

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

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Abstract

Fitness is typically represented in heavily simplified terms in evolutionary genetics, often using
3 constant selection coefficients. This excludes fundamental ecological factors such as dynamic
population size or density-dependence from our most genetically-realistic treatments of evolu-
tion, a problem that inspired MacArthur’s influential but problematic r/K theory. Following
6 in the spirit of r/K -selection as a general-purpose theory of density-dependent selection, but
grounding ourselves empirically in “primary strategy” trait classification schemes like Grime’s
triangle, we develop a new model of density-dependent selection which revolves around terri-
9 torial contests. To do so, we generalize the classic lottery model of territorial acquisition, which
has primarily been used for studying species co-existence questions, to accommodate arbitrary
densities. We use this density-dependent lottery model to predict the direction of trait evolution
12 under different environmental conditions and thereby provide a mathematical underpinning for
Grime’s verbal scheme. We revisit previous concepts of density-dependent selection, including r
and K selection, and argue that our model distinguishes between different aspects of fitness in a
15 more natural and intuitive manner.

“...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, the resulting picture of evolution does not include even basic elements of the ecological underpinnings of selection, including dynamic population size and density-dependence.

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 generalize beyond particular traits or ecological scenarios to models of fitness that still capture 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). For example, MacArthur’s famous r/K scheme (MacArthur, 1962; MacArthur and Wilson, 1967) is now almost exclusively known as

a framework for understanding life-history traits, and judged on its failure in that role (Boyce,
42 1984; Pianka, 1970; Reznick et al., 2002; Stearns, 1977). The r/K scheme's original purpose was
as an extension of the existing population-genetic treatment of selection to account for popu-
lation density (MacArthur, 1962), but few attempts have been made to develop it further as a
45 mathematical analysis of the major different forms of selection.

Nevertheless, there are strong indications there exist broader principles governing the oper-
ation of selection. In many groups of organisms, including corals (Darling et al., 2012), insects
48 (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).
51 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 de-
54 struction 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
(Fig. 1): competitors "C" excel in low stress, low disturbance environments; stress tolerators
57 "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 envi-
ronments. Grime showed that measures of C, S and R across a wide range of plant species are
60 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).

63 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 reproductive rate or stress-tolerance. However, while trait classification
66 schemes are firmly grounded in trait data, they are verbal and descriptive rather than mathemat-
ical, a recognized hinderance to their broader applicability (e.g. Tilman 2007).



Figure 1: Schematic of Grime’s triangle. The two axes show increasing levels of environmental stress and disturbance, respectively. Survival is not possible if the combination of stress and disturbance is too large (dashed line). This creates a triangle, each corner of which corresponds to a “primary strategy”.

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 more with Grime’s $C/S/R$ scheme in mind, and represents a quantitative formalization of how $C/S/R$ manifests at the level of within-population genotype evolution (as opposed to phenotypic divergence between species). This choice is motivated in part by the substantial empirical support for $C/S/R$ -like schemes, 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).

As we will see, a generalized version of the classic lottery model of Chesson and Warner (1981) is a convenient starting point for ecologically-grounded models of selection in evolution-

ary genetics. In the classic lottery model, mature individuals (“adults”) each require their own territory, whereas newborn individuals (“propagules”) disperse to, and subsequently compete for, territories made available by the death of adults. Territorial contest among propagules leaves a single victorious adult per territory, the victor chosen at random from the propagules present (akin to a lottery; Sale 1977), with probabilities weighted by a coefficient for each type representing competitive ability. This representation of competition is much simpler than having coefficients for the pairwise effects of types on each other (e.g. the α coefficients in the generalized Lotka-Volterra equations), or than modeling resource consumption explicitly (Tilman, 1982). The classic lottery model is also closely connected to one of the central models of population genetics, the Wright-Fisher model of genetic drift.

However, the classic lottery model breaks down at low densities (that is, if there are only a few propagules dispersing to each territory; see “Model”). This was not a limitation in the lottery model’s original application to reef fishes, where a huge number of larvae from each species compete to secure territories each generation (Chesson and Warner, 1981), but is a critical limitation for studying density-dependent selection. We analytically extend the classic lottery model to correctly account for low density behavior.

In the 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. In the following two sections, we then discuss the behavior of rare mutants (invasion and coexistence) and our formalization of Grime’s triangle.

Model

We assume that the reproductively mature individuals in a population (“adults”) each require their own territory to survive and reproduce (Fig. 2). 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 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 new offspring ("propagules"), m_i of which disperse to unoccupied territories. We assume that adults cannot be ousted from occupied territories, and so only propagules landing on unoccupied territories are included in m_i . Propagules disperse globally, at random, and independently of each other; all propagules have the same probability of landing on any of the U unoccupied territories. Thus, there is no interaction between propagules (e.g. avoidance of territories crowded with propagules). Loss of propagules during dispersal is subsumed into m_i . In general, m_i will increase with n_i , and will also depend on population density N . For example, if b_i is the number of successfully dispersing propagules produced per genotype i adult, then the loss of propagules due to dispersal to occupied territories implies $m_i = b_i(1 - N/T)n_i$, akin to Levins' competition-colonization model (Levins and Culver, 1971; Tilman, 1994). Here we assume $m_i = b_i n_i$, where b_i is a constant, meaning that all propagules land on unoccupied territories (a form of directed dispersal). This choice simplifies the mathematics without seriously restricting the generality of our analysis, since the results presented here are not sensitive to the specific functional form of m_i . Note that due to our assumption of global dispersal, the parameter b_i can be thought of as a measure of "colonization ability", which combines fecundity and dispersal ability (Bolker and Pacala, 1999; Levins and Culver, 1971; Tilman, 1994).

