

# 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 the 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

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, 2004, 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, 2004, 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, 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 mathematical analysis of the major different forms of selection.

Nevertheless, there are strong indications there exist 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 (Fig. 1): 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 reproductive 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 hindrance 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, territorial model of 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



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”.

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 (“ $\alpha$ -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 genotypic 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 evolutionary 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

87 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 rep-  
 resenting competitive ability. This representation of competition is much simpler than having  
 90 coefficients for the pairwise effects of types on each other (e.g. the  $\alpha$  coefficients in the general-  
 ized 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  
 93 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  
 96 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  
 99 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  
 102 “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.

## 105 **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  
 108 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  
 111 is  $U(t) = T - N(t)$ .

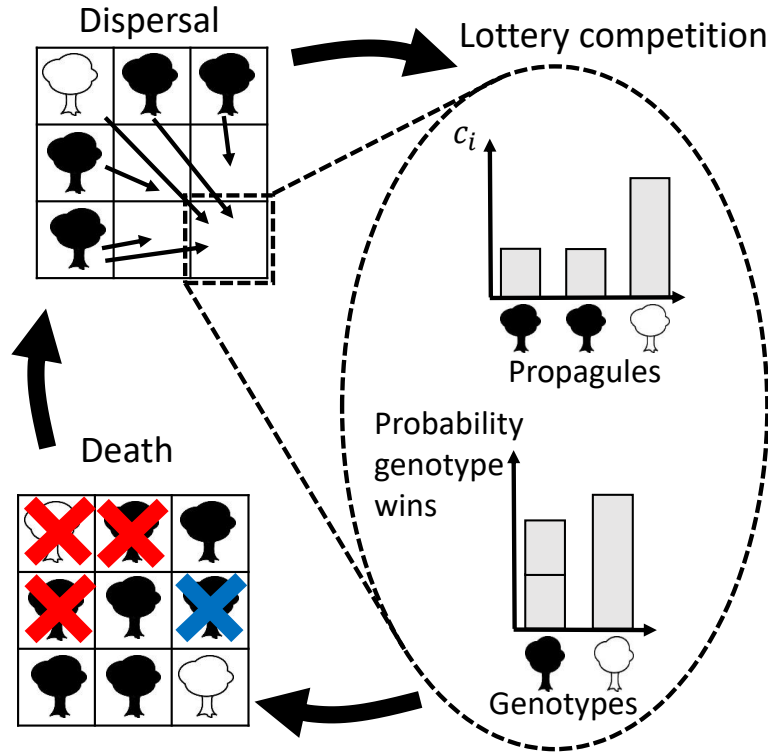


Figure 2: Each iteration of our lottery model has three main elements. First, propagules are produced by adults which are dispersed at random over the unoccupied territories (only propagules landing on unoccupied territories are shown). Lottery competition then occurs in each unoccupied territory (competition 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”).

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 at random uniformly, 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 uniform 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 as they develop. This territorial contest is modeled as a weighted lottery: the probability that 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 classic lottery model allows us to replace the abstract Wright-Fisher relative fitnesses  $w_i$   
 159 with more ecologically-grounded fecundity, competitive ability and mortality parameters  $b_i$ ,  $c_i$   
 and  $d_i$ , respectively. Since birth and death rates affect absolute abundances, this allows us to  
 evaluate selection at different densities (after appropriate extensions are made), in an otherwise  
 162 very similar model to the canonical Wright-Fisher model. Note that the classic lottery model,  
 and the present work, both 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 ( $l_i \ll 1$ ): 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  $U$ . Intuitively, the cause of this discrepancy is that individuals are discrete. Genotypes with few propagules depend on the outcome of contests in territories where they have at least one propagule present, not some small fraction of a propagule as would be implied by low propagule density  $l$  in the classic lottery model. In other words, deviations from the mean propagule composition  $l_1, l_2, \dots, l_G$  are important at low density.

Dispersal is Poisson, and so 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 lottery model, not all  $U$  unoccupied territories are claimed each iteration; a fraction  $e^{-L}$  remain unoccupied.

## Results

### 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. 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  $l_i$  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 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 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

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

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 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 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  $A_i \rightarrow 1/L$ ). 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 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. 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 of the classic lottery model at low densities (low  $l_1$  and  $l_2$ ).

