consequently turning favorable patches into unfavorable ones due to increased seed densities (e.g., Wenny 2000, Purves and Dushoff 2005). Where anthropogenic activity intensifies habitat degradation, this may lead to an ecological trap, where formerly beneficial vectors and arrival cues decrease plant fitness (Gilroy and Sutherland 2007).

Thus, future studies assessing the impact of habitat loss should account for the negative potential of high DrD levels.

Our models are simplistic; they incorporate only a few parameters and disregard other potential influencing factors (e.g., localized dispersal, inter-habitat differences in fecundity, differential survival or inter-annual differences of adult size and other factors). Yet, they are sufficient to highlight the DrD paradox and its potential to explain a wide variety of frequently observed patterns. Future empirical studies testing the DrD hypothesis need to consider the features of our models that were found most critical to the revised DrD hypothesis. For example, field measurements of density-dependent and density-independent suitability of habitats may provide the essential parameters (H, α ratio, and β ratio) for generating quantitative predictions of optimal and beneficial DrD levels (Ω^*, Ω_a, and Ω_b). Most importantly, as density effects may appear only at late stages of seedling establishment, any habitat-quality assessment should be determined based on a sufficient time interval (Schupp and Fuentes 1995). Then, one can quantify the quality of deposition (e.g., Schupp 1993) and DrD level of the focal vectors and make conclusions concerning their dispersal effectiveness and potential contribution to plant fitness.

To conclude, we suggest revising the DrD hypothesis to explicitly incorporate a third component, the detrimental effect that its first component (enhanced arrival) has on its second (enhanced survival). The revised hypothesis accounts for the widespread pattern of density-dependent mortality, and provides a unifying concept for studies testing the advantageous aspects of seed dispersal. It also facilitates future empirical studies of this discipline by emphasizing important and measurable parameters.

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APPENDIX A

Analytical model for the directed-dispersal (DrD) paradox (a linear version) (*Ecological Archives* E091-106-A1).

APPENDIX B

Analytical model for the DrD paradox (the quadratic version) (*Ecological Archives* E091-106-A2).

APPENDIX C

Simulation model of a DrD mutant invasion in a random-dispersal population (*Ecological Archives* E091-106-A3).

APPENDIX D

The effect of variation in the DrD level on mutant establishment (*Ecological Archives* E091-106-A4).