

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

Manuscript elements:

Keywords: r/K selection, absolute fitness, eco-evo, interference competition, competition-colonization trade-off.

Manuscript type: Article.

Prepared using the suggested L^AT_EX template for *Am. Nat.*

Abstract

[Needs to be revised but won't do that now in case it has to become Nature introductory paragraph] Fitness is typically represented in heavily simplified terms in evolutionary genetics, often using constant selection coefficients. This excludes fundamental ecological factors such as dynamic population size or density-dependence from the most genetically-realistic treatments of evolution, a problem that inspired MacArthur's influential but problematic r/K theory. Following 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 territorial 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 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 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 [Should be removed if we need space]

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. This simplified treatment of selection facilitates genetic realism, and can be justified over sufficiently short time intervals (Ewens, 2004, p. 276). The emphasis here is to infer past selection, migration and demographic change given a sample of nucleotide sequences, or to predict how allele frequencies change over time based on their fitness effects, population structure, genetic drift and linkage. The resulting picture of evolution excludes basic elements of the ecological underpinnings of selection, including density dependence, and how selection affects population size. This complicates the inference of past selection, because demographic changes can look genealogically very similar to selective frequency changes (Barton, 1998).

By contrast, models of phenotypic trait evolution use absolute fitness functions to describe how some traits of interest affect survival and reproduction in particular ecological scenarios (Diekmann et al., 2004; Metz et al., 1992). These fitness functions can be quite problem-specific and often only account for a few traits at a time. The emphasis here is on the conditions for invasion from low frequencies and co-existence, rather than frequency or abundance trajectories over time. For instance, adaptive dynamics uses “invasion fitness” to explore the consequences of eco-evolutionary feedbacks (Diekmann et al., 2004).

It is challenging to generalize beyond particular traits or ecological scenarios to model fundamentally 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) and its environment. A detailed, trait-based, predictive model of fitness would be enormously complicated and situation-specific. 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
45 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). However, the r/K scheme's original purpose
48 was to extend the existing population-genetic treatment of selection to account for population
density (MacArthur, 1962). Few attempts have been made to develop it further along these lines.

Empirical trait classification studies have suggested the existence of a few "primary strate-
51 gies", reflecting broadly distinct responses to selection (Winemiller et al., 2015). Grime famously
considered (Grime, 1974, 1977, 1988; Westoby, 1998) two broad determinants of population den-
sity: stress (persistent hardship e.g. due to resource scarcity or unfavorable temperatures) and
54 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 de-
noted by C/S/R respectively (Fig. 1): competitors "C" excel in low stress, low disturbance envi-
57 ronments; stress tolerators "S" excel in high stress, low disturbance environments; and ruderals
"R" excel in low stress, high disturbance environments. Population persistence is not possible in
high-stress, high-disturbance environments. Grime showed that measures of C, S and R across
60 a wide range of plant species are anti-correlated, so that strong C-strategists are weak S and R
strategists, and so on, creating a triangular C/S/R ternary plot (Grime, 1974). Similar schemes
were proposed for insects (Southwood, 1977), fishes (Winemiller and Rose, 1992), and zooplank-
63 ton (Allan, 1976). More recently, modern hierarchical clustering techniques have revealed distinct
trait clusters in corals analogous to Grime's primary strategies (Darling et al., 2012). These em-
pirical findings suggest that functional traits contribute to fitness predominantly via a few key
66 factors such as intrinsic reproductive rate or stress-tolerance.

Here we explore the interplay between some "key factors" of fitness in a simplified, terri-
torial model of growth, dispersal and competition. This broadly follows the original spirit of
69 MacArthur's r/K scheme. More specifically, our aim is to begin adding some ecological realism
to population genetics' time-depedendent, genetically-focused view of evolution. We revisit the



Figure 1: Schematic of Grime's triangle. The two axes show increasing levels of environmental stress and disturbance, respectively. Population persistence 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".

classic lottery model of Chesson and Warner (1981), which has two features that make it well suited for this role, but one critical flaw that we rectify here.