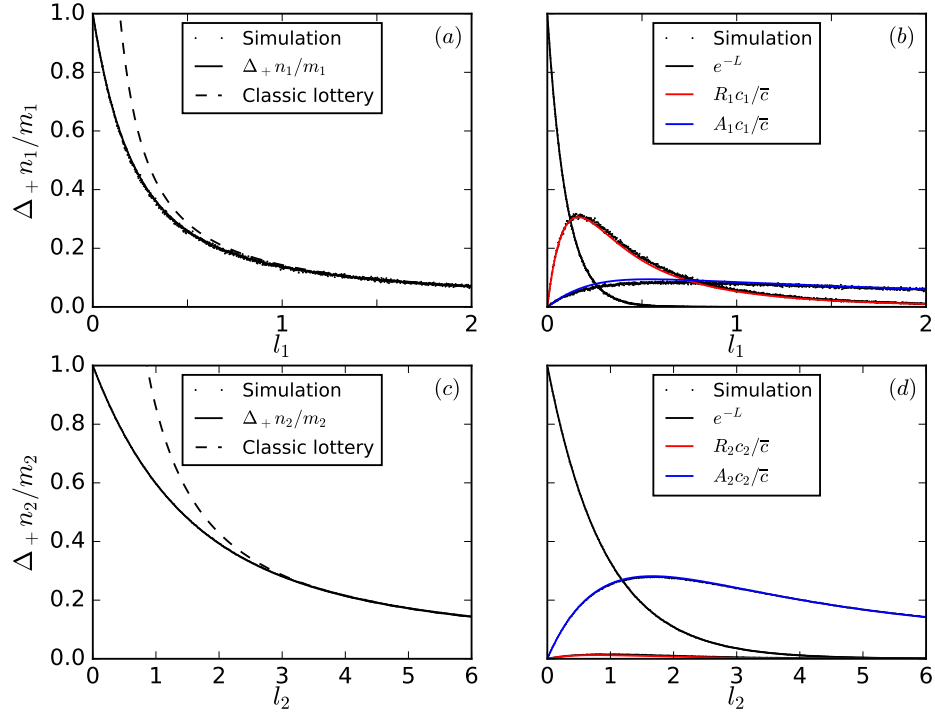


Figure 3: The change in genotype abundances in a density dependent lottery model is closely approximated by Eq. (5).  $\Delta_+ n_i / m_i$  from Eq. (5) (and its separate components) are shown, along with direct simulations of random dispersal and lottery competition over one iteration over a range of 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.

## Invasion of rare genotypes and coexistence

228 In our model (section “Model”), each genotype is defined by three traits:  $b$ ,  $c$  and  $d$ . To determine 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 231 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

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

234 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

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

237 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  $c_j = c_i$ , 240 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 243 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.

246 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 249 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-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 colonizer ( $b$ -specialist) can then exploit. A similar argument applies for coexistence between high- $c$  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 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 (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), 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 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 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

parameter regimes in our model.

The ideal environment 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. (4), 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  $L \approx b_i/d_i \gg 1$ , and every unoccupied territory will be heavily contested. The population density is also high  $N/T \approx 1$  (since  $L = b_i N/(N - T) = b_i/(1 - T/N)$ ).

Disturbed environments are characterized by unavoidably high extrinsic mortality caused by physical destruction. Environmental variability, and disturbances in particular, can be modeled 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 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 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  $c_i/\bar{c}$  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 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 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



no territorial contests occurring.

The alternative view is that, while stressful environments imply lower  $b$  and support a lower  
303 number of individuals per unit area compared what is attainable in ideal environments, stressed  
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  
306 consumable resources, we might expect intense resource competition (for empirical support, see  
Davis et al. 1998). Thus,  $b$  may actually appreciably exceed  $d$  under stressful conditions, even  
though the absolute value of  $b$  is small.

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

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 surviv-  
315 ing to deterministic abundances 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 de-  
318 termined by which trait changes are both available, and confer an appreciable benefit, where  
availability is subject to constraints imposed by the environment.

For example, in Grime's interpretation of stressful environments,  $L$  is low, so competition is  
321 not important, and only mutants with greater  $b$  or lower  $d$  will have an appreciably greater  $\Delta n_i$ .  
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  
324 that mutations lower  $d$ , making this the expected direction of evolutionary change.