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 $T \rightarrow \infty$, with n_i/T held fixed, x_i is Poisson distributed with mean territorial propagule density $l_i = m_i/U$ (this dispersal Poisson distribution is denoted $p_i(x_i) = l_i^{x_i} e^{-l_i} / x_i!$). Although T is finite in our model, we assume that T and the n_i are large enough that x_i is Poisson-distributed to a good approximation (details in Appendix A). 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.

When multiple propagules land on the same territory, they compete to secure the territory



Figure 2: Each iteration of our lottery model has three main elements. First, propagules are produced by adults which are dispersed at random over the unoccupied territories (only propagules landing on unoccupied territories are shown). Lottery competition then occurs in each unoccupied territory (competition in only one territory is illustrated): each genotype has a probability proportional to $b_i n_i c_i$ of securing the territory. Then occupied territories are freed up by adult mortality. In Eq. (3) and most of the paper, only adults can die (red crosses), but we will also consider the case where juveniles die (blue cross; section “Primary strategies and Grime’s triangle”).

as they develop. This territorial contest is modeled as a weighted lottery: the probability that
 138 genotype i wins a territory by the next iteration, assuming that at least one of its propagules is
 present, is $c_i x_i / \sum_j c_j x_j$, where c_i is a constant representing relative competitive ability (Fig. 2).

In the classic lottery model (Chesson and Warner, 1981), unoccupied territories are assumed
 141 to be saturated with propagules from every genotype i.e. $l_i \gg 1$ where $l_i = m_i/U$ is the mean
 propagule density. From the law of large numbers, the composition of propagules in each terri-
 tory will then not deviate appreciably from the mean composition l_1, l_2, \dots, l_G (G is the number
 144 of genotypes present), and so the probability that genotype i wins any particular unoccupied
 territory is approximately $c_i l_i / \sum_j c_j l_j$. Let $\Delta_+ n_i$ denote the number of territories won by geno-
 type i . Then $\Delta_+ n_1, \Delta_+ n_2, \dots, \Delta_+ n_G$ follow a multinomial distribution with U trials and success
 147 probabilities $\frac{c_1 l_1}{\sum_j c_j l_j}, \frac{c_2 l_2}{\sum_j c_j l_j}, \dots, \frac{c_G l_G}{\sum_j c_j l_j}$, respectively. Genotype i is expected to win $c_i l_i / \sum_j c_j l_j$ of the
 U available territories, and deviations from this expected outcome are small (since T is large by
 assumption), giving

$$\Delta_+ n_i(t) = \frac{c_i l_i}{\sum_j c_j l_j} U(t) = b_i n_i \frac{1}{L} \frac{c_i}{\bar{c}}, \quad (1)$$

150 where $\bar{c} = \sum_j c_j m_j / M$ is the mean propagule competitive ability for a randomly selected propag-
 ule, $L = M/U$ is the total propagule density and $M = \sum_j m_j$ is the total number of propagules.

There is a close connection between the classic lottery model and the Wright-Fisher model of
 153 genetic drift (Svardal et al., 2015). In the Wright-Fisher model, genotype abundances are sampled
 each generation from a multinomial distribution with success probabilities $w_i n_i / \sum_j w_j n_j$, where
 w is relative fitness and the n_i are genotype abundances in the preceding generation. Population
 156 size N remains constant. This is mathematically equivalent to the classic lottery model with
 non-overlapping generations ($d_i = 1$ for all i) and $w_i = b_i c_i$.

The main advantage of the classic lottery model for our purposes here is that the abstract
 159 population-genetic relative fitnesses w_i can be replaced with more ecologically-grounded fecun-
 dity, competitive ability and mortality parameters b_i , c_i and d_i . In particular, differences in indi-
 vidual longevity are only possible because generations are overlapping. However, note that the

classic lottery model, and the present work, ignore the stochastic drift in type frequencies (large T approximation) which is the focus of the Wright-Fisher model.

In our extension of the classic lottery model, we do not restrict ourselves to high propagule densities. Eq. (1) is nonsensical at low densities (if l_i is small): genotype i can win at most m_i territories, yet Eq. (1) demands $c_i l_i / \sum_j c_j l_j$ of the U unoccupied territories, for any value of U . Thus, at low densities, deviations from the mean propagule composition l_1, l_2, \dots, l_G are important.

Since dispersal is Poisson, we expect that a fraction $p_1(x_1) \dots p_G(x_G)$ of the U unoccupied territories will have the propagule composition x_1, \dots, x_G . Genotype i is expected to win $c_i x_i / \sum_j c_j x_j$ of these. Ignoring fluctuations about these two expectations (large T approximation), genotype i 's territorial acquisition is given by

$$\Delta_+ n_i(t) = U(t) \sum_{x_1, \dots, x_G} \frac{c_i x_i}{\sum_j c_j x_j} p_1(x_1) \dots p_G(x_G), \quad (2)$$

in our extended lottery model, where the sum only includes territories with at least one propagule present.

For the majority of this manuscript we assume that mortality only occurs in adults (setting aside the juvenile deaths implicit in territorial contest), and at a constant, genotype-specific per capita rate d_i , so that the overall change in genotype abundances is

$$\Delta n_i(t) = \Delta_+ n_i(t) - d_i n_i(t). \quad (3)$$

This is reasonable approximation in the absence of disturbances; when we come to consider the effects of disturbances (Section "Primary strategies and Grime's triangle"), we will incorporate disturbance-induced mortality in competing juveniles (Fig. 2).

Note that the competitive ability coefficients c_i represent a strictly relative aspect of fitness in the sense that they can only influence population size N indirectly by changing genotype frequencies. This can be seen by summing Eq. (3) over genotypes to get the change in population

size N ,

$$\Delta N = U(1 - e^{-L}) - \sum_i d_i n_i, \quad (4)$$

which is independent of c_i (here $L = \sum_j l_j$ is the overall propagule density). Unlike the classic
 186 lottery model, not all U unoccupied territories are claimed each iteration; a fraction e^{-L} remain
 unoccupied.