The first feature is that the lottery representation of competition is particularly concise. 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, with probabilities weighted by a coefficient for each type representing competitive ability, akin to a lottery (Sale, 1977). By comparison, coefficients for the pairwise effects of types on each other (e.g. the α coefficients in the generalized Lotka-Volterra equations and the associated concept of " α -selection"; Case and Gilpin 1974; Gill 1974; Joshi et al. 2001), or explicit resource consumption (Tilman, 1982), are much more complicated. The second feature is the close connection between the lottery model and one of the foundational models of population genetics, the Wright-Fisher model of genetic drift, which we discuss further below.

The critical flaw of the classic lottery model is that it breaks down at low densities (few propagules dispersing to each territory), precluding density-dependent behaviour. Our first task is to analytically extend the classic lottery model to correctly account for low density behavior

(sections “Model” and “Mean field approximation”).

Using our extended lottery model, we then revisit Grime’s C/S/R scheme, and evaluate how
90 C/S/R manifests at the level of within-population genotypic evolution (as opposed to phenotypic
divergence between species; sections “Invasion of rare genotypes and coexistence” and “Primary
strategies and Grime’s triangle”). This represents a “sanity check” on our density-dependent
93 lottery model. The resulting formulation of the C/S/R scheme is mathematical, in contrast
to Grime’s original verbal and descriptive approach, which is a recognized hindrance to the
evaluation or broader application of the C/S/R scheme (e.g. Tilman 2007).

96 We then explore some time-dependent behavior of our extended lottery model. Taking an
example inspired by recent studies of rapid, seasonal evolution in *Drosophila* (Bergland et al.,
2014), we discuss how environmental fluctuations might stabilize polymorphisms in the presence
99 of cyclical population density.

Model

We assume that reproductively mature individuals (“adults”) each require their own territory to
102 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 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
105 adults is $N(t) = \sum_i n_i(t)$, and the number of unoccupied territories is $U(t) = T - N(t)$.

We assume that the n_i and T are large enough that stochastic fluctuations in the n_i (“drift”) can
be ignored. We derive deterministic equations for the expected change in the n_i over time, leaving
108 the evaluation of drift for future work. In particular, we do not evaluate the initial stochastic
behaviour of adaptive mutant lineages while they are at low abundance. When considering
mutations, we restrict our attention to the earliest deterministic behavior of mutant lineages while
111 they are still at low frequency (the transition to deterministic growth occurs at an abundance of
order equal to their inverse expected absolute growth rate; Uecker and Hermisson 2011).