Following Grime's original argument for a triangular scheme (Grime, 1977), Fig. 5 repre-  
sents each environmental extreme schematically as a vertex on a triangular space defined by  
327 perpendicular stress and disturbance axes. The ideal environment lies at the origin (no stress or

	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).

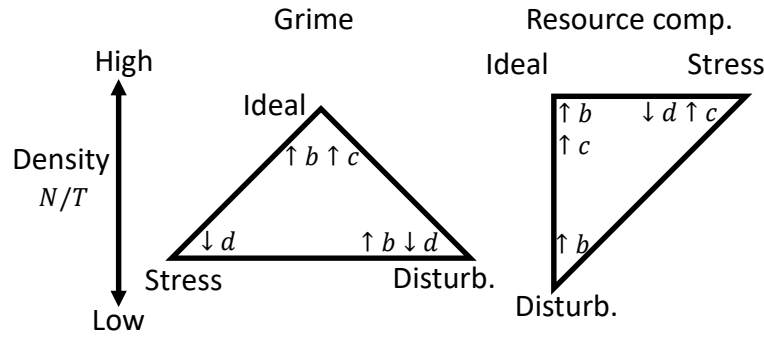


Figure 5: The realization of Grime’s triangle in our model. Schematic representation of the triangular space bounded by the low/high extremes of stress/disturbance. The low- $T$  interpretation of stress is also shown. The vertices of the triangles correspond to different environmental extremes. Selection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

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 evolutionary change is different at each vertex, leading to the emergence of 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  $c$ .  
339 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 produc-  
tion (Grime, 1977), whereas the analogous “opportunistic” fishes have large intrinsic growth rates  
342 (Winemiller and Rose, 1992). In corals, ruderals are distinguished by brood spawning (rather  
than broadcast spawning; Darling et al. 2012). This corresponds to higher parental investment  
and lower overall propagule production — counter-intuitively, a stereotypical “K-selected”, high-  
345 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  
348 resistance to physical destruction, but it is hard to reduce mortality in the face of severe dis-  
turbances. 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  
351 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  
354 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.

357 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  
360 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  
363 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.

## Cyclical birth and death rates

So we have only considered the low-frequency invasion behavior of Eq. (5). Here we start  
exploring its full time-dependent behaviour. We consider a situation where birth and death rates  
vary periodically with amplitude sufficient to cause large changes in population density. This is  
inspired by natural *Drosophila* populations, which expand rapidly in the warmer months when  
fruit is abundant, but largely die off in the colder months. Within this seasonal population  
density cycle, hundreds of polymorphisms also cycle in frequency (Bergland et al., 2014). Some  
of these polymorphisms may be adaptive and potentially millions of years old (Bergland et al.,  
2014). In this situation, selection on allele frequencies occurs on the same time scale as population  
demography, a considerably more complicated scenario than classical sweeps in demographically  
stable populations (Messer et al., 2016).

The classical population genetic treatment of fluctuating selection suggests that environmen-  
tal fluctuations do not promote coexistence. Allele frequencies are successively multiplied by  
relative fitness values for each environmental iteration, and so two alleles favored in different  
environments can only stably coexist if the product of fitnesses for one type exactly equals the  
product for the other (Dempster, 1955). Thus, stable coexistence still requires frequency depen-

dent selection or heterozygote advantage (as is required in a constant environment).

390 This classical argument overlooks two general mechanisms by which fluctuating selection promotes coexistence. The first is the “storage effect”, which introduces a form of frequency dependent selection that promotes coexistence in the presence of environmental fluctuations but  
393 not in a constant environment. The storage effect occurs when some individuals are protected from selection. This is the case in both the classic lottery model and our extension of it: a fraction  $(1 - d_i)n_i$  of each type’s adults do not experience selection in a given iteration. Protection  
396 from selection favors rare types in fluctuating environments because abundant types cannot fully exploit environmental periods that favor them (since only some of the rare type can be displaced), whereas rare types gain the full benefits of their favorable periods (far more abundant type  
399 individuals can be displaced than the rare types could possibly displace) (Chesson and Warner, 1981).

The second mechanism we will call the “bounded density effect”, since it is a consequence of  
402 the inhibition of reproduction at high population densities (Dempster’s (1955) argument ignores density-dependent effects). If there is growth from low to high density in each environmental cycle, then types that are abundant determine the time available for growth every cycle, with less  
405 time spent growing in cycles where they are favored, and more in cycles where they are not. This promotes coexistence even in the absence of frequency dependent selection (Yi and Dean, 2013).