Results

189 Mean Field Approximation

Eq. (2) involves an expectation over the time-dependent dispersal distributions p_i , and is thus
 too complicated to give intuition about the dynamics of density-dependent lottery competition.
 192 We now evaluate this expectation using a “mean field” approximation.

Our approximation is similar to the high- l_i approximation behind the classic lottery model in
 that we replace the x_i with appropriate mean values. However, we cannot simply replace x_i with
 195 l_i as in the classic lottery model. For a genotype with a low propagule density $l_i \ll 1$, we have
 $x_i = 1$ in the few territories where its propagules land, and so its growth comes entirely from
 territories which deviate appreciably from l_i . In our more general approximation, territories with
 198 a single propagule from the focal genotype 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
 201 discussion in section “Discussion”). We obtain (details in Appendix B)

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

where

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

and

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

204 Comparing Eq. (5) to Eq. (1), 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, $R_i c_i/\bar{c}$ represents competitive victories when the i genotype is a rare invader in a high density population: from 207 Eq. (6), $R_i \rightarrow 0$ 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 210 is abundant: $A_i \rightarrow 0$ 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 m_i/M . 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 $A_i \rightarrow 1/L$). 213 Thus, Eq. (5) generalizes the classic lottery model to account for arbitrary propagule densities for each genotype.

Fig. 3 shows that Eq. (5) (and its components) closely approximate direct simulations of 216 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 219 $R_i c_i/\bar{c}$, 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. 3b). 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. 3d). Fig. 3 also shows the breakdown 222 of the classic lottery model at low densities (low l_1 and l_2).

Invasion of rare genotypes and coexistence

In our model (section “Model”), each genotype is defined by three traits: b , c and d . To determine 225 how these will evolve in a population where they are being modified by mutations, we need to know whether mutant lineages will grow (or decline) starting from low densities. In this section

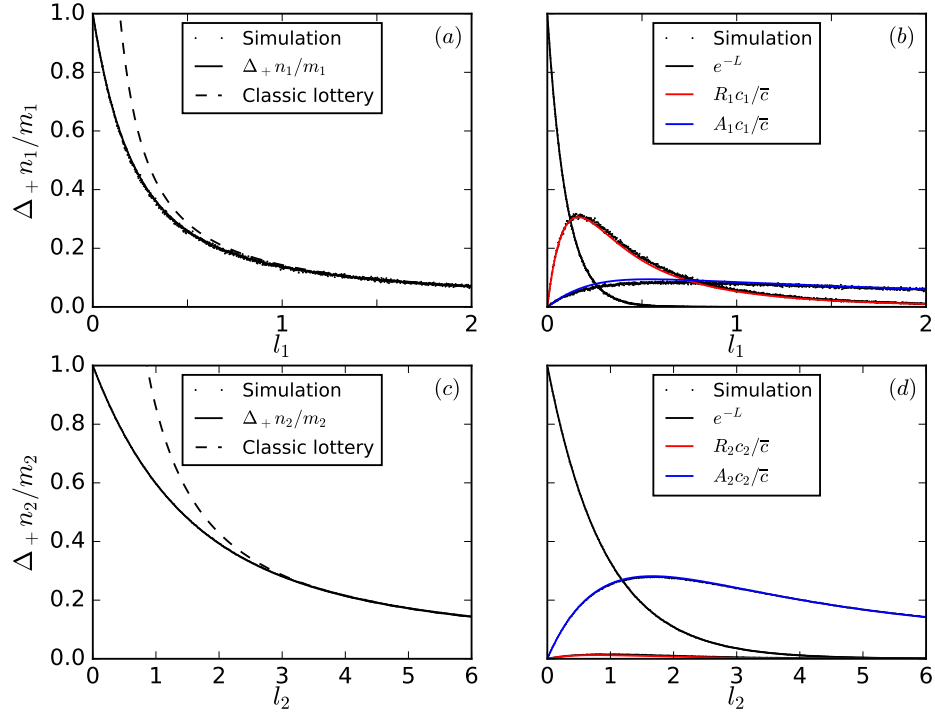


Figure 3: The change in genotype abundances in a density dependent lottery model is closely approximated by Eq. (5). $\Delta + n_i/m_i$ from Eq. (5) (and its separate components) are shown, along with direct simulations of random dispersal and lottery competition over one iteration over a range of propagule densities (varied by changing U with the m_i fixed). Two genotypes are present. (a) and (b) show the low-frequency genotype with c -advantage ($m_1/M = 0.1, c_1 = 1.5$), (c) and (d) show the high-frequency predominant genotype ($m_2/M = 0.9, c_2 = 1$). Simulation points are almost invisible in (c) and (d) due to near exact agreement with Eq. (5). Dashed lines in (a) and (c) show the corresponding classic lottery model predictions.

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

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

where $A_i = (1 - (1 + L)e^{-L})/L$. Now suppose that a new genotype j , which is initially rare, appears in the population. Then $A_j \ll R_j$, $l_j \approx 0$ and $\bar{c} \approx c_i$, and so, from Eq. (5), n_j will increase if

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

where $R_j \approx (1 - e^{-L}) / \left(\frac{c_j}{c_i} + \frac{L-1-e^{-L}}{1-(1+L)e^{-L}} \right)$.

Combining Eqs. (8) and (9), we see that j will invade if it is superior in any one of the three traits, but is otherwise identical to i . If the new genotype has the same competitive ability $c_j = c_i$, then $R_j \approx A_i$ and Eqs. (8) and (9) imply that invasion occurs when $b_j d_i - b_i d_j > 0$, and in particular when $b_j > b_i$ with $d_j = d_i$, or when $d_j < d_i$ with $b_j = b_i$. In the case that the new genotype has a different competitive ability but the same b_i and d_i , Eqs. (8) and (9) imply that invasion occurs when $R_j c_j / c_i > A_i$; it is not hard to verify that this occurs if and only if $c_j > c_i$ using the simplified expressions for A_i and R_j given after Eqs. (8) and (9) respectively. Moreover, if j invades in any of these cases, it will eventually exclude i , since it is strictly superior.