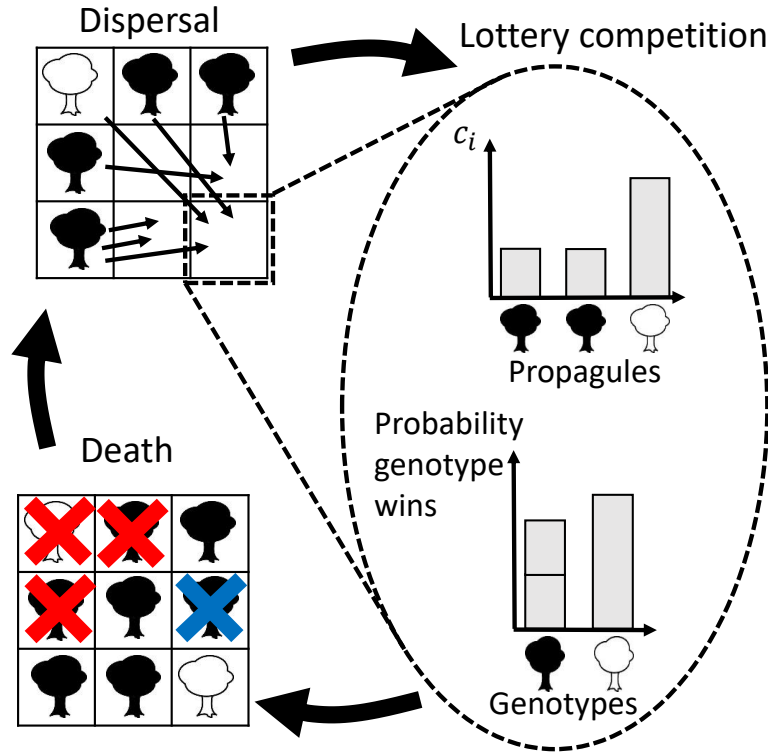


Figure 2: Each iteration of our lottery model has three main elements. First, propagules are produced by adults which are dispersed at random over the unoccupied territories (only propagules landing on unoccupied territories are shown). Lottery competition then occurs in each unoccupied territory (competition 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 un-
 114 occupied territories. We assume that adults cannot be ousted from their territories, so that m_i
 only includes propagules landing on unoccupied territories. Propagules disperse at random over
 the unoccupied territories, regardless of distance from their parents, and independently of each
 117 other. 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 depend on population density N . For example,
 120 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). In section
 123 “Cyclical birth and death rates” we evaluate Eq. (4) numerically using this functional form for
 m_i , with b_i assumed to be constant.

In the sections “Invasion of rare genotypes and coexistence” and “Primary strategies and
 126 Grime’s triangle”, we assume the simpler form $m_i = b_i n_i$, with constant b_i , meaning that all
 propagules land on unoccupied territories (a form of directed dispersal). This simplifies the
 mathematics without affecting the results of those sections, which only depend on the low-
 129 frequency invasion behavior of Eq. (4). 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
 132 x_i . We assume that x_i follows a Poisson distribution $p_i(x_i) = l_i^{x_i} e^{-l_i} / x_i!$, where $l_i = m_i / U$ is the
 mean territorial propagule density. This only approximates uniform random dispersal, but is
 135 essentially exact provided that the n_i are large enough that drift can be ignored (Appendix A).

When multiple propagules land on the same territory, the victor is determined by lottery
 competition: genotype i wins a territory with probability $c_i x_i / \sum_j c_j x_j$, where c_i is a constant
 138 representing relative competitive ability (Fig. 2).

In the classic lottery model (Chesson and Warner, 1981), unoccupied territories are assumed

to be saturated with propagules from every genotype $l_i \gg 1$. From the law of large numbers,
 141 the composition of propagules in each territory will then not deviate appreciably from the mean
 composition l_1, l_2, \dots, l_G (G is the number of genotypes present), and so the probability that geno-
 type i wins any particular unoccupied territory is approximately $c_i l_i / \sum_j c_j l_j$. Let $\Delta_+ n_i$ denote the
 144 number of territories won by genotype i . Then $\Delta_+ n_1, \Delta_+ n_2, \dots, \Delta_+ n_G$ follow a multinomial dis-
 tribution with U trials and success 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
 147 outcome are small (since T is large by assumption), giving

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

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

150 There is a close connection between the classic lottery model and the Wright-Fisher model of
 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
 153 w is relative fitness and the n_i are genotype abundances in the preceding generation. Population
 size N remains constant. This is mathematically equivalent to the classic lottery model with non-
 overlapping generations ($d_i = 1$ for all i) and $w_i = b_i c_i$. Thus, the classic lottery model allows
 156 us to replace the abstract Wright-Fisher relative fitnesses w_i 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
 159 (after appropriate extensions are made), in an otherwise very similar model to the canonical
 Wright-Fisher. We therefore expect that drift in our extended lottery model should be similar to
 that in the Wright-Fisher model, but we leave this for future work.

162 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,
 165 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 small l_i in the classic lottery model.
 168 In other words, deviations from the mean propagule composition l_1, l_2, \dots, l_G are important at
 low density.

We expect that a fraction $p_1(x_1) \dots p_G(x_G)$ of the U unoccupied territories will have the
 171 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 (due to our no-drift, large T , large n_i approximation),
 genotype i 's territorial acquisition is given by

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

174 in our extended lottery model, where the sum only includes territories with at least one propag-
 ule present. Note that unlike the classic lottery model, not all unoccupied territories are claimed
 each iteration, since under Poisson dispersal a fraction e^{-L} remain unoccupied.

177 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

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

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

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.