Figure 6 shows the behavior of Eq. (5) for an example where  $b$  and  $d$  cycle between zero and  
408 positive values (“summers” with rapid growth and no mortality, and “winters” with mortality and no growth). Both the storage effect (winter survivors are protected from selection when summer starts) and the bounded density effect (expansion to high density occurs every cycle) are  
411 present. Two types are present, a  $b$  specialist, which is better at rapidly growing in the summer (higher  $b$ ), and a  $d$  specialist which is better at surviving the winter (lower  $d$ ). Neither type has an advantage over a full environmental cycle, and they stably coexist. Stable coexistence between  
414  $b$  and  $d$  specialists was not possible in a constant environment (section “”).

Current approaches to population genetic inference treat demography and selection sepa-

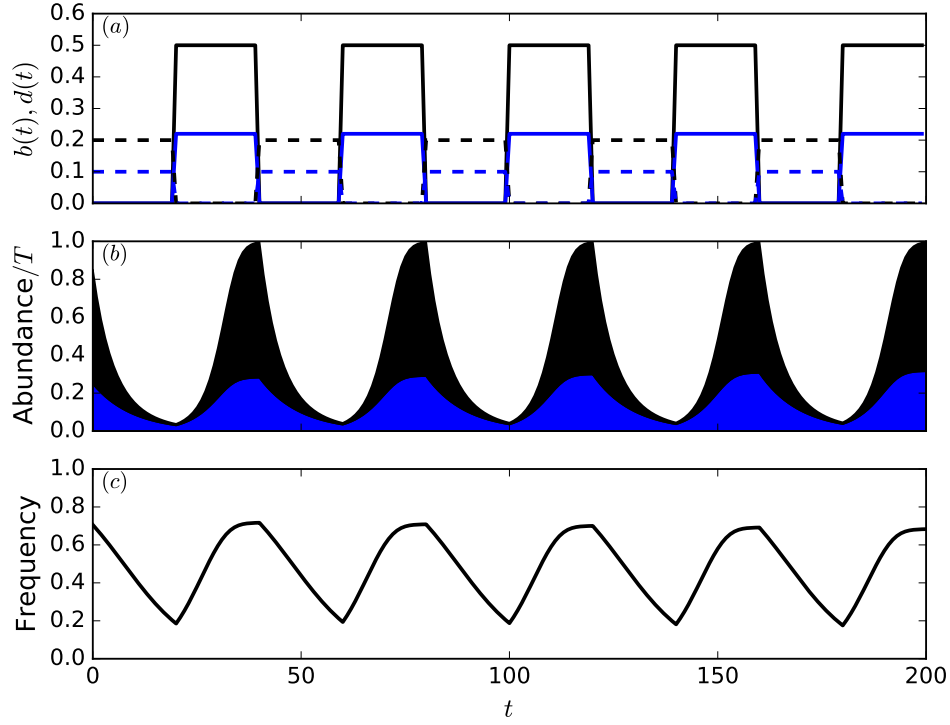


Figure 6: Stable coexistence between  $b$  and  $d$  specialists when  $b$  and  $d$  fluctuate. (a)  $b$  (solid lines) and  $d$  (dashed lines) alternate being nonzero. The  $b$  specialist (black) has higher  $b$  and  $d$  than the  $d$  specialist (blue). (b) Both types grow during the positive  $b$  phase, and decline during the positive  $d$  phase, but the  $d$  specialist (blue) does so at a slower rate than the  $b$  specialist (black). Total height (blue+black) is population size  $N$ . (c) The positive  $b$  and  $d$  phases favor the  $b$  and  $d$  specialists, respectively, and they stably coexist due to the storage effect.

rately, typically fitting a demographic model to data on ostensibly neutral sites, and then infer-  
 417 ring selection against this demographic background, despite the recurrent problem that the two  
 are confounded (Schridder et al., 2016). in expanding (e.g. humans) or seasonally-cycling (e.g. .

## Discussion

420 As discussed in “Primary strategies and Grime’s triangle”, adaptive evolution in the direction  
 predicted by our generalized lottery model produces traits consistent with Grime’s scheme, with  
 the possible exception of selection for  $b$  at high density, because the corresponding trait data is

423 ambiguous. Selection for  $b$  at high density 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), yet in the  $r/K$  scheme, high density populations  
426 should be subject to  $K$ , not  $r$ , selection.