Stable coexistence is possible between genotypes that are superior in different traits. 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"). 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

$$b_j c_j R_j - b_i c_i A_i > 0. \quad (10)$$

Thus, coexistence occurs if c_j/c_i is large enough. This is a version of the classic competition-
 249 colonization trade-off (Levins and Culver, 1971; Tilman, 1994): the competitor (c -specialist) leaves
 many territories unoccupied (low L) due to its poor colonization ability (low b), which the colo-
 nizer (b -specialist) can then exploit. A similar argument applies for coexistence between high- c
 252 and low- d specialists; a “competition-longevity” trade-off (Tilman, 1994). Mutual invasibility is
 not possible between b - and d -specialists.

If the rare genotype j arises due to mutation, then its initial low-density behavior is more com-
 255 plicated 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
 258 (2) and (5) do not apply (section “Model”). 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 occurs when n_j is of order $1/s$ (Desai and Fisher, 2007),
 261 where the selection coefficient s is the mutant’s fitness advantage (i.e. $s = \frac{\Delta n_i/n_i}{\sum_i \Delta n_i/n_i \times n_i/N} - 1$).
 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
 264 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

267 We now discuss which changes in the traits b, c and d will be particularly favored under different
 environmental conditions. Of specific interest are Grime’s “disturbed”, “stressful” and “ideal”
 environments. To proceed, we need to map these verbally-defined environments to quantitative
 270 parameter regimes in our model.

The ideal environment is characterized by the near-absence of stress and disturbance. Con-
 sequently, $d_i \ll 1$, whereas b_i is potentially much larger than 1. From Eq. (4), the equi-
 273 librium 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 $L \approx b_i/d_i \gg 1$, and every unoccupied territory will be heavily contested. The population density is also high $N/T \approx 1$ (since
 276 $L = b_i N/(N - T) = b_i/(1 - T/N)$).

Disturbed environments are characterized by unavoidably high extrinsic mortality caused by physical destruction. Environmental variability, and disturbances in particular, can be modeled
 279 as stochastic fluctuations in b , c and d (Chesson and Warner, 1981). For simplicity, we do not pursue this more complicated stochastic approach. Instead, we represent disturbance by high constant mortality rates d_i , and extend this mortality to juveniles in the process of territorial contest (since disturbances do not only affect adults as in Eq. (3)). We assume that disturbances are
 282 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. [More justification?] Disturbed environments then correspond to d_i being close to 1 for all genotypes (almost all adults and juveniles are killed each
 285 iteration). From Eq. (4), the single genotype equilibrium is given by $L/(1 - e^{-L}) = d_i/[(1 - d_i)b_i]$, and since $L \ll 1$ and $N/T \ll 1$ due to high mortality, we have $L \approx 2(1 - d_i)/[(1 - d_i)b_i]$. Clearly b_i must be exceptionally large to ensure population persistence. The terms proportional to c_i/\bar{c}
 288 in Eq. (5) are then negligible, and $\Delta_+ n_i$ depends primarily on b_i .

Stressful environments are more ambiguous, and have been the subject of an extensive debate
 291 in the plant ecology literature (the “Grime-Tilman” debate; Aerts 1999 and references therein). Severe stress inhibits growth and reproduction, so that $b \ll 1$ Grime (1974, 1977). Mutations which appreciably improve b will be either non-existent or extremely unlikely, so b is constrained
 294 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 $b/d \approx 1$ in our model, and so the propagule density L is suppressed to such low levels that there are essentially
 297 no territorial contests occurring.

The alternative view is that, while stressful environments imply lower b and support a lower number of individuals per unit area compared what is attainable in ideal environments, stressed
 300 populations are actually at high densities relative to the environmental carrying capacity, and are

highly competitive (Taylor et al., 1990). In the particular case that stress is caused by scarcity of consumable resources, we might expect intense resource competition (for empirical support, see
303 Davis et al. 1998). Thus, b may actually appreciably exceed d under stressful conditions, even though the absolute value of b is small.

The mapping of different environments to our model parameters is summarized in the first
306 two rows of Fig. 4. Also shown is the approximate dependence of $\Delta_+ n_i$ on b_i and c_i for each environment (third row). These can be used infer the expected direction of evolution for the traits b , c and d (fourth row) as follows.

309 As noted in the previous section, if beneficial mutations survive the low-abundance stochastic regime, they proceed to grow deterministically according to Eq. (5). The probability of surviving to deterministic abundances increases with the mutant fitness advantage, and is therefore
312 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 are both available, and confer an appreciable benefit, where
315 availability is subject to constraints imposed by the environment.

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

321 Following Grime's original argument for a triangular scheme (Grime, 1977), Fig. 5 represents each environmental extreme schematically as a vertex on a triangular space defined by perpendicular stress and disturbance axes. The ideal environment lies at the origin (no stress or
324 disturbance), while the stressful and disturbed environments 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
327 evolutionary change is different at each vertex, leading to the emergence of different trait clusters

	Ideal	Disturbance*	Stress (G)	Stress (HD)
Constraints	$d \ll 1$	$d \approx 1$	$b \ll 1$	$b \ll 1$
Other parameters	$b \gg d$	$b \gg d$	$b \approx d$	$b > d$
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 4: The realization of Grime’s three environmental extremes in our model, as well as the high-density variant of the stressful environment. Shown are the mapping of each environment to our parameters, 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 disturbed environment, with $\Delta_+ n_i$ replaced by $(1 - d_i)\Delta_+ n_i$ in Eq. (3).