Similarly to the high- l_i approximation of classic lottery model, we replace the x_i with appropriate mean values, although we cannot simply replace x_i with l_i . For a genotype with low propagule density $l_i \ll 1$, we have $x_i = 1$ in the territories where its propagules land, and so its growth comes entirely from territories which deviate appreciably from l_i . To account for this, we separate Eq. (2) into $x_i = 1$ and $x_i > 1$ parts. Our more general mean field approximation only requires that there are no large discrepancies in competitive ability (i.e. we do not have $c_i/c_j \gg 1$ for any two genotypes). We obtain (details in Appendix B)

$$\Delta_+ n_i(t) \approx b_i n_i \left[e^{-L} + (R_i + A_i) \frac{c_i}{\bar{c}} \right], \quad (4)$$

where

$$R_i = \frac{\bar{c} e^{-l_i} (1 - e^{-(L-l_i)})}{c_i + \frac{L-1+e^{-L}}{1-(1+l_i)e^{-L}} \frac{\bar{c} L - c_i l_i}{L-l_i}}, \quad (5)$$

and

$$A_i = \frac{\bar{c}(1 - e^{-l_i})}{\frac{1-e^{-l_i}}{1-(1+l_i)e^{-L}} c_i l_i + \frac{1}{L-l_i} \left(L \frac{1-e^{-L}}{1-(1+L)e^{-L}} - l_i \frac{1-e^{-l_i}}{1-(1+l_i)e^{-L}} \right) \sum_{j \neq i} c_j l_j}. \quad (6)$$

To supplement our analytical mean field derivation, we did numerical simulations of our exact density-dependent lottery model, and verified that Eq. (4) is a good approximation (Appendix B). Thus, Eq. (4) describes how type abundances change over time in a lottery model where population density can itself vary with time.

Comparing Eq. (4) 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. (5), $R_i \rightarrow 0$ 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 $A_i \rightarrow 1/L$).

Primary strategies and Grime's triangle

Here we describe how Grime's "disturbed", "stressful" and "ideal" environments can be captured mathematically in our scheme. To proceed, we map these verbally-defined environments to quantitative parameter regimes in our model.

Ideal environments are characterized by the near-absence of stress and disturbance, so that survival is easy $d \ll 1$ and growth and reproduction can be rapid $b \gg d$. The propagule density is determined by the magnitude of b/d , and in this case $L \gg 1$ (Appendix C).

Disturbed environments are characterized by short bursts of high extrinsic mortality d caused by physical destruction. 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 type i each iteration. In the heavily disturbed extreme of Grime's triangle, many such bursts (which could each be spatially localized) will occur each iteration (i.e. over the time required for a propagule to reach reproductive maturity), which we approximate as almost all adults and juveniles dying every iteration $d \approx 1$. Population persistence is only possible if $(1 - d)b > d$, and so $b \gg 1$ (Appendix C). We assume that fulfilling this requirement presents such a challenge that $(1 - d)b$ does not greatly exceed d , which implies $L \ll 1$.

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 $b \ll 1$. In Grime's view, this means that

	Ideal	Disturbance*	Stress (G)	Stress (HD)
Constraints	$d \ll 1$	$d \approx 1$	$b \ll 1$	$b \ll 1$
Other parameters	$b/d \gg 1$	$(1 - d)b \approx d$	$b \approx d$	$b/d \gg 1$
Density L	$\gg 1$	$\ll 1$	$\ll 1$	$\gg 1$

Figure 3: The mathematical realization of Grime’s three environmental extremes in our model, as well as the high-density variant of the stressful environment. *Mortality affects both adults and juveniles in the disturbed environment, with $\Delta_+ n_i$ replaced by $(1 - d_i)\Delta_+ n_i$.

the rate at which propagules successfully develop to adulthood cannot appreciably exceed the mortality rate $b/d \approx 1$; coping with the environmental stress is barely possible (Grime, 1974, 1977). In our model, this implies $L \ll 1$.

The alternative view is that stressed environments are highly competitive. In spite of inhibited growth and reproduction, density is actually high relative to carrying capacity of the stressful environment (Taylor et al., 1990). For example, if consumable resources are scarce, we expect intense resource competition (Davis et al., 1998). Thus, even though $b \ll 1$, we can still have $b/d \gg 1$ and $L \ll 1$.

Given our mathematical representation of Grime’s environmental extremes, summarized in Fig. 3, the expected direction of evolution is determined by three factors. First, the probability that an adaptive mutant survives its initial low abundance phase and begins to grow deterministically is proportional to its expected absolute growth rate (Haldane, 1927; Uecker and Hermisson, 2011). By comparison, neutral mutations rarely fix (probability of order $1/N$). Thus b , c and d will tend to change in the direction of greatest fitness increase. Under Eq. (4), proportional changes of the form $b_j = (1 + \epsilon)b_i$ or $d_j = (1 + \epsilon)d_i$ have identical fitness effects

When propagule density is low $L \ll 1$, c does not contribute to fitness.

