Density-dependent selection in evolutionary genetics: a lottery model of Grime's triangle

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Abstract

The classic lottery model is generalized to accomodate arbitrary densities. We use the model to predict the direction of trait evolution under different environmental conditions and thereby provide a mathematical underpinning for Grime's verbal theory of plant primary strategies. We evaluate the density-dependent lottery model as a general-purpose model of density-dependent selection in the spirit of MacArthur's r/K theory.

"...the concept of fitness is probably too complex to allow of a useful mathematical development. Since it enters fundamentally into many population genetics considerations, it is remarkable how little attention has been paid to it." — Warren J. Ewens, Mathematical Population Genetics I, 2004

Introduction

Evolutionary models differ greatly in their treatment of fitness. In models of genetic evolution, genotypes are typically assigned constant (or frequency-dependent) selection coefficients describing the change in their relative frequencies over time due to differences in viability. This considerably simplifies the mathematics of selection, facilitating greater genetic realism, and can be justified over sufficiently short time intervals (Ewens, 2012, p. 276). However, selection can have very different effects when operating on different types of traits, and evolutionary changes in one population can lead to complicated ecological responses.

By contrast, models of phenotypic trait evolution represent the change in phenotypic abundances over time using absolute fitness functions which describe how those traits affect survival and reproduction in particular ecological scenarios. This approach is powerful enough to model eco-evolutionary feedbacks between co-evolving traits, but is generally problem-specific and restricted to only a few traits at a time.

Far less work has been done to model fitness in more general terms than particular traits or ecological scenarios, while still capturing key distinctions between different forms of selection. Perhaps this is not surprising given that fitness is such a complex quantity, dependent on all of a phenotype's functional traits (Violle et al., 2007) as well as its biotic and abiotic environment. In most cases, a detailed, trait-based, predictive model of fitness would be enormously complicated and have narrow applicability. It is therefore easy to doubt the feasibility of a simplified, general mathematical treatment of fitness (Ewens, 2012, p. 276). Even MacArthur's famous r/K selection scheme is now almost exclusively known as a framework for understanding life-history traits,

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and judged on its failure in that role (Boyce, 1984; Pianka, 1970; Reznick et al., 2002; Stearns, 1977). In spite of the r/K scheme's original purpose as an extension of the existing population-genetic treatment of selection to account for population density (MacArthur, 1962), comparatively few attempts have been made to develop it further as a mathematical analysis of the major different forms of selection.

Nevertheless, there are strong indications there are broader principles governing the operation of selection. In many groups of organisms, including corals (Darling et al., 2012), insects (Southwood, 1977), fishes (Winemiller and Rose, 1992), zooplankton (Allan, 1976) and plants (Grime, 1988; Westoby, 1998), different species can be divided into a small number of distinct trait clusters corresponding to fundamentally distinct "primary strategies" (Winemiller et al., 2015). The most famous example is Grime's plant trait classification scheme (Grime, 1974, 1977, 1988). Grime considered two broad determinants of population density: stress (persistent hardship e.g. due to resource scarcity, unfavorable temperatures or toxins) and disturbance (intermittent destruction of vegetation e.g. due to trampling, herbivory, pathogens, extreme weather or fire). The extremes of these two factors define three primary strategies denoted by C/S/R respectively: competitors "C" excel in low stress, low disturbance environments; stress tolerators "S" excel in high stress, low disturbance environments; and ruderals "R" excel in low stress, high disturbance environments. Survival is not possible in high-stress, high-disturbance environments. Grime showed that measures of C, S and R across a wide range of plant species are anti-correlated, so that strong C-strategists are weak S and R strategists, and so on. Thus, plant species can be classified on a triangular C/S/R ternary plot (Grime, 1974). Trait classification schemes for other organisms are broadly analogous to Grime's scheme (Winemiller et al., 2015).

Trait classification schemes show empirically that, beneath the complicated details of trait variation, even among closely-related species, fitness is predominantly determined by a few key factors such as intrinsic reproducive rate or stress-tolerance. However, while trait classification schemes are firmly grounded in trait data, they are verbal and descriptive rather than mathematical, a recognized hinderance to their broader applicability (e.g. (Tilman, 2007)).

The aim of this paper is explore the interplay between some major dimensions of fitness in a simplified, spatially-homogeneous model of genotype growth, dispersal and competition. Building on the earlier r/K and C/S/R schemes, a central question is how fitness depends on the interaction between population density, intrinsic birth/death rates and competitive ability.

We broadly follow the spirit of MacArthur's r/K selection scheme in that our model is intended to account for fundamentally different forms of selection without getting entangled in the intricacies of particular ecological scenarios. However, rather than building directly on MacArthur's formalism and its later extensions using Lotka-Volterra equations to incorporate competition ("α-selection") (Case and Gilpin, 1974; Gill, 1974; Joshi et al., 2001), our model is devised primarily with Grime's C/S/R scheme in mind, and represents a quantitative formalization of how C/S/R manifests at the level of genotype evolution (as opposed to divergence between species). This choice is motivated in part by the substantial empirical support for the C/S/R scheme, and in part by the failings of the r/K low/high density dichotomy — many growth ability traits will confer advantages at both low and high densities (more details in the Discussion).

In section "Model" we introduce the basic assumptions of our generalized lottery model. Analytical expressions for the change in genotype abundances over time are introduced in section "Mean field approximation", with mathematical details relegated to the Appendices. The following two sections discuss the behaviour of rare mutants and our treatment of Grime's triangle.

Model

We assume that each individual in a population requires its own territory to survive and reproduce (a site-occupancy model). All territories are identical, and the total number of territories is T. Time t advances in discrete iterations, each representing the average time from birth to reproductive maturity. In iteration t, the number of reproductively mature individuals (henceforth called "adults") of the i'th genotype is $n_i(t)$, the total number of adults is $N(t) = \sum_i n_i(t)$, and

the number of unoccupied territories is $U(t) = T - N(t)$.

Each iteration, adults produce m_i new offspring (henceforth called "propagules") which disperse at random over the U unnocupied territories (no dispersal limitation). We assume adults cannot be ousted from occupied territories, so only propagules landing on occupied territories are included in m_i. More generally, m_i does not include propagules which never even begin the development cycle. For simplicity, we assume $m_i = b_i n_i$, where b_i is a constant, genotype-specific birth rate.

The number of individuals of the i'th genotype landing in any particular territory is denoted x_i. Random dispersal implies that in the limit $K \to \infty$, with n_i/K held fixed, x_i is Poisson distributed with mean territorial propagule density $l_i = m_i/U$. Although T is finite in our model, we assume that K and the n_i are large enough that x_i is Poisson-distributed to a good approximation (details in Appendix A). This dispersal Poisson distribution is denoted $p_i(x_i)$. Note that the large n_i, large T approximation places no restrictions on our densities n_i/T, but it does preclude consideration of demographic stochasticity when n_i itself is very small (this will be discussed further in Section).