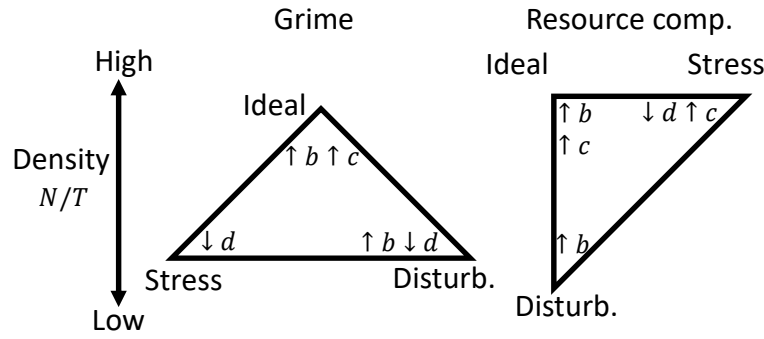


Figure 5: The realization of Grime’s triangle in our model. Schematic representation of the triangular space bounded by the low/high extremes of stress/disturbance. The low- T interpretation of stress is also shown. The vertices of the triangles correspond to different environmental extremes. Selection favors different traits at each vertex, leading to different trait clusters.

or “primary strategies”.

How does Fig. 4 compare to empirical analyses of 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 fish strategies is necessarily more tentative, given that fishes are motile and not all territorial.

In disturbed environments, we predict evolution for higher b and lower d , but not higher c . Higher b means higher fecundity and/or dispersal ability (see “Model”). This is consistent with a ruderal strategy. Plant ruderals devote a large proportion of their productivity to seed

production (Grime, 1977), whereas the analogous “opportunistic” fishes have large intrinsic growth rates (Winemiller and Rose, 1992). In corals, ruderals are distinguished by brood spawning (rather than broadcast spawning). This corresponds to higher parental investment and lower overall propagule production — counter-intuitively, a stereotypical “K-selected”, high-density trait Pianka (1970). However, since broadcast spawners are vulnerable to an Allee effect at the egg fertilization stage (Knowlton, 2001), brood spawning could actually be a way to ensure high b at low densities (Darling et al., 2012). 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. Alternatively, shortening the time to reproductive maturity (the iteration time in our model) is an effective way of reducing the chance of death per iteration, d , for a given frequency of disturbance. An exceptionally short life cycle is probably the most defining characteristic of ruderals (Darling et al., 2012; Grime, 1977; Winemiller and Rose, 1992). Note that if evolution manages to appreciably reduce d for a given disturbance intensity, then the population no longer lies at the extreme disturbance vertex of Grime’s triangle. Thus, ruderals are characterized by both high b and high d , but there is a constant pressure for a shorter life cycle, extending the limits of disturbance that can be tolerated.

In stressful environments, we predict evolution for lower d , and also for higher c in the high-density interpretation of stressful environments. Low d is essential when $b \ll 1$, and stress tolerant plants and corals have long life spans, allowing for long intervals between successful 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 consumable resource limitation, and is also characterized by long life span, as well as high parental investment in tiny broods. This may reflect a high- c strategy in the face of intense competition for severely limited resources (the high-density interpretation).

In ideal environments, we predict evolution for higher b and c , but not lower d . In plants and corals, a key mechanism for winning territorial contests (higher c) is rapidly outgrowing and “shading out” competitors; empirically, rapid individual growth is a defining feature of the

competitor trait cluster (Darling et al., 2012; Grime, 1977). The situation for b is more ambiguous. Competitor strategies in plants and corals span a range of b . For fishes, the analogous “periodic” strategy is characterized by enormous spawn sizes as well as rapid development (Winemiller et al., 2015; Winemiller and Rose, 1992). The role of b in ideal environments will be discussed further below.

Discussion

Adaptive evolution in the direction predicted by our model should produce traits consistent with Grime’s scheme (“Primary strategies and Grime’s triangle”), with the possible exception of evolution for higher b at high-densities. The latter is also counter to the expectations of MacArthur’s r/K dichotomy (MacArthur and Wilson, 1967), since b is closely related to the maximal, low-density growth rate $r = b - d$ (Pianka, 1972), and yet ideal environments support high population densities which should be subject to “ K -selection”.

Intuitively, it makes sense that b can matter at high densities. In our model (or any lottery model of competition), b matters at high densities because territorial contests among juveniles are intrinsically unpredictable. This is a realistic feature of the model. Even if one genotype is guaranteed to win a territory in a “fair” contest (e.g. it is the most efficient exploiter of a limiting consumable resource; Tilman 1982), inferior competitors can win by chance. For example, an inferior competitor’s propagules may happen to arrive first, gaining a decisive developmental head start. First arrivals are more likely to occur for genotypes with a fecundity and/or dispersal advantage, as represented by higher b in lottery models. The analogous intuition in terms of the Wright-Fisher model is that fecundity confers a relative fitness advantage, even though population size is not changing. The logistic model for which r and K are named, does not capture this intuition.

Confusingly, the term “ K -selection” has sometimes been used to refer generally to selection at high density (Pianka, 1972); this encompasses both selection for higher saturation density

— “ K ” in the logistic equation — as well as selection for competitive ability. Contrary to an
 390 r/K dichotomy, empirical studies have shown 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
 393 evolution (Luckinbill, 1978, 1979). From the perspective of our model, this correlation is not
 surprising since the saturation density, which is determined by a balance between births and
 deaths, increases with b .

396 Unlike saturation density, and consistent with an r/K dichotomy, there is support for a nega-
 tive relationship between competitive success at high densities and maximal growth rate (Luck-
 inbill, 1979); this could be driven by a tradeoff between individual size and reproductive rate.
 399 To avoid confusion with other forms of “ K -selection”, selection for competitive ability has been
 called “ α -selection” after the competition coefficients in the Lotka-Volterra equation (Case and
 Gilpin, 1974; Gill, 1974; Joshi et al., 2001). Competitive success as measured by α (i.e. the per-
 402 capita effect of one genotype on another genotype’s growth rate) is only partly determined by
 individual competitive ability — in the presence of age-structured competition and territoriality,
 it also includes the ability of each genotype to produce contestants i.e. b in our model. Our c is
 405 strictly competitive ability only — as such, changes in c do not directly affect population density
 (section “Model”), nor the ability to produce contestants.