It is not surprising 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 intrinsi-  
429 cally 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 competi-  
432 tor's propagules may happen to arrive first, gaining a decisive developmental advantage. 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 the Wright-Fisher model is  
435 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" sometimes refers generally to selection at high density  
438 (Pianka, 1972), encompassing both selection for higher saturation density (MacArthur and Wil-  
son, 1967) and competitive ability (Gill, 1974). Contrary to an  $r/K$  dichotomy, empirical studies  
have shown that maximal growth rate and saturation density (measured by abundance) are posi-  
441 tively 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, this correlation is not surprising since the saturation density,  
444 which is determined by a balance between births and deaths, increases with  $b$ .

There is support for a negative relationship between competitive success at high density  
and maximal growth rate (Luckinbill, 1979), consistent with an  $r/K$  dichotomy. This could be  
447 driven by a tradeoff between individual size and reproductive rate. To avoid confusion with  
other forms of " $K$ -selection", selection for competitive ability has been called " $\alpha$ -selection" after  
the competition coefficients in the Lotka-Volterra equation (Case and Gilpin, 1974; Gill, 1974;

450 Joshi et al., 2001). However, competitive success as measured by  $\alpha$  (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-structured competition and territoriality, it also includes the ability of each genotype to produce contestants i.e.  $b$  in our model. Our  $c$  is strictly competitive ability only — as such, changes in  $c$  do not directly affect population density (section “Model”). The clean separation of a strictly-relative  $c$  parameter is particularly useful from an evolutionary genetics perspective, essentially embedding a zero-sum relative 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 and Fisher, 2007; Gerrish and Lenski, 1998) between  $c$ -strategists on the one hand, and  $b$ - and  $d$ -strategists on the other.

$K$ -selection in the narrow logistic sense of selection for a greater environmental carrying 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 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 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, where  $b$  and  $c$  specialists can co-exist. This “colonization-competition trade-off” 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



477 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  
480 retain overlapping generations and competition at the juvenile phase only.

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), referring instead to  
483 the direction of adaptive trait evolution under different regimes of stress and disturbance, which  
is closer in spirit to Grime’s arguments (Grime, 1974, 1977). In addition, we have not assumed any  
kind of trade-offs or pleiotropy between  $b$ ,  $c$  and  $d$ , only constraints imposed by the environment  
486 on the order of magnitude of  $b$  and  $d$ . 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  
489 (Darling et al., 2012). Fig. 5 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.  
492 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.

495 One limitation of our model as a general-purpose model of density-dependent selection is  
the restriction of competition to interference competition between juveniles for durable resources  
(lottery recruitment to adulthood), analogous to the ubiquitous assumption of viability selection  
498 in population genetics (Ewens, 2004, p. 45). 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  
501 to restricted movement through soils), resource competition and territorial acquisition effectively  
coincide, and in principle resource competition could be represented by a competitive ability  $c$  (or  
conversely,  $c$  should be derivable from resource competition). The situation is more complicated

504 if the resources are well-mixed, since, in general, resource levels then need to be explicitly tracked.  
It seems plausible that explicit resource tracking may not be necessary when the focus is on the  
evolution of similar genotypes rather than the stable co-existence of widely differing species  
507 (Ram et al., 2016). 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  
510 competition models to the density-dependent selection literature, since most of the former has  
to date been focused on narrower issues of the role of competition at low resource availability  
(Aerts, 1999; Davis et al., 1998; Tilman, 2007).

513 While our model can be applied to species rather than genotypes (e.g. ecological invasions),  
our focus is genotype evolution. 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  
516 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  
519 when looking at species interactions rather than genotype evolution.

## References

- Aerts, R. 1999. Interspecific competition in natural plant communities: mechanisms, trade-offs  
522 and plant-soil feedbacks. *Journal of Experimental Botany* 50:29–37.
- Allan, J. D. 1976. Life history patterns in zooplankton. *The American Naturalist* 110:165–180.
- Bergland, A. O., E. L. Behrman, K. R. O’Brien, P. S. Schmidt, and D. A. Petrov. 2014. Genomic  
525 evidence of rapid and stable adaptive oscillations over seasonal time scales in drosophila. *PLOS*  
*Genetics* 10:1–19.
- Bolker, B. M., and S. W. Pacala. 1999. Spatial moment equations for plant competition: Under-

standing spatial strategies and the advantages of short dispersal. *The American Naturalist* 153:575–602.