Second, the fact that some mutations are more common than others can change the direction of evolution (Yampolsky and Stoltzfus, 2001), and in particular, very few mutations can perform difficult trait changes such as increasing b under stress. These mutational constraints are different under different environments (Fig. 3).

Finally, there may be pleiotropy between b , c , and d , which could be due to trade-offs between

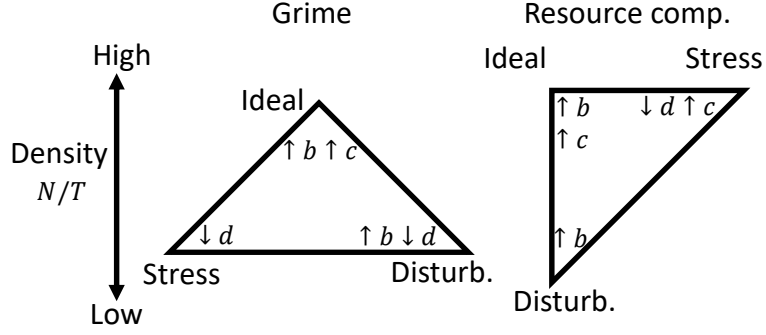


Figure 4: The realization of Grime’s triangle in our model. Schematic representation of the triangular space bounded by the low/high extremes of stress/disturbance. The low- T interpretation of stress is also shown. The vertices of the triangles correspond to different environmental extremes. Selection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

them. Here we do not attempt to account for the complications posed by pleiotropy, and treat b , c and d independently.

Following Grime’s original argument for a triangular scheme (Grime, 1977), Fig. 4 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 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”.

Coexistence in constant and cyclical environments

In the previous section we only considered how b , c and d should respond in Grime’s environmental extremes. Here we further explore the low frequency behavior of Eq. (4) to determine which types can coexist in a constant environment, and then consider the full time-dependent behaviour of Eq. (4) in a cyclical environment.

In a population with a single genotype i which is in equilibrium, $R_i = 0$, $\bar{c} = c_i$ and $\Delta n_i = 0$,

and Eq. (4) gives

$$b_i \left(e^{-L} + A_i \right) - d_i = 0, \quad (7)$$

267 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. (4), n_j will increase if

$$b_j \left(e^{-L} + R_j \frac{c_j}{c_i} \right) - d_j > 0, \quad (8)$$

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

Now suppose that j is better at securing territories ($c_j > c_i$), that i is better at producing propagules ($b_i > b_j$), with $d_i = d_j$. Coexistence occurs if j will invade an i -dominated population, 273 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. (7) and (29), we find that i invades j because $b_i > b_j$, while j invades i provided that

$$b_j c_j R_j - b_i c_i A_i > 0. \quad (9)$$

276 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 279 and low- d specialists; a “competition-longevity” trade-off (Tilman, 1994). These forms of coexistence require density dependence, and are not present in the classic lottery model. Mutual 282 invasion is not possible between b - and d -specialists.

Now suppose that birth and death rates vary periodically with amplitude sufficient to cause large changes in population density. This example is inspired by natural *Drosophila* populations, 285 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
288 potentially millions of years old, suggesting stable coexistence (Bergland et al., 2014; Messer
et al., 2016). Selection on allele frequencies thus occurs on the same time scale as population
demography, a situation vastly more complicated than classical sweeps in demographically stable
291 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
294 relative fitness values for each environmental iteration, and so two alleles favored in different en-
vironments can only stably coexist if the product of fitnesses for one type exactly equals the prod-
uct for the other (Dempster, 1955). Thus, stable coexistence still requires frequency-dependent
297 selection or heterozygote advantage (as is required in a constant environment).

This classical argument overlooks two general effects that promote coexistence in fluctuating
environments. The first is the storage effect, which occurs when part of the population is pro-
300 tected from selection (due to overlapping generations in the lottery model; Chesson and Warner
1981). The second is the bounded population size effect of Yi and Dean (2013), which occurs
when each environmental cycle involves growth from low to high density, with the time spent
303 growing each cycle dependent on the fitness of the types present.