K -selection in the narrow logistic sense of selection for a greater environmental carrying
 408 capacity for given birth and death rates, sometimes referred to as “efficiency” (MacArthur and
 Wilson, 1967), could 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
 411 territorial requirements only should have the same fitness since the costs or benefits of a change in
 the amount of unoccupied territory is shared equally among genotypes via the propagule density
 per territory L . The situation is more complicated when the differences in territorial requirements
 414 become large enough that territorial contests can occur on different scales for different genotypes.
 We leave these complications for future work.

In the classic lottery model, b_i and c_i are essentially equivalent in the sense that the number of territorial victories only depends on the product $b_i c_i$ (see “Model”). This is no longer the case in our density- and frequency-dependent generalization of the classic lottery model, where there is co-existence between b and c specialists. This trade-off is similar to the colonization-competition trade-off that is well known in the co-existence literature (Tilman, 1994). It and similar forms of “spatial co-existence” in stable environments have previously been modeled either with Levin’s qualitative representation of competition (Levins and Culver, 1971; Tilman, 1994), as opposed to the quantitative c of lottery competition, or with a more sophisticated treatment of space (non-uniform dispersal) (Bolker and Pacala, 1999; Shmida and Ellner, 1984). In fluctuating environments our model would likely give similar co-existence predictions as Chesson and Warner (1981), which revolve around the “storage effect”, since we retain overlapping generations and competition at the juvenile phase only.

While our model can be applied to questions at an inter-species level (e.g. ecological invasions), our focus here is on the evolution of genotype frequencies within a population. 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 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.

Our realization of Grime’s triangle (Fig. 4) differs from approaches which identify primary strategies as trait combinations which can co-exist (Bolker and Pacala, 1999). Instead, it refers to the direction of adaptive trait evolution under different regimes of stress and disturbance, which is closer in the spirit to Grime’s original arguments. In making our predictions, we have not assumed any kind of trade-offs or pleiotropy between b , c and d , only constraints imposed by the environment on the order of magnitude of b and d . Thus, while trade-offs may amplify specialization, and are sometimes invoked to explain primary strategy schemes (Aerts, 1999; MacArthur

and Wilson, 1967; Winemiller and Rose, 1992), they are not necessary for it. As an example of a
trade-off, 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 (Darling et al., 2012). Fig. 5 gives the same conclusion with-
out 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.

In Fig. 6 we compare our model with some of the other models and schemes touched upon
here. In a sense this comparison is unfair: for instance, Grime's scheme was developed for an
entirely different purpose (species classification by traits). As such, Fig. 6 is not exhaustive and
should be read more as a summary of our model's purpose. Like MacArthur's r/K scheme,
our model is motivated by the need to expand the treatment of selection in population genetics
(MacArthur, 1962), i.e. to incorporate crucial ecological factors in our most genetically realistic
models of evolution. Thus, viewing evolutionary ecology (Kokko and López-Sepulcre, 2007; Pel-
letier et al., 2009; Schoener, 2011) as a spectrum ranging from evolution-only to ecology-only, our
model lies close to the understudied evolution-only end of the spectrum. By comparison, more
familiar approaches to evolutionary ecology such as adaptive dynamics — essentially ecology
coupled with mutant invasion (Diekmann et al., 2004) — lie close to the ecology-only end of the
spectrum.

In our view, our 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 re-
spects 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 re-
source competition and territorial acquisition effectively coincide, and in principle resource com-
petition could be represented by a competitive ability c (or conversely, c should be derivable from

	Formal model?	Ecologically meaning- ful?	Empirically- grounded trait scheme?	Generality beyond specific scenarios?	Genetically flexible?
Density-dependent lottery	✓	✓	✓	✓	✓
MacArthur's $r/K + \alpha$	✓	✓	✗*	✓	✓
Grime's C/S/R	✗	✓	✓	✓	NA
Traditional pop. gen.	✓	✗	✗	✓	✓
Eco-evo:					
Adaptive dynamics	✓	✓	✓	✓**	✗
Brute force simulation	✓	✓	NA	✗	✓

Figure 6: Comparison of our density-dependent lottery model with related models and schemes in ecology and evolutionary biology. *MacArthur's r - and K -, as well as α selection, were all derived theoretically. Applications to traits came later, and with mixed success (see "Introduction" and "Discussion"). **In practice, most of the adaptive dynamics literature focuses on specific eco-evolutionary outcomes such as evolutionary "branching" (Geritz et al., 1997) or mutualisms (Ferriere et al., 2002), but in principle it can be applied with any fitness model including our density-dependent lottery.

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 seems plausible that explicit resource tracking is 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 conditions under which explicit resource tracking is unnecessary even if it is assumed that community structure is ultimately determined by competition for consumable resources. More work is needed connecting resource competition models to the density-dependent selection literature, since most of the existing work is focused on the narrower Grime-Tilman-debate issue of resolving the role of competition at different levels of resource availability (Aerts, 1999; Davis et al., 1998; Tilman, 2007).

On the other hand, our 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 zero-sum fitness trait within a non-zero-sum fitness model. This could have interesting applications for modeling the impacts of

intra-specific competition on species extinction, for example due to clonal interference (Desai
486 and Fisher, 2007; Gerrish and Lenski, 1998) between *c*-strategists on the one hand, and *b*- and *d*-
strategists on the other.