Boyce, M. S. 1984. Restitution of r-and k-selection as a model of density-dependent natural selection. *Annual Review of Ecology and Systematics* 15:427–447.

Case, T. J., and M. E. Gilpin. 1974. Interference competition and niche theory. *Proceedings of the National Academy of Sciences* 71:3073–3077.

Chesson, P. L., and R. R. Warner. 1981. Environmental variability promotes coexistence in lottery competitive systems. *American Naturalist* pages 923–943.

Darling, E. S., L. Alvarez-Filip, T. A. Oliver, T. R. McClanahan, and I. M. Côté. 2012. Evaluating life-history strategies of reef corals from species traits. *Ecology Letters* 15:1378–1386.

Davis, M. A., K. J. Wragge, and P. B. Reich. 1998. Competition between tree seedlings and herbaceous vegetation: support for a theory of resource supply and demand. *Journal of Ecology* 86:652–661.

Dempster, E. R. 1955. Maintenance of genetic heterogeneity. *Cold Spring Harb Symp Quant Biol* 20:25–31.

Desai, M. M., and D. S. Fisher. 2007. Beneficial mutation–selection balance and the effect of linkage on positive selection. *Genetics* 176:1759–1798.

Ewens, W. J. 2004. *Mathematical Population Genetics 1: Theoretical Introduction*, vol. 27. Springer Science & Business Media.

Fitzsimmons, J. M., S. E. Schoustra, J. T. Kerr, and R. Kassen. 2010. Population consequences of mutational events: effects of antibiotic resistance on the r/k trade-off. *Evolutionary Ecology* 24:227–236.

Gerrish, P. J., and R. E. Lenski. 1998. The fate of competing beneficial mutations in an asexual population. *Genetica* 102:127–144.

- 552 Gill, D. E. 1974. Intrinsic rate of increase, saturation density, and competitive ability. ii. the  
evolution of competitive ability. *American Naturalist* 108:103–116.
- Grime, J. P. 1974. Vegetation classification by reference to strategies. *Nature* 250:26–31.
- 555 ———. 1977. Evidence for the existence of three primary strategies in plants and its relevance to  
ecological and evolutionary theory. *American Naturalist* 111:1169–1194.
- . 1988. *Plant Evolutionary Biology*, chap. The C-S-R model of primary plant strategies —  
558 origins, implications and tests, pages 371–393. Springer Netherlands, Dordrecht.
- Hendriks, A. J., J. L. Maas-Diepeveen, E. H. Heugens, and N. M. van Straalen. 2005. Meta-analysis  
of intrinsic rates of increase and carrying capacity of populations affected by toxic and other  
561 stressors. *Environmental Toxicology and Chemistry* 24:2267–2277.
- Joshi, A., N. Prasad, and M. Shakarad. 2001. K-selection,  $\alpha$ -selection, effectiveness, and tolerance  
in competition: density-dependent selection revisited. *Journal of Genetics* 80:63–75.
- 564 Knowlton, N. 2001. The future of coral reefs. *Proceedings of the National Academy of Sciences  
of the United States of America* 98:5419–5425.
- Kuno, E. 1991. Some strange properties of the logistic equation defined with  $r$  and  $K$ : Inherent  
567 defects or artifacts? *Researches on Population Ecology* 33:33–39.
- Levins, R., and D. Culver. 1971. Regional coexistence of species and competition between rare  
species. *Proceedings of the National Academy of Sciences* 68:1246–1248.
- 570 Luckinbill, L. S. 1978.  $r$  and  $k$  selection in experimental populations of *escherichia coli*. *Science*  
(New York, NY) 202:1201–1203.
- . 1979. Selection and the  $r/k$  continuum in experimental populations of protozoa. *Ameri-  
573 can Naturalist* pages 427–437.

MacArthur, R. H. 1962. Some generalized theorems of natural selection. *Proceedings of the National Academy of Sciences* 48:1893–1897.

576 MacArthur, R. H., and E. O. Wilson. 1967. *Theory of Island Biogeography*. Princeton University Press.

Messer, P. W., S. P. Ellner, and N. G. Hairston. 2016. Can population genetics adapt to rapid  
579 evolution? *Trends in Genetics* 32:408–418.