Fig. 5a-c shows the behavior of Eq. (4) for an example where b and d cycle between zero and
positive values (“summers” with rapid growth and no mortality, and “winters” with mortality
306 and no growth). Both the storage effect (adults are sheltered from selection during the summer
growth phase) and the bounded density effect (expansion to high density occurs every cycle)
are operating. Two types are present, a b specialist, which is better at rapidly growing in the
309 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. This is due to a
combination of the storage and bounded density effects (recall that stable coexistence between b
312 and d specialists was not possible in a constant environment).

The classic lottery model (Eq. 1) gives dramatically different co-existence predictions because

expansion to carrying capacity occurs immediately at the start of the summer (Fig. 5d-f). As
 315 a result, coexistence requires that the winter survivor's b must be about 5 times smaller than
 predicted when we properly account for the growth in the abundance of each type using Eq. (4)
 (keeping the other parameters the same). Previous models of the promotion of genetic variation
 318 via the storage effect (Ellner and Hairston Jr, 1994) similarly assume that the total number of
 offspring per iteration is constant, and would produce a similar error.

Discussion

321 In the introduction we mentioned the recurring difficulties with confounding selection and de-
 mography in population genetic inference. While we have not directly attempted to perform
 inference with our model, and Eq. (4) may not be appropriate for particular inference problems,
 324 it seems that something similar (and hopefully more analytically tractable) to our extension of
 the lottery model is unavoidable because, fundamentally, selective births and deaths affect both
 abundances and frequencies, not one or the other in isolation. Moreover, some aspects of al-
 327 lele frequency change are intrinsically density-dependent. In the classic lottery model, which as
 we have seen is essentially the Wright-Fisher model with overlapping generations, b_i and c_i are
 equivalent in the sense that the number of territorial victories only depends on the product $b_i c_i$
 330 (see "Model"). This is no longer the case in our extension, 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
 333 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 sophis-
 ticated treatment of space (non-uniform dispersal; Bolker and Pacala 1999; Shmida and Ellner
 336 1984). In cyclical environments, polymorphisms can be stabilized by the bounded density effect
 which is completely lost if there is an exclusive focus on allele frequencies (Yi and Dean, 2013).
 We leave the details of how our model might be applied in inference, including the crucial issue