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Appendix A: Poisson approximation

588 For each genotype’s dispersal, the counts of propagules across unoccupied territories follows a multinomial distribution with equal probabilities of landing in each territory. Thus, the x_i in different territories are not independent random variables. However, for sufficiently large T ,
591 holding n_i/T fixed, the Poisson limit theorem implies that this multinomial distribution for the x_i accross territories is closely approximated by a product of independent Poisson distributions for each territory, each with rate parameter l_i (large T implies large U except in the biologically
594 uninteresting case that there is vanishing population turnover $d_i \sim 1/T$).

Alternatively, we could have assumed a Poisson distribution for the x_i as our model of dispersal from the outset. The total number of genotype i propagules $\sum x_i$ (summed over unoccupied
597 territories) would then no longer be a constant m_i , but would fluctuate between generations for

a given mean m_i , which is more biologically realistic. Nevertheless, for simplicity and ease of comparison with the classic lottery model, we ignore the possibility of fluctuations in m_i and focus instead on Poisson fluctuations propagule composition in each territory.

Appendix B: Derivation of growth equation

We separate the right hand side of Eq. (2) 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

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

Competition when rare

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

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

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.

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

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

We first calculate $\langle x_j \rangle_{\tilde{p}}$. Let $X = \sum_j x_j$ denote the total number of propagules in a territory and $\mathbf{x}_i = (x_1, \dots, x_{i-1}, x_{i+1}, \dots, x_G)$ denote the vector of non-focal abundances, so that $p(\mathbf{x}_i) = p_1(x_1) \dots p_{i-1}(x_{i-1}) p_{i+1}(x_{i+1}) \dots p_G(x_G)$. Then, \tilde{p} can be written as

$$\begin{aligned} \tilde{p}(\mathbf{x}_i) &= p(\mathbf{x}_i | X \geq 2, x_i = 1) \\ &= \frac{P(\mathbf{x}_i, X \geq 2 | x_i = 1)}{P(X \geq 2)} \\ &= \frac{1}{1 - (1 + L)e^{-L}} \sum_{X=2}^{\infty} P(X) p(\mathbf{x}_i | X_i = X - 1), \end{aligned} \quad (14)$$

and so

$$\begin{aligned} \langle x_j \rangle_{\tilde{p}} &= \sum_{\mathbf{x}_i} \tilde{p}(\mathbf{x}_i) x_j \\ &= \frac{1}{1 - (1 + L)e^{-L}} \sum_{X=2}^{\infty} P(X) \sum_{\mathbf{x}_i} p(\mathbf{x}_i | X_i = X - 1) x_j. \end{aligned} \quad (15)$$

The inner sum over \mathbf{x}_i is the mean number of propagules of a given nonfocal type j that will be found in a territory which received $X - 1$ nonfocal propagules in total, which is equal to $\frac{l_j}{L - l_i}(X - 1)$. Thus,

$$\begin{aligned} \langle x_j \rangle_{\tilde{p}} &= \frac{l_j}{1 - (1 + L)e^{-L}} \frac{1}{L - l_i} \sum_{k=2}^{\infty} P(X)(X - 1) \\ &= \frac{l_j}{1 - (1 + L)e^{-L}} \frac{L - 1 + e^{-L}}{L - l_i}, \end{aligned} \quad (16)$$

where the last line follows from $\sum_{X=2}^{\infty} P(X)(X - 1) = \sum_{X=1}^{\infty} P(X)(X - 1) = \sum_{X=1}^{\infty} P(X)X -$

621 $\sum_{X=1}^{\infty} P(X)$.

For analyzing the relative fluctuations in $\sum_{j \neq i} c_j x_j$, Eq. (16) 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 x_i Poisson dispersal probabilities conditional on $X_i \geq 1$, independently of the outcome of x_i . This gives $\langle x_j \rangle_{\tilde{q}} = \langle x_j \rangle_p / C = l_j / C$,

$$\begin{aligned} \sigma_{\tilde{q}}^2(x_j) &= \langle x_j^2 \rangle_{\tilde{q}} - \langle x_j \rangle_{\tilde{q}}^2 \\ &= \frac{1}{C} \langle x_j^2 \rangle_p - \frac{l_j^2}{C^2} \\ &= \frac{1}{C} (l_j^2 + l_j) - \frac{l_j^2}{C^2} \\ &= \frac{l_j^2}{C} \left(1 - \frac{1}{C}\right) + \frac{l_j}{C}, \end{aligned} \tag{17}$$

and

$$\begin{aligned} \sigma_{\tilde{q}}(x_j, x_k) &= \langle x_j x_k \rangle_{\tilde{q}} - \langle x_j \rangle_{\tilde{q}} \langle x_k \rangle_{\tilde{q}} \\ &= \frac{1}{C} \langle x_j x_k \rangle_p - \frac{l_j l_k}{C^2} \\ &= \frac{l_j l_k}{C} \left(1 - \frac{1}{C}\right), \end{aligned} \tag{18}$$

where $C = 1 - e^{-(L-l_i)}$ and $j \neq k$. The distribution \tilde{q} only approximates the situation after dispersal, since knowing that one focal genotype is among the propagules present restricts the possible outcomes for the x_j , so that the x_j cannot strictly be treated as independent of x_i . This seemingly minor distinction has meaningful consequences. To illustrate, suppose that the focal genotype is rare and the propagule density is high ($l_j \approx L \gg 1$). Then Eq. (16) correctly predicts that there are on average $L - 1$ nonfocal propagules $\langle x_j \rangle_{\tilde{p}} \approx L - 1$, with the focal propagule correctly excluded, whereas \tilde{q} predicts one extra $\langle x_j \rangle_{\tilde{q}} \approx L$. As a result, \tilde{q} gives pathological behavior for rare invaders (they have a rarity disadvantage), but its moments are quantitatively similar enough to those of \tilde{p} that it is sufficient for analyzing the relative fluctuations in $\sum_{j \neq i} c_j x_j$.

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

$$\sigma_{\bar{q}}^2(\sum_{j \neq i} c_j x_j) = \sum_{j \neq i} \left[c_j^2 \sigma_{\bar{q}}^2(x_j) + 2 \sum_{k > j} c_j c_k \sigma_{\bar{q}}(x_j, x_k) \right], \quad (19)$$

and using the fact that $\sigma_{\bar{q}}(x_j, x_k)$ and the first term in Eq. (17) are negative because $C < 1$, we

obtain an upper bound on the relative fluctuations in $\sum_{j \neq i} c_j x_j$,

$$\frac{\sigma(\sum_{j \neq i} c_j x_j)}{\langle \sum_{j \neq i} c_j x_j \rangle} < C^{1/2} \frac{\left(\sum_{j \neq i} c_j^2 l_j \right)^{1/2}}{\sum_{j \neq i} c_j l_j}. \quad (20)$$

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 $C = 1 - e^{-(L-l_i)}$).

Then, when at least some of the nonfocal propagule densities are large $l_j \gg 1$, the RHS of Eq. (20) 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 scenario occurs when $L - l_i \sim O(1)$, in which case the negative covariances (Eq. (18)) which were neglected in the RHS of Eq. (20) significantly reduce the overall variance $\sigma_{\bar{q}}^2(\sum_{j \neq i} c_j x_j)$.

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 than the others. Specifically, in the presence of a rare, extremely strong competitor ($c_j l_j \gg c_{j'} l_{j'}$ for all other nonfocal genotypes j' , and $l_j \ll 1$), then the RHS of Eq. (20) can be large and we cannot make the replacement Eq. (13).

Substituting Eqs. (13) and (16) into Eq. (12), we obtain

$$\Delta_r n_i \approx m_i R_i \frac{c_i}{C}, \quad (21)$$

where R_i is defined in Eq. (6).

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The final contribution, $\Delta_a n_i$, accounts for territories where two or more focal propagules are present (a stands for “abundant”). Similarly to Eq. (12), we have

$$\Delta_a n_i = U(1 - (1 + l_i)e^{l_i}) \left\langle \frac{c_i x_i}{\sum_j c_j x_j} \right\rangle_{\hat{p}} \quad (22)$$

651 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.

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

$$\left\langle \frac{c_i x_i}{\sum_j c_j x_j} \right\rangle_{\hat{p}} \approx \frac{c_i \langle x_i \rangle_{\hat{p}}}{\sum_j c_j \langle x_j \rangle_{\hat{p}}}. \quad (23)$$

Following a similar procedure as for $\Delta_r n_i$, where the vector of propagule abundances is denoted \mathbf{x} , the mean focal genotype abundance is,

$$\begin{aligned} \langle x_i \rangle_{\hat{p}} &= \sum_{\mathbf{x}} x_i p(\mathbf{x} | x_i \geq 2) \\ &= \sum_{x_i} x_i p(x_i | x_i \geq 2) \\ &= \frac{1}{1 - (1 + l_i)e^{-l_i}} \sum_{x_i \geq 2} p(x_i) x_i \\ &= l_i \frac{1 - e^{-l_i}}{1 - (1 + l_i)e^{-l_i}}. \end{aligned} \quad (24)$$

For nonfocal genotypes $j \neq i$, we have

$$\begin{aligned}
\langle x_j \rangle_{\hat{p}} &= \sum_{\mathbf{x}} x_j p(\mathbf{x} | x_i \geq 2) \\
&= \sum_X P(X | x_i \geq 2) \sum_{\mathbf{x}} x_j p(\mathbf{x} | x_i \geq 2, X) \\
&= \sum_X P(X | x_i \geq 2) \sum_{x_i} p(x_i | x_i \geq 2, X) \sum_{\mathbf{x}_i} x_j p(\mathbf{x}_i | X_i = X - x_i) \\
&= \sum_X P(X | x_i \geq 2) \sum_{x_i} p(x_i | x_i \geq 2, X) \frac{l_j(X - x_i)}{L - l_i} \\
&= \frac{l_j}{L - l_i} \left[\sum_X P(X | x_i \geq 2) X - \sum_{x_i} p(x_i | x_i \geq 2) x_i \right] \\
&= \frac{l_j}{L - l_i} \left(L \frac{1 - e^{-L}}{1 - (1 + L)e^{-L}} - l_i \frac{1 - e^{-l_i}}{1 - (1 + l_i)e^{-l_i}} \right). \tag{25}
\end{aligned}$$

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 \hat{q} , defined as the \mathbf{x} dispersal probabilities in a territory conditional on $x_i > 2$ (that is, treating the x_j as independent of x_i). All covariances between nonfocal genotypes 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, but in the relevant regime where $p(x_i = 0) \approx 0$ (since otherwise $D \gg 1$ and Δn_a is negligible), then

$$\sigma_{\hat{q}}^2(x_i) \approx \frac{l_i^2}{D} \left(1 - \frac{1}{D} \right) + \frac{l_i}{D}, \tag{26}$$

where $D = 1 - (1 + l_i)e^{-l_i}$, analogous to Eq. (17), and

$$\frac{\sigma_{\hat{q}}(\sum c_j x_j)}{\langle \sum c_j x_j \rangle} \approx \frac{\left(\sum_{j \neq i} c_j^2 l_j + c_i^2 \sigma_{\hat{q}}^2(x_i) \right)^{1/2}}{\sum_{j \neq i} c_j l_j + c_i l_i / D}. \tag{27}$$

Similarly to Eq. (20), the RHS of (27) will not be $\ll 1$ in the presence of a rare, extremely strong competitor. When this is not the case, then since l_i must be of order 1 or larger for $\Delta_a n$ to make an appreciable contribution to $\Delta_+ n_i$, the RHS of Eq. (27) is $\ll 1$ as desired.

Combining Eqs. (22) and (23), we obtain

$$\Delta_a n_i = m_i A_i \frac{c_i}{c}, \quad (28)$$

666 where A_i is defined in Eq. (7).