Pianka, E. R. 1970. On r- and K-Selection. *The American Naturalist* 104:592–597.

———. 1972. r and K selection or b and d selection? *The American Naturalist* 106:581–588.

582 Ram, Y., E. Dellus-Gur, M. Bibi, U. Obolski, J. Berman, and L. Hadany. 2016. Predicting microbial relative growth in a mixed culture from growth curve data. *bioRxiv* .

Reznick, D., M. J. Bryant, and F. Bashey. 2002. r- and k-selection revisited: The role of population  
585 regulation in life-history evolution. *Ecology* 83:1509–1520.

Sale, P. F. 1977. Maintenance of high diversity in coral reef fish communities. *The American Naturalist* 111:337–359.

588 Schrider, D. R., A. G. Shanku, and A. D. Kern. 2016. Effects of linked selective sweeps on demographic inference and model selection. *Genetics* 204:1207–1223.

Shmida, A., and S. Ellner. 1984. Coexistence of plant species with similar niches. *Vegetatio*  
591 58:29–55.

Southwood, T. R. E. 1977. Habitat, the templet for ecological strategies? *Journal of Animal Ecology* 46:337–365.

594 Stearns, S. C. 1977. The evolution of life history traits: A critique of the theory and a review of the data. *Annual Review of Ecology and Systematics* 8:145–171.

Svardal, H., C. Rueffler, and J. Hermisson. 2015. A general condition for adaptive genetic poly-  
597 morphism in temporally and spatially heterogeneous environments. *Theoretical Population*  
*Biology* 99:76 – 97.

Taylor, D. R., L. W. Aarssen, and C. Loehle. 1990. On the relationship between r/k selection and  
600 environmental carrying capacity: a new habitat templet for plant life history strategies. *Oikos*  
pages 239–250.

Tilman, D. 1982. *Resource competition and community structure*. 17. Princeton University Press.

603 ———. 1994. Competition and biodiversity in spatially structured habitats. *Ecology* 75:2–16.

———. 2007. Resource competition and plant traits: a response to craine et al. 2005. *Journal of*  
*Ecology* 95:231–234.

606 Violle, C., M.-L. Navas, D. Vile, E. Kazakou, C. Fortunel, I. Hummel, and E. Garnier. 2007. Let  
the concept of trait be functional! *Oikos* 116:882–892.

Westoby, M. 1998. A leaf-height-seed (LHS) plant ecology strategy scheme. *Plant and soil*  
609 199:213–227.

Winemiller, K. O., D. B. Fitzgerald, L. M. Bower, and E. R. Pianka. 2015. Functional traits, con-  
vergent evolution, and periodic tables of niches. *Ecology Letters* 18:737–751.

612 Winemiller, K. O., and K. A. Rose. 1992. Patterns of life-history diversification in north american  
fishes: implications for population regulation. *Canadian Journal of Fisheries and Aquatic*  
*Sciences* 49:2196–2218.

615 Yi, X., and A. M. Dean. 2013. Bounded population sizes, fluctuating selection and the tempo and  
mode of coexistence. *Proceedings of the National Academy of Sciences* 110:16945–16950.

## Appendix A: Poisson approximation

618 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  $T$ ,  
621 holding  $n_i/T$  fixed, the Poisson limit theorem implies that this multinomial distribution for the  
 $x_i$  across 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  
624 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 disper-  
sal from the outset. The total number of genotype  $i$  propagules  $\sum x_i$  (summed over unoccupied  
627 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  
630 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  
633 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.

### 636 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  
639 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  $Up_i(1)P(X_i \geq 1) = b_i n_i e^{-l_i}(1 - e^{-(L-l_i)})$ . 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.

Our “mean field” approximation is to replace  $x_j$  with its mean in the last term in Eq. (12),

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

Below we justify this replacement by arguing that the standard deviation  $\sigma_{\tilde{p}}(\sum_{j \neq i} c_j x_j)$  (with respect to  $\tilde{p}$ ), is much smaller than  $\langle \sum_{j \neq i} c_j x_j \rangle_{\tilde{p}}$ .