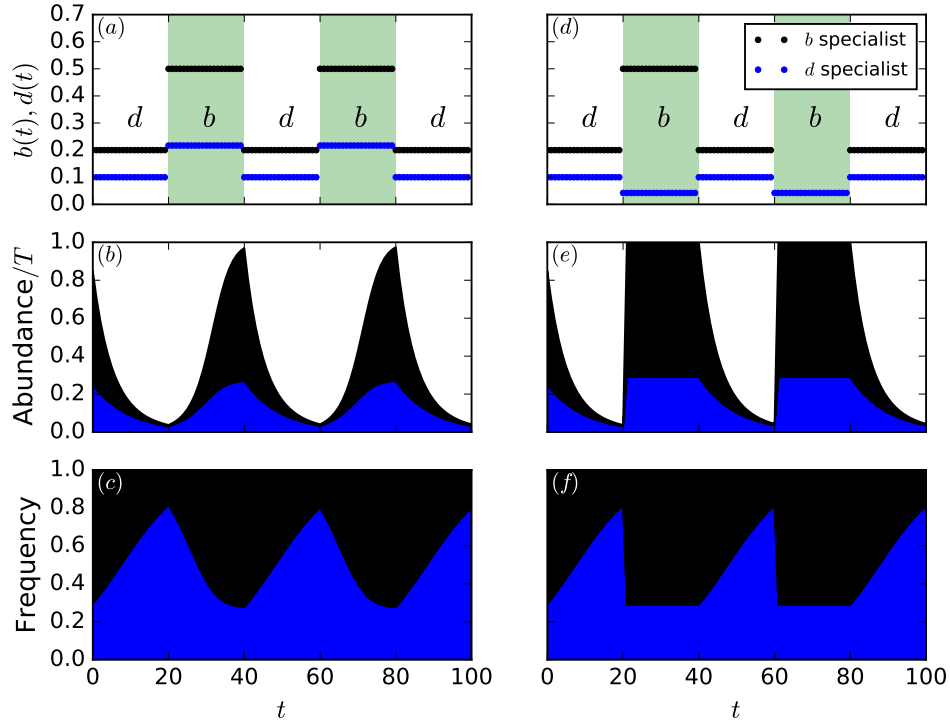


Figure 5: Stable coexistence between b and d specialists in a fluctuating environment. (a) Birth and death rates seasonally alternate being nonzero (white for winter, green for summer). The b specialist (black) has higher b and d ($b = 0.5, d = 0.2$) than the d specialist ($b = 0.217, d = 0.1$) (blue). (b) Both types grow during the positive b phase, and decline during the positive d phase, but the d specialist does so at a lower rate. Total height (blue+black) is population density N/T . (c) Summer favors the b specialist, winter the d specialist, and they stably coexist. (d-f) Same as (a-c) for the classic lottery model, where coexistence now requires $b \approx 0.0421$ for the d specialist. For illustration, the propagule abundances are assumed to have the form $m_i = b_i(1 - N/T)n_i$, reflecting non-directed dispersal.

of its genetic drift predictions, for future work.

It is interesting to compare the predictions of the extended lottery model with earlier approaches, such as the r/K scheme, where $r = b - d$ is the maximal, low-density growth rate (Pianka, 1972). Confusingly, the term “ K -selection” sometimes refers generally to selection at high density (Pianka, 1972), encompassing both selection for higher saturation density (MacArthur and Wilson, 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 positively correlated, both between species/strains (Fitzsimmons et al., 2010; Hendriks et al., 2005; Kuno, 1991; Luckinbill, 1979), and as a result of experimental evolution (Luckinbill, 1978, 1979). From the perspective of our model, this correlation is not surprising since the saturation density, 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 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 “ α -selection” after the competition coefficients in the Lotka-Volterra equation (Case and Gilpin, 1974; Gill, 1974; Joshi et al., 2001). However, competitive success as measured by α (i.e. the per-capita effect of one genotype on another genotype’s growth rate) is only partly determined by individual competitive ability — in the presence of age-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 (the total number of territories occupied per iteration is $\Delta_+ N = U(1 - e^{-L})$, which does not depend directly on the c_i). 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.

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 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 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 (Darling et al., 2012). Fig. 4 gives the same conclusion without invoking trade-offs; mutations which reduce disturbance vulnerability are essentially neutral under ideal conditions, leading to no improvements in mortality from disturbances, whereas c will tend to increase over time. 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.

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

393 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
396 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
399 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
402 (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
405 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).

408 While our model can be applied to species rather than genotypes (e.g. ecological invasions),
our focus is genotype evolution i.e. the change in allele frequencies over time. Our assumption
that there are no large c discrepancies (section “Mean field approximation”) amounts to a restric-
411 tion 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.
414 However, this restriction could become important when looking at species interactions rather
than genotype evolution.

References

- 417 Aerts, R. 1999. Interspecific competition in natural plant communities: mechanisms, trade-offs
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.
- 420 Arenbaev, N. 1977. Asymptotic behavior of the multinomial distribution. *Theory of Probability
& Its Applications* 21:805–810.
- Barton, N. H. 1998. The effect of hitch-hiking on neutral genealogies. *Genetical Research* 72:123–
423 133.
- Bergland, A. O., E. L. Behrman, K. R. O'Brien, P. S. Schmidt, and D. A. Petrov. 2014. Genomic
evidence of rapid and stable adaptive oscillations over seasonal time scales in *Drosophila*. *PLOS*
426 *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*
429 153:575–602.
- Boyce, M. S. 1984. Reconstitution of r- and k-selection as a model of density-dependent natural
selection. *Annual Review of Ecology and Systematics* 15:427–447.
- 432 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
435 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.

- 438 Davis, M. A., K. J. Wragg, 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.
- 441 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
444 linkage on positive selection. *Genetics* 176:1759–1798.
- Diekmann, O., et al. 2004. A beginner’s guide to adaptive dynamics. *Banach Center Publications* 