When multiple propagules land on the same territory, they compete to secure the territory as they develop. This territorial contest is modeled as a weighted lottery: the probability that genotype i wins a given territory by the next iteration is $c_i x_i / \sum_j c_j x_j$ where c_i is a constant representing relative competitive ability.

The increase in n_i over one iteration due to territorial acquisition, $\Delta_+ n_i$, is the sum of genotype i's victories over all U unoccupied territories. Since $p_1(x_1) \ldots p_G(x_G)$ is equal to the proportion of unoccupied territories with $x_1, \ldots, x_G$ of the respective propagules (again, we assume that T is large enough that fluctuations in this proportion are negligible), this sum can be replaced by an expectation over the p_i. This gives

$$\Delta_+ n_i(t) = U(t) \sum_{x_1, \ldots, x_G} \frac{c_i x_i}{\sum_j c_j x_j} p_1(x_1) \ldots p_G(x_G). \tag{1}$$

In addition to propagule birth and competition, occupied territories become unoccupied due

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to mortality. For the vast majority of this manuscript we assume that mortality only occurs in adults, and at a constant, genotype-specific per-capita rate d_i, so that the overall change in genotype abundances is

$$\Delta n_i(t) = \Delta_+ n_i(t) - d_i n_i(t). \tag{2}$$

We will introduce a different mortality model when we consider the effects of disturbances (Section **??**), which will also affect competing juveniles.

Note that the competitive ability coefficients c_i represent a strictly relative aspect of fitness in the sense that they only influence population size N indirectly by changing genotype frequencies; that may in turn change the population mean birth and death rates. This can be seen by summing Eq. (2) over genotypes to get the change in population size N,

$$\Delta N = U(1 - e^{-L}) - \sum_i d_i n_i, \tag{3}$$

which is independent of c_i (here $L = \sum_j l_j$ is the overall propagule density).

Results

Mean Field Approximation

Eq. (2) gives little intuition about the dynamics of density-dependent lottery competition, since (1) involves an expectation over the random dispersal distributions p_i, which depend on how the n_i change over time. We now evaluate this expectation using a "mean field" approximation; the intuition behind this approximation is as follows.

If the unoccupied territories are saturated with propagules from every genotype ($l_i \gg 1$ for all genotypes), the fluctuations in the x_i are small compared to their means l_i (since the x_i are Poisson distributed), and so the composition of propagules in a territory will only rarely differ appreciably from the mean composition $l_1, l_2, \ldots, l_G$. Consequently, we can replace x_i with l_i in Eq. (1). This gives the classic lottery model (Chesson and Warner, 1981),

$$\Delta_+ n_i(t) = U(t) \frac{c_i m_i}{\sum_j c_j m_j} = b_i n_i \frac{1}{L} \frac{c_i}{\bar{c}}, \tag{4}$$

where $\bar{c} = \sum_j c_j m_j / M$ is the mean propagule competitive ability for a randomly selected propagule ($M = \sum_j m_j$ is the total number of propagules).

However, in general the l_i are not all large, and the x_i cannot simply be replaced by their means in Eq. (1). Indeed, Eq. (4) is nonsensical if l_i is sufficiently small: genotype i can win at most m_i territories, yet Eq. (4) demands a fraction $c_i m_i / \sum_j c_j m_j$ of the unoccupied territories U, no matter how large U is. The source of this pathological behavior when $l_i \ll 1$ is that $x_i = 1$ in the few territories where i propagules do land, and so i's growth comes entirely from territories which deviate appreciably from the mean.

Our mean field approximation is similar to the high-l_i approximation leading to Eq. (4) in that we replace the x_i with appropriate mean values. The key distinction is that territories with a single propagule from the focal genotype, which are critical at low densities, are handled separately. In place of the requirement of $l_i \gg 1$ for all i, our approximation only requires that there are no large discrepancies in competitive ability (specifically, that we do not have $c_i / c_j \gg 1$ for any two genotypes; further discussion in section "Discussion"). We obtain (details in Appendix B)

$$\Delta_+ n_i(t) \approx b_i n_i \left[e^{-L} + (R_i + A_i) \frac{c_i}{\bar{c}} \right],\tag{5}$$

where

$$R_i = \frac{\bar{c} e^{-l_i}(1 - e^{-(L-l_i)})}{c_i + \frac{L-1+e^{-L}}{1-(1+L)e^{-L}} \frac{\bar{c}L - c_i l_i}{L - l_i}},\tag{6}$$

and

$$A_i = \frac{\bar{c}(1 - e^{-l_i})}{c_i l_i \frac{1-e^{-l_i}}{1-(1+l_i)e^{-l_i}} + \sum_{j \neq i} \frac{c_j l_j}{L - l_j} \left(L \frac{1-e^{-L}}{1-(1+L)e^{-L}} - l_j \frac{1-e^{-l_j}}{1-(1+l_j)e^{-l_j}} \right)}.\tag{7}$$

Comparing Eq. (5) to Eq. (4), the classic lottery per-propagule success rate $c_i / \bar{c} L$ has been replaced by three separate terms. The first, e^{-L}, accounts for propagules which land alone on unoccupied territories; these territories are won without contest. The second term, $R_i c_i / \bar{c}$ represents competitive victories when the i genotype is a rare invader in a high density population: from Eq. (6), $R_i \to 0$ when the i genotype is abundant ($l_i \gg 1$), or other genotypes are collectively rare ($L - l_i \ll 1$). The third term, $A_i c_i / \bar{c}$, represents competitive victories when the i genotype

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is abundant: $A_i \to 0$ if $l_i \ll 1$. The relative importance of these three terms varies with both the overall propagule density L and the relative propagule frequencies l_i/L. If $l_i \gg 1$ for all genotypes, we recover the classic lottery model (only the $A_i c_i / \bar{c}$ term remains, and $A_i \to 1/L$). Thus, Eq. (5) generalizes the classic lottery model to account for arbitrary propagule densities for each genotype.

Fig. 1 shows that Eq. (5) (and its components) closely approximate direct simulations of random dispersal and lottery competition over a wide range of propagule densities (obtained by varying U). Two genotypes are present, one of which has a c-advantage and is at low frequency. The growth of the low-frequency genotype relies crucially on the low-density competition term $R_i c_i / \bar{c}$, and also to a lesser extent on the high density competition term $A_i c_i / \bar{c}$ if l_1 is large enough (Fig. 1b). On the other hand, $R_i c_i / \bar{c}$ is negligible for the high-frequency genotype, which depends instead on high density territorial victories (Fig. 1d).

Invasion of rare genotypes and coexistence

To determine how b, c and d will evolve in a population where those traits are being modified by mutations, we need to know whether mutant lineages will grow (or decline) starting from low densities. In this section we discuss the behavior of rare genotypes predicted by Eq. (5).

Suppose that a population with a single genotype i is in equilibrium. Then $R_i = 0$, $\bar{c} = c_i$ and $\Delta n_i = 0$, and so Eq. (5) gives

$$b_i \left(e^{-L} + A_i \right) - d_i = 0. \tag{8}$$

Now suppose that a new genotype j, which is initially rare, appears in the population. Then $A_j \ll 1$, $l_j \ll L$ and $\bar{c} \approx c_i$, and so, from Eq. (5), n_j will increase if

$$b_j \left(e^{-L} + R_j \frac{c_j}{c_i} \right) - d_j > 0. \tag{9}$$

Combining Eqs. (8) and (9), it is easily verified that if j is superior in one trait, but otherwise identical to i, it will invade. Moreover, j will eventually exclude i, since it is strictly superior. However, stable coexistence is possible between genotypes that are superior in different traits.
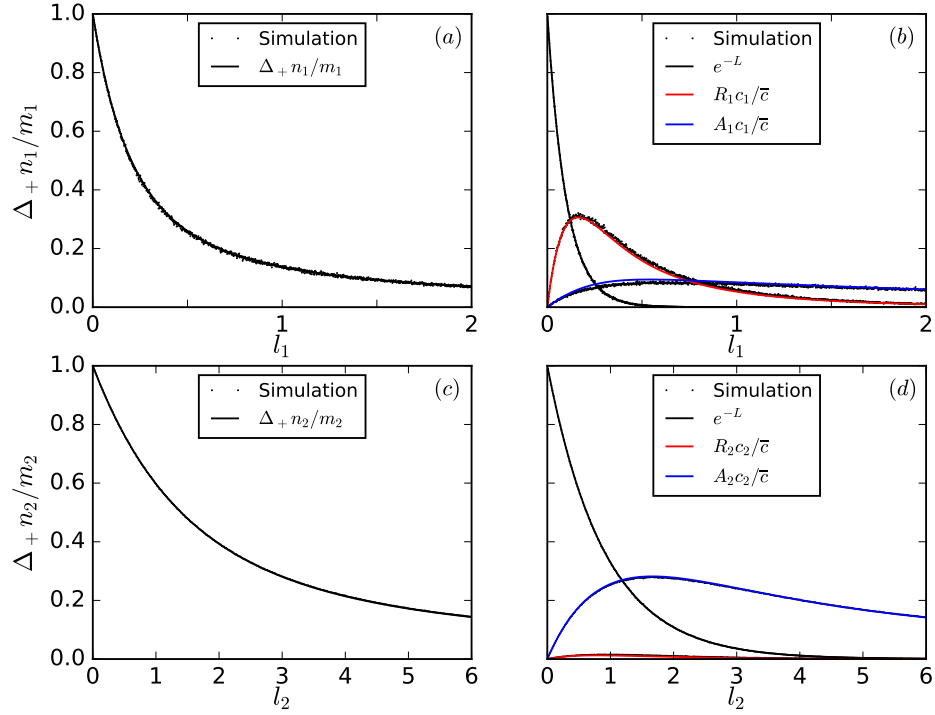
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Figure 1: The change in genotype abundances in a density dependent lottery model is closely approximated by Eq. (5). $\Delta_+ n_i / m_i$ from Eq. (5) (and its separate components) are shown, along with direct simulations of random dispersal and lottery competition over one iteration over a range of propagule densities (varied by changing U with the m_i fixed). Two genotypes are present. (a) and (b) show low-frequency genotype with c-advantage ($m_1/M = 0.1$, $c_1 = 1.5$), (c) and (d) show the high-frequency predominant genotype ($m_2/M = 0.9$, $c_2 = 1$).
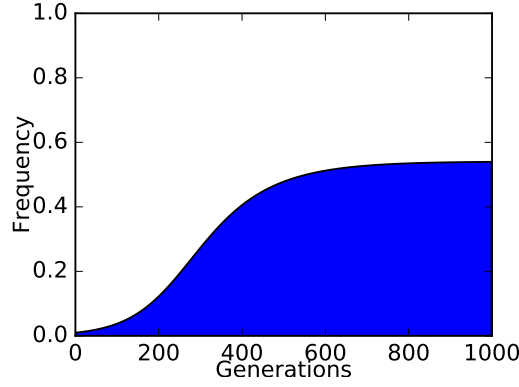
Figure 2: Coexistence between b ($c_i = 1$, $b_i = 1$) and c ($c_j = 2$, $b_j = 0.7$) specialists, where $d_i = d_j = 0.3$. Vertical axis shows frequency of the c-specialist predicted by Eq. (5).

To illustrate, suppose that j is better at securing territories ($c_j > c_i$), that i is better at producing propagules ($b_i > b_j$), and that $d_i = d_j$. Coexistence occurs if j will invade an i-dominated population, but i will also invade a j-dominated population ("mutual invasion"). It is not hard to show that this is possible, since if b_i is so large that $L \gg 1$ when i is dominant, and b_j is so small that $L \ll 1$ when j is dominant, then, combining Eqs. (8) and (9), we find that i invades j because $b_i > b_j$, while j invades i provided that

$$b_j c_j R_j - b_i c_i A_i > 0. \tag{10}$$

Thus, coexistence occurs if c_j is large enough. Intuitively, the mechanism for coexistence is that territorial contests are important in an i-dominated population (high L), ensuring that the c-specialist j is not excluded, yet territorial contests are irrelevant in a j-dominated population (low L), ensuring that the b-specialist i is not excluded. Fig. 2 shows an example of this coexistence between b and c specialists.

A similar argument applies for coexistence between high-c and low-d specialists; again coexistence occurs because the importance of territorial contests declines along with propagule density L as the c-specialist increases in frequency. Coexistence is technically possible between b- and d-specialists which exactly satisfy $b_i/d_i = b_j/d_j$ (this follows from the fact that all propagules have

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the same probability of success when $c_i = c_j$ i.e. $A_i + R_i = A_j + R_j$). However, this coexistence scenario is not biologically relevant, since the tiniest deviation from $b_i/d_i = b_j/d_j$ will lead to the eventual exclusion of the genotype with greater b_i/d_i.

If the rare genotype j arises due to mutation, then it's initial low-density behavior is more complicated than the above invasion analysis suggests. The mutant lineage starts with one individual $n_j = 1$, and remains at low abundance for many generations after its initial appearance. During this period, the mutant abundance n_j will behave stochastically, and the deterministic equations (1) and (5) do not apply (Section). However, if n_j becomes large enough, its behavior will become effectively deterministic, and governed by Eq. (5). For mutants with fitness greater than the population mean fitness, this process is known as "establishment", and occurs when n_j is of order $1/s$, where s is the mutant's fitness advantage relative to the mean (Desai and Fisher, 2007). Here we do not consider the initial stochastic behavior of novel mutants, and have restricted our attention to the earliest deterministic behavior of rare genotypes. In particular, for beneficial mutations we have only considered the case where s is large enough that deterministic behavior starts when $n_j \ll N$.

Primary strategies and Grime's triangle

We now discuss which changes in the traits b, c and d will be most favored under different environmental conditions. Of particular interest are Grime's "disturbance", "stress" and "ideal" environmental archetypes. To proceed, we need to map these verbal archetypes to quantitative parameter regimes in our model.

The ideal environmental archetype is characterized by the near-absence of stress and disturbance. Consequently, $d_i \ll 1$, whereas b_i is potentially much larger than 1. From Eq. (3), the equilibrium value of L only depends on the ratio of birth and death rates. For one genotype, $L/(1 - e^{-L}) = b_i/d_i$, and so the propagule density is high $L \approx b_i/d_i \gg 1$. Moreover, since $L = b_i N/(N - T)$ by definition, population density is also high $N/T \approx 1$. Thus, territorial contests are decisively important.

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The disturbance archetype is characterized by unavoidably high extrinsic mortality caused by physical destruction. Disturbances do not only affect adults as in Eq. (2), but also juveniles in the process of territorial contest. These juvenile deaths can be represented as a fractional reduction in the number of territories secured. To illustrate, we assume that the disturbance is equally damaging to adults and juveniles, so that only $(1 - d_i)\Delta_+ n_i$ rather than $\Delta_+ n_i$ territories are secured by genotype i each iteration. Then, the disturbance archetype is characterized by d_i being close to 1 for all genotypes (almost all adults and juveniles are killed each iteration). The single genotype equilibrium then gives $L \approx 2(1 - d_i/[(1 - d_i)b_i])$, where b_i must be exceptionally large to ensure population persistence, and we have $L \ll 1$ and $N/T \ll 1$. The terms proportional to $c_i/\bar{c}$ in Eq. (5) are then negligible, and $\Delta_+ n_i$ depends primarily on b_i.

The stress archetype is more ambiguous, and has been the subject of an extensive debate in the plant ecology literature (the "Grime-Tilman" debate; Aerts 1999). Stressful environments severely restrict growth and reproduction, so that $b_i \ll 1$ Grime (1974, 1977). Mutations which appreciably improve b_i will be either non-existent or extremely unlikely, so b_i is constrained to remain low. In Grime's view, under these conditions the rate at which propagules successfully develop to adulthood cannot appreciably exceed the mortality rate. This implies $b_i/d_i \approx 1$ in our model, and so the propagule density L is suppressed to such low levels that there are essentially no territorial contests occurring.

The alternative view is that stressful environments simply have a lower carrying capacity (Taylor et al., 1990); in our model, this means a greater per-individual territorial requirement represented by a lower T for a given amount of space. In particular, it is argued that when stress is induced by a scarcity of consumable resources, competition for those resources would likely be intense. Thus, b_i need not be particularly close to d_i, but contests for consumable resources at the juvenile phase would kill off most propagules before adulthood. In other words, the stressed population is a high density population where competition is important (Taylor et al., 1990) (that is, high density relative to T, not relative to ideal conditions).

The mapping of environmental achetypes to our model parameters is summarized in the first

two rows of Fig. 3. Also shown is the approximate dependence of $\Delta_+ n_i$ on b_i and c_i for each archetype (third row). These can be used infer the expected direction of evolution for the traits b, c and d (fourth row) as follows.

As noted in the previous section, if beneficial mutations establish (i.e survive the low-abundance stochastic regime), they will proceed to grow deterministically according to Eq. (5). The probability of establishment increases with the mutant fitness advantage, and is therefore typically on the order of one percent, whereas the fixation of neutral mutations is exceedingly unlikely (probability of order $1/N$). Consequently, the direction of evolutionary change is determined by which trait changes confer an appreciable benefit, subject to the constraints imposed by the environment.

For example, in Grime's version of the stress archetype, L is low, so competition is not important, and only mutants with greater b or lower d will have an appreciably greater Δn_i. Mutations in c are effectively neutral, and will rarely fix. However, by definition of the stress archetype, b is constrained to be small. Thus, while some rare mutations may produce small improvements in b, it is much more likely that mutations will arise that lower d, making this the expected direction of evolutionary change for Grime's stress archetype.

Following Grime's original argument for a triangular scheme (Grime, 1977), Fig. 4 represents each environmental archetype schematically as a vertex on a triangular space defined by perpendicular stress and disturbance axes. The ideal archetype lies at the origin (no stress or disturbance), while the stress and disturbance archetypes lie at the limits of survival on their respective axes. The hypotenuse connecting the stress and disturbance endpoints represents the limits of survival in the presence of a combination of stress and disturbance. The direction of evolutionary change is different at each vertex, leading to the emergence of different trait clusters or "primary strategies".

How does Fig. 4 compare to Grime's C/S/R strategies? In our comparison we will stick to fishes, corals and plants, for which three-way primary strategy schemes are well developed (Darling et al., 2012; Grime, 1977; Winemiller and Rose, 1992). The connection of our model to

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	Ideal	Disturbance*	Stress (G)	Stress (T)
Parameter-	$d \ll 1$	$d \approx 1$	$b \ll 1$	$b \ll 1$
regime	$b/d \gg 1$	$b/d \gg 1$	$b \approx d$	$b > d$
Density N/T	High	Low	Low	High
$\Delta_+ n_i \propto$	$b_i c_i$	b_i	b_i	$b_i c_i$
Evolution for	$\uparrow b, \uparrow c$	$\uparrow b, \downarrow d$	$\downarrow d$	$\uparrow c, \downarrow d$

Figure 3: The realization of Grime's environmental archetypes in our model, as well as the low-T variant of the stress archetype. Shown are the mapping to our parameters of each archetype, the approximate dependence of $\Delta_+ n_i$ on b_i and c_i, as well as the corresponding expected evolutionary changes in b_i, c_i and d_i. *Mortality affects both adults and juveniles in the disturbance archetype, with $\Delta_+ n_i$ replaced by $(1 - d_i)\Delta_+ n_i$ in Eq. (2).
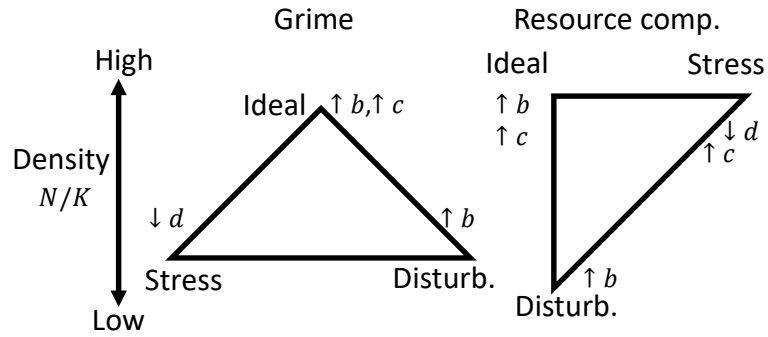

Figure 4: The realization of Grime's triangle in our model. Schematic representation of the triangular space bounded by the low/high extremes of stress/disturbance. The low-T interpretation of stress is also shown. The vertices of the triangles correspond to environmental archetypes. Selection favors different traits at each vertex, leading to different trait clusters.

fish strategies is necessarily more tentative, given that fishes are motile and not all territorial and the starting assumption of our model is site-occupancy .

273   In disturbed environments, we predict evolution for higher b and lower d, but not higher c. Higher b means higher fecundity, but not necessarily mass propagule production: b represents only those propagules which sucessfully develop into juveniles in unoccupied territories. This is

276 broadly consistent with the ruderal primary strategy. Plant ruderals devote a large proportion of their productivity to seed production Grime (1977), whereas the analogous "opportunistic" strategists in fishes have large intrinsic growth rates (Winemiller and Rose, 1992). In corals, a dis-

279 tinguishing feature of the ruderal cluster is brood spawning (rather than broadcast spawning). This corresponds to higher parental investment and lower overall propagule production, but potentially also higher b at low densities, since broadcast spawners are vulnerable to a powerful

282 Allee effect at the egg fertilization stage (Knowlton, 2001). Lower d could be achieved by improved individual resistance to physical destruction, but it is hard to reduce mortality in the face of severe disturbances. Given this constraint, shortening the time to reproductive maturity (the

285 iteration time in our model) is an effective way of reducing the chance of death per iteration d. An exceptionally short life cycle is probably the most defining characteristic of ruderals (Darling et al., 2012; Grime, 1977; Winemiller and Rose, 1992).

288   In stressful environments, we predict evolution for lower d, and also for higher c in the low-T interpretation of the stress archetype. Lowering d is obviously essential when $b \ll 1$, and stress tolerant plants and corals have long life spans, allowing for long intervals between successful

291 recruitments (and episodic broadcast spawning in corals). For fishes, the "equilibrium" strategy is the analogue of Grime's stress tolerator. This strategy is associated with resource limitation, and is also characterized by long life span, as well as high parental investment in tiny broods.

294 This may reflect a high-c strategy in the face of intense competition for severely limited resources (the low-T interpretation).

    In ideal environments, we predict evolution for higher b and c, but not lower d. In plants and

297 corals, a key mechanism for winning territorial contests is rapidly outgrowing and "shading out"

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competitors; not surprisingly, rapid individual growth is a defining feature of the competitor trait cluster (Darling et al., 2012; Grime, 1977). Evolution for higher b under high-density, competitive conditions may seem counter-intuitive. Neither particularly high nor low b have been associated with the competitor strategy in plants and corals. However, for fishes, the analogous "periodic" strategy is characterized by enormous brood sizes as well as rapid development (Winemiller et al., 2015; Winemiller and Rose, 1992), suggesting a strategy of ensuring that many propagules actually end up contesting areas favorable for development (higher b). The evolution of b in ideal environments will be discussed further in the Discussion.

Discussion

Unlike Grime's classic ternary plot (Grime, 1974), which represents anti-correlations between traits relevant for success in different environmental archetypes, our realization of Grime's triangle (Fig. 4) refers instead to the direction of trait evolution under different regimes of stress and disturbance. As discussed in section "Primary strategies and Grime's triangle", over time our predicted trait evolution should lead to trait values consistent with Grime's scheme. In making these predictions, we have made no reference to any kind of trade-offs or pleitropy, even though trade-offs are often invoked to explain primary strategy schemes (Aerts, 1999; MacArthur, 1962; Winemiller and Rose, 1992). Thus, while trade-offs may amplify specialization, they are not necessary for it. For example, corals which rapidly out-shade neighbors have a tall, branched morphology which is vulnerable to disturbances, and so, all else being equal, ideal environment c-strategists will suffer higher mortality from disturbances. Fig. 4 gives the same conclusion without invoking trade-offs; mutations which reduce disturbance vulnerability are essentially neutral under ideal conditions, leading to no improvements in mortality from disturbances, whereas c will tend to increase over time.

Our prediction of evolution for higher b in ideal environments is counter to the expectations of MacArthur's r/K dichotomy (MacArthur, 1962) since b is closely related to the maximal,

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low-density growth rate $b - d$, and ideal environments support high population densities which should be subject to "K-selection". However, in the Introduction, we noted that the r-K dichotomy is not consistent with empirical studies showing that maximal growth rate and saturation density (measured by abundance) are positively correlated, both between species/strains (Fitzsimmons et al., 2010; Hendriks et al., 2005; Kuno, 1991; Luckinbill, 1979), and as a result of experimental evolution (Luckinbill, 1978, 1979). From the perspective of our model, these correlations are not surprising since the saturation density, which is determined by a balance between births and deaths, increases with b. Our higher-b prediction simply reflects the fact that, all else being equal, producing more propagules is always advantageous, regardless of population density, a fact lost in the simple logistic interpretation of the r/K scheme.

Confusingly, the term "K-selection" has sometimes been used to refer generally to selection at high density; this encompasses both selection for higher saturation density — "K" in the logistic equation — as well as selection for competitive ability. To avoid this ambiguity, the latter form of selection has been called "α-selection" after the competition coefficients in the Lotka-Volterra equation (Case and Gilpin, 1974; Gill, 1974; Joshi et al., 2001). Unlike saturation density, there is support for a negative relationship between competitive success at high densities and maximal growth rate Luckinbill (1979); this could be driven by a tradeoff between individual size and reproductive rate. However, competitive success as measured by α (i.e. the per-capita effect of one genotype on another genotype's growth rate) is only partly determined by individual competitive ability — in the presence of age-structure and territoriality, it also includes the ability of each genotype to produce contestants i.e. b in our model. In contrast, our c is strictly competitive ability only — as such, it does not directly affect population density (section "Model").

K-selection in the sense of selection for a greater environmental carrying capacity for given birth and death rates, sometimes referred to as "efficiency" (MacArthur, 1962), would be represented in our model by smaller individual territorial requirements. To a first approximation, two co-occurring genotypes which differ by a small amount in their territorial requirements only should have the same fitness since the costs or benefits of a change in the amount of unoccupied

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territory is shared equally among genotypes via the propagule density L. The situation is more complicated if those genotypes differ in multiple traits, and when the differences in territorial requirements become large enough that territorial contests can occur on different scales. We leave these complications for future work.

The importance of b in securing territories is a general feature of lottery competition. Indeed, as can be seen from Eq. (4), in the classic lottery model b_i and c_i are essentially equivalent in that only the products $b_i c_i$ matter (Chesson and Warner, 1981). This is no longer the case in our density-dependent generalization of the classic lottery model, where stable co-existence is possible between b and c strategists. Given that the classic lottery model was specifically developed for studying species co-existence questions, this may seem surprising, but the focus in that case was on the role of environmental fluctuations in promoting co-existence rather than stable coexistence (Chesson and Warner, 1981). It is not clear whether correctly accounting for the behavior of species with low density would significantly alter the conclusions of Chesson and Warner (1981), but, in any case, species co-existence questions are beyond the scope of this manuscript.

More broadly, while our model can be applied to questions at a species level (e.g. ecological invasions), our focus here is on the evolution of genotype frequencies within a population. This is only possible because the model accounts for the growth of mutants from low densities. Given this focus, our assumption that there are no large c discrepancies (section "Mean field approximation") amounts to a restriction on the amount of genetic variation in c that will be sustained in the population. Since beneficial mutation effect sizes will typically not be much larger than a few percent, large c discrepancies can only arise if the mutation rate is extremely large, and so the assumption will not be violated in most cases. However, this restriction could become important when looking at species interactions rather than genotype evolution.

In our view, the density dependent lottery model has two major limitations as a general-purpose model of density-dependent selection: a reliance on interference competition for durable resources (territoriality), and the restriction of competition to juveniles (lottery recruitment to

adulthood). In some respects this is the complement of resource competition models, which restrict their attention to exploitation competition, typically without age structure (Tilman, 1982). In the particular case that resources are spatially localized (e.g. due to restricted movement through soils), then resource competition and territorial acquisition effectively coincide, and in principle a competitive ability c should be derivable from resource competition. The situation is more complicated if the resources are well-mixed, since, in general, resource levels then need to be explicitly tracked. It is possible that explicit resource tracking is in fact not necessary when the focus is on the evolution of similar genotypes rather than the stable co-existence of widely differing species. We are not aware of any attempts to delineate the conditions under which explicit resource tracking is not required, let alone to connect those results to the density-dependent selection literature [need to check Tilman's book].

On the other hand, the model does remarkably well in capturing apparently general features of selection under different environmental conditions using only three trait parameters: b, c and d. The clean separation of a strictly-relative c parameter is particularly useful from an evolutionary genetics perspective, essentially embedding a relative fitness trait within an absolute fitness model. This could have interesting applications for modeling the impacts of intra-specific competition on species extinction, without arbitrarily assigning an absolute fitness cost to that competition. [citations to sexual selection stuff?]

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Appendix A: Poisson approximation

459 The propagule numbers x_i in different territories are not independent random variables. To determine the dispersal outcomes in all unoccupied territories exactly, we would need to proceed territory-by-territory as follows. In the first territory we evaluate, x_i drawn from a binomial
462 distribution with m_i trials and success probability $1/U$. In the second, x_i is drawn from a binomial distribution with $m_i - x$ trials and success probability $1/(U - 1)$, where x is the number of propagules that landed in the first territory. And so on.

465   For sufficiently large K, holding n_i/T fixed, the Poisson limit theorem implies that the binomial distributions for x_i at each successive stage of this procedure are all closely approximated by a Poisson distribution with mean l_i, where we have used the fact that large T implies large U
468 except in the biologically uninteresting case that there is vanishing population turnover $d_i \sim 1/T$.

    Under the Poisson approximation, the total number of genotype i propagules $\sum x_i$ (summed over unoccupied territories) will deviate about its mean value m_i. Since the coefficient of variation

of $\sum x_i$ is proportional to $1/\sqrt{m_i}$, these deviations are negligible unless m_i is very small (say of order 10^2 or less).

Appendix B: Derivation of growth equation

We separate the right hand side of Eq. (1) into three components $\Delta_+ n_i = \Delta_u n_i + \Delta_r n_i + \Delta_a n_i$ which vary in relative magnitude depending on the propagule densities l_i. Following the notation in the main text, the Poisson distributions for the x_i (or some subset of the x_i) will be denoted p, and we use P as a general shorthand for the probability of particular outcomes.

Growth without competition

The first component, $\Delta_u n_i$, accounts for territories where only one focal propagule is present $x_i = 1$ and $x_j = 0$ for $j \neq i$ (u stands for "uncontested"). The proportion of territories where this occurs is $l_i e^{-L}$, and so

$$\Delta_u n_i = U l_i e^{-L} = m_i e^{-L}. \tag{11}$$

Competition when rare

The second component, $\Delta_r n_i$, accounts for territories where a single focal genotype propagule is present along with at least one non-focal propagule (r stands for "rare") i.e. $x_i = 1$ and $\sum_{j \neq i} x_j \geq 1$. The number of territories where this occurs is $U p_i(1) P(\sum_{j \neq i} x_j \geq 1) = b_i n_i e^{-l_i}(1 - e^{-(L-l_i)})$. Thus

$$\Delta_r n_i = m_i e^{-l_i}(1 - e^{-(L-l_i)}) \left\langle \frac{c_i}{c_i + \sum_{j \neq i} c_j x_j} \right\rangle_{\tilde{p}}, \tag{12}$$

where $\langle \rangle_{\tilde{p}}$ denotes the expectation with respect to $\tilde{p}$, and $\tilde{p}$ is the probability distribution of nonfocal propagule abundances x_j *after* dispersal, in those territories where exactly one focal propagule, and at least one non-focal propagule, landed.

We now show that, with respect to $\tilde{p}$, the standard deviation $\sigma_{\tilde{p}}(\sum_{j \neq i} c_j x_j)$, is much smaller

than its mean $\langle \sum_{j\neq i} c_j x_j \rangle_{\tilde{p}}$. Then x_j can be replaced by its mean in the last term in Eq. (12),

$$\left\langle \frac{c_i}{c_i + \sum_{j\neq i} c_j x_j} \right\rangle_{\tilde{p}} \approx \frac{c_i}{c_i + \sum_{j\neq i} c_j \langle x_j \rangle_{\tilde{p}}}. \tag{13}$$

The exact expression for $\langle x_j \rangle_{\tilde{p}}$ is somewhat complicated. Let k denote the total number of propagules in a territory, and $\mathbf{x}_i = (x_1, \ldots, x_{i-1}, x_{i+1} \ldots, x_G)$ denote the vector of non-focal abundances, so that $p(\mathbf{x}_i) = p_1(x_1) \ldots p_{i-1}(x_{i-1}) p_{i+1}(x_{i+1}) \ldots p_G(x_G)$. Then, applying Bayes' theorem, $\tilde{p}$ can be written as

$$
\begin{aligned}
\tilde{p}(\mathbf{x}_i) &= p(\mathbf{x}_i | k \geq 2, x_i = 1), \\
&= \frac{P(\mathbf{x}_i, k \geq 2 | x_i = 1)}{P(k \geq 2)}, \\
&= \frac{1}{1 - (1+L)e^{-L}} \sum_{k=2}^{\infty} P(k) p(\mathbf{x}_i | \sum_{j\neq i} x_j = k-1), \\
&= \frac{1}{1 - (1+L)e^{-L}} \sum_{k=2}^{\infty} \frac{P(k) \delta_{k-1}^{\sum_{j\neq i} x_j}}{P(\sum_{j\neq i} x_j = k-1)} p(\mathbf{x}_i),
\end{aligned} \tag{14}
$$

492 where $P(k) = L^k e^{-L}/k!$, $P(\sum_{j\neq i} x_j = k-1) = (L-l_i)^{k-1} e^{-(L-l_i)}/(k-1)!$ and $\delta_k^{\sum_{j\neq i} x_j} = 1$ if $\sum_{j\neq i} x_j = k$, and equals zero otherwise. This implies $P(k)/P(\sum_{j\neq i} x_j = k-1) = \frac{Le^{-l_i}}{k} \left(\frac{L}{L-l_i} \right)^{k-1}$, and so, changing the summation index to start from one, we obtain

$$\tilde{p}(\mathbf{x}_i) = \frac{Le^{-l_i}}{1 - (1+L)e^{-L}} \sum_{k=1}^{\infty} \left(\frac{L}{L-l_i} \right)^k \frac{\delta_k^{\sum_{j\neq i} x_j}}{k+1} p(\mathbf{x}_i). \tag{15}$$

To evaluate $\langle x_j \rangle_{\tilde{p}} = \sum_{\mathbf{x}_i} \tilde{p}(\mathbf{x}_i) x_j$, we first evaluate the sum $\sum_{\mathbf{x}_i} \delta_k^{\sum_{j\neq i} x_j} p(\mathbf{x}_i) x_j$, since the only term in Eq. (15) which depends on $\mathbf{x}_i$ is $\delta_k^{\sum_{j\neq i} x_j} p(\mathbf{x}_i)$. By inspection, this sum is equal to the mean number of j propagules in a territory which received k nonfocal propagules, times the probability that a territory receives k nonfocal propagules. Given that k nonfocal propagules are present, on average genotype j will make up $\frac{l_j}{L-l_j} k$ of them, and so

$$
\begin{aligned}
\sum_{\mathbf{x}_i} \delta_k^{\sum_{j\neq i} x_j} p(\mathbf{x}_i) x_j &= \frac{l_j}{L-l_j} k \frac{(L-l_i)^k e^{-(L-l_i)}}{k!} \\
&= l_j \frac{(L-l_i)^{k-1} e^{-(L-l_i)}}{(k-1)!}.
\end{aligned} \tag{16}
$$

25

The final step is to evaluate the sum over k,

$$
\begin{aligned}
\langle x_j \rangle_{\tilde{p}} &= \frac{l_j L e^{-l_i}}{1 - (1+L)e^{-L}} \sum_{k=1}^{\infty} \left(\frac{L}{L - l_i} \right)^k \frac{(L - l_i)^{k-1} e^{-(L-l_i)}}{(k+1)(k-1)!}. \\
&= \frac{l_j L e^{-l_i}}{1 - (1+L)e^{-L}} \frac{1}{L - l_i} \sum_{k=1}^{\infty} \frac{L^k e^{-(L-l_i)}}{(k+1)!} k. \\
&= \frac{l_j}{1 - (1+L)e^{-L}} \frac{1}{L - l_i} \sum_{k=1}^{\infty} \frac{L^{k+1} e^{-L}}{(k+1)!} k. \\
&= \frac{l_j}{1 - (1+L)e^{-L}} \frac{L - 1 + e^{-L}}{L - l_i},
\end{aligned}
\tag{17}
$$

where the last line follows from the fact that $\sum_{k=1}^{\infty} \frac{L^{k+1} e^{-L}}{(k+1)!} k = \sum_{k=1}^{\infty} \frac{L^k e^{-L}}{k!} (k-1) = L - (1 - e^{-L})$.

For analyzing the relative fluctuations in $\sum_{j \neq i} c_j x_j$, Eq. (17) is unnecessarily complicated. We instead use the following approximation. Rather than evaluating the situation in each territory after dispersal as above, we replace $\tilde{p}$ by $\tilde{q}$, defined as the $\mathbf{x}_i$ dispersal probabilities, conditional on $\sum_{j \neq i} x_j > 1$, in a territory where one focal propagule is assumed to already be present. This gives $\langle x_j \rangle_{\tilde{q}} = l_j / C$,

$$
\sigma_{\tilde{q}}^2(x_j) = \frac{l_j^2}{C} \left(1 - \frac{1}{C} \right) + \frac{l_j}{C},
\tag{18}
$$

and

$$
\sigma_{\tilde{q}}(x_j, x_k) = \frac{l_j l_k}{C} \left(1 - \frac{1}{C} \right),
\tag{19}
$$

where $C = 1 - e^{-(L-l_i)}$. Comparing $\langle x_j \rangle_{\tilde{q}} = l_j / C$ and Eq. (17), the subtle difference in how we defined $\tilde{q}$ leads to considerably simpler expressions for the mean of x_j (similarly for variances and covariances of the x_j, which we did not have the courage to calculate with $\tilde{p}$). To be sure, $\tilde{q}$ is incorrect: assuming that one propagule is already present biases total number of propagules by one. For example, if the focal genotype is rare and the propagule density is high ($l_j \approx L \gg 1$), Eq. (17) correctly gives $\langle x_j \rangle_{\tilde{p}} \approx L - 1$, whereas $\langle x_j \rangle_{\tilde{q}} \approx L$. As a result, $\tilde{q}$ leads to pathological behavior for rare invaders (they have a rarity disadvantage), but its moments are quantitatively similar enough to $\tilde{p}$'s that it is sufficient for analyzing the relative fluctuations in $\sum_{j \neq i} c_j x_j$.

Decomposing the variance in $\sum_{j \neq i} c_j x_j$,

$$
\sigma_{\tilde{q}}^2 \left(\sum_{j \neq i} c_j x_j \right) = \sum_{j \neq i} \left[c_j^2 \sigma_{\tilde{q}}^2(x_j) + 2 \sum_{k > j} c_j c_k \sigma_{\tilde{q}}(x_j, x_k) \right],
\tag{20}
$$

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and using the fact that $1/C > 1$ which implies that the first term in Eq. (18) and $\sigma_{\tilde{q}}(x_j, x_k)$ are negative, the relative fluctuations in $\sum_{j \neq i} c_j x_j$ satisfy

$$\frac{\sigma(\sum_{j \neq i} c_j x_j)}{\langle \sum_{j \neq i} c_j x_j \rangle} < C^{1/2} \frac{\left(\sum_{j \neq i} c_j^2 l_j\right)^{1/2}}{\sum_{j \neq i} c_j l_j}. \tag{21}$$

513   Without loss of generality, we restrict attention to the case that the total nonfocal density $L - l_i$ is of order 1 or larger (otherwise $\Delta_r n_i$ does not contribute significantly to $\Delta_+ n_i$ because $\Delta_r n_i$ is proportional to $C = 1 - e^{-(L-l_i)}$).

516   When at least some of the nonfocal propagule densities are large $l_j \gg 1$, then the RHS of Eq. (21) is $\ll 1$, as desired. This is also the case if none of the nonfocal genotype densities are large and the c_j are all of similar magnitude (their ratios are of order one); the worst case

519   scenario occurs when $(L - l_i) \sim O(1)$, in which case the negative covariances (Eq. (19)) which were neglected in the RHS of Eq. (21) significantly reduce the overall variance $\sigma_{\tilde{q}}^2(\sum_{j \neq i} c_j x_j)$.

However, the relative fluctuations in $\sum_{j \neq i} c_j x_j$ can be large if some of the c_j are much larger

522   than the others. Specifically, if $c_j l_j \gg c_k l_k$ ($j, k \neq i$; $j \neq k$) and $l_j \ll 1$ (i.e. in the presence of a rare, extremely strong competitor), then we cannot make the replacement Eq. (13).

Substituting Eqs. (13) and (17) into Eq. (12), we obtain

$$\Delta_r n_i \approx m_i R_i \frac{c_i}{\bar{c}}, \tag{22}$$

525   where R_i is defined in Eq. (6).

Competition when abundant

The final contribution, $\Delta_a n_i$, accounts for territories where two or more focal propagules are

528   present (a stands for "abundant"). Similarly to Eq. (12), we have

$$\Delta_a n_i = U(1 - (1 + l_i)e^{l_i}) \left\langle \frac{c_i x_i}{\sum_j c_j x_j} \right\rangle_{\hat{p}} \tag{23}$$

where $\hat{p}$ is the probability distribution of both focal and nonfocal propagaule abundances *after* dispersal in those territories where at least two focal propagules landed.

27

Again, we show that the relative fluctuations in $\sum c_j x_j$ are much smaller than 1 (with respect to $\hat{p}$), so that we have

$$\left\langle \frac{c_i x_i}{\sum_j c_j x_j} \right\rangle_{\hat{p}} \approx \frac{c_i \langle x_i \rangle_{\hat{p}}}{\sum_j c_j \langle x_j \rangle_{\hat{p}}}. \tag{24}$$

Following a similar procedure as for $\Delta_r n_i$, where the vector of propagule abundances is denoted $\mathbf{x}$, we have

$$\begin{aligned}
\langle x_j \rangle_{\hat{p}} &= \sum_{\mathbf{x}} x_j p(\mathbf{x} | x_i \geq 2) \\
&= \sum_k P(k | x_i \geq 2) \sum_{\mathbf{x}} x_j p(\mathbf{x} | x_i \geq 2, k) \\
&= \sum_k P(k | x_i \geq 2) \sum_{x_i} \sum_{x_i} x_j p(\mathbf{x}_i | \sum_{j \neq i} x_j = k - x_i) p(x_i | x_i \geq 2, k) \\
&= \sum_k P(k | x_i \geq 2) \sum_{x_i} \frac{l_j(k - x_i)}{L - l_j} p(x_i | x_i \geq 2, k) \\
&= \frac{l_j}{L - l_j} \left(L \frac{1 - e^{-L}}{1 - (1 + L)e^{-L}} - l_j \frac{1 - e^{-l_j}}{1 - (1 + l_j)e^{-l_j}} \right)
\end{aligned} \tag{25}$$

for $j \neq i$, and

$$\langle x_i \rangle_{\hat{p}} = l_i \frac{1 - e^{-l_i}}{1 - (1 + l_i)e^{-l_i}}. \tag{26}$$

To calculate the relative fluctuations in $\sum_{j \neq i} c_j x_j$, we use a similar approximation as for $\Delta_r n_i$: $\hat{p}$ is approximated by the $\mathbf{x}$ dispersal probabilities in a territory where at least two focal propagule is assumed to be present. All covariances are now zero, so that $\sigma^2(\sum c_j x_j) = \sum c_j^2 \sigma^2(x_j)$, where $\sigma^2(x_j) = l_j$ for $j \neq i$. The expression for $\sigma^2(x_i)$ is more complicated. We assume $p(x_i = 0) \approx 0$ without loss of generality (since otherwise $D \gg 1$ and Δn_a is negligible). Then

$$\sigma^2(x_i) = \frac{l_i^2}{D}\left(1 - \frac{1}{D}\right) + \frac{l_i}{D}, \tag{27}$$

where $D = 1 - (1 + l_i)e^{-l_i}$, analogous to Eq. (18), and

$$\frac{\sigma(\sum c_j x_j)}{\langle \sum c_j x_j \rangle} \approx \frac{\left(\sum_{j \neq i} c_j^2 l_j + c_i^2 \sigma^2(x_i)\right)^{1/2}}{\sum_{j \neq i} c_j l_j + c_i l_i / D}. \tag{28}$$

Similarly to Eq. (21), the RHS of (28) will not be $\ll 1$ if there is a nonfocal genotype j with $l_j \ll 1$ and $c_j l_j \gg c_k l_k$ for $j, k \neq i$, $j \neq k$. When this is not the case, then since l_i must be of order

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1 or larger for $\Delta_a n$ to make an appreciable contribution to $\Delta_+ n_i$, the RHS of Eq. (28) is $\ll 1$ as desired.

Combining Eqs. (23) and (24), we obtain

$$\Delta_a n_i = m_i A_i \frac{c_i}{\overline{c}},$$

(29)

where A_i is defined in Eq. (7).