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,

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

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

The exact analysis of the fluctuations in  $\sum_{j \neq i} c_j x_j$  is complicated because the  $x_j$  are not independent with respect to  $\tilde{p}$ . These fluctuations are part of the “drift” in type abundances which we leave for future work. Here we use the following approximation to give some insight into the magnitude of these fluctuations and also the nature of the correlations between the  $x_j$ . We replace  $\tilde{p}$  with  $\tilde{q}$ , defined as the  $\mathbf{x}_i$  Poisson dispersal probabilities conditional on  $X_i \geq 1$  (which are independent). The distinction between  $\tilde{p}$  with  $\tilde{q}$  will be discussed further below. The  $\tilde{q}$  approximation

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  $j \neq k$ .

654 The exact distribution  $\tilde{p}$  assumes that exactly one of the propagules present in a given site after dispersal belongs to the focal type, whereas  $\tilde{q}$  assumes that there is a focal propagule present before non-focal dispersal commences. As a result,  $\tilde{q}$  predicts that the mean propagule  
657 density is greater than  $L$  (in sites with only one focal propagule is present) when the focal type is rare and the propagule density is high. This is erroneous, because the mean number of propagules in every site is  $L$  by definition. Specifically, if  $L - l_i \approx L \gg 1$ , then the mean  
660 propagule density predicted by  $\tilde{q}$  is approximately  $L + 1$ . The discrepancy causes rare invaders to have an intrinsic rarity disadvantage (territorial contests under  $\tilde{q}$  are more intense than they should be). In contrast, Eq. (16) correctly predicts that there are on average  $\sum_{j \neq i} \langle x_j \rangle_{\tilde{p}} \approx L - 1$   
663 nonfocal propagules because  $\tilde{p}$  accounts for potentially large negative covariances between the  $x_j$  “after dispersal”. By neglecting the latter covariences,  $\tilde{q}$  overestimates the fluctuations in  $\sum_{j \neq i} c_j x_j$ ; thus  $\tilde{q}$  gives an upper bound on the fluctuations. The discrepancy between  $\tilde{q}$  and  $\tilde{p}$  will be largest

when  $L$  is of order 1 or smaller, because then the propagule assumed to already be present under  $\tilde{q}$  is comparable to, or greater than, the entire propgaule density.

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

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

and using the fact that  $\sigma_{\tilde{q}}(x_j, x_k)$  and the first term in Eq. (17) are negative because  $C < 1$ , we obtain an upper bound on the relative fluctuations in  $\sum_{j \neq i} c_j x_j$ ,

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

Suppose that the  $c_j$  are all of similar magnitude (their ratios are of order one). Then Eq. (20) is  $\ll 1$  for the case when  $L - l_i \ll 1$  (due to the factor of  $C^{1/2}$ ), and also for the case when at least some of the nonfocal propagule densities are large  $l_j \gg 1$  (since it is then of order  $1/\sqrt{L - l_i}$ ). The worst case scenario occurs when  $L - l_i$  is of order one. Then Eq. (20) gives a relative error of approximately 50%, which from our earlier discussion we know to be a substantial overestimate when  $L$  is of order 1. Our numerical results (Fig. 3) confirm that the relative errors are indeed small.

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

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

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

## 684 Competition when abundant

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

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

687 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 argue that the relative fluctuations in  $\sum c_j x_j$  are much smaller than 1 (with respect  
690 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). All covariances between nonfocal genotypes are now zero, so that  $\sigma_{\hat{q}}^2(\sum c_j x_j) = \sum c_j^2 \sigma_{\hat{q}}^2(x_j)$ , where  $\sigma_{\hat{q}}^2(x_j) = l_j$  for  $j \neq i$ , and

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

where  $D = 1 - (1 + l_i)e^{-l_i}$ , and

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

Similarly to Eq. (20), the RHS of Eq. (27) is  $\ll 1$  for the case that  $L \ll 1$  (due to a factor of  $D^{1/2}$ ), and also for the case when at least some of the propagule densities (focal or nonfocal) are large — provided that  $c_i$  and the  $c_j$  are all of similar magnitude. Again, the worst case scenario occurs when  $l_i$  and  $L - l_i$  are of order 1, in which case Eq. (27) is around 35%, which is again where the  $\hat{q}$  approximation produces the biggest overestimate of the fluctuations in  $\mathbf{x}$ . Similarly

to Eq. (20), the RHS of (27) will not be  $\ll 1$  in the presence of a rare, extremely strong competitor.

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

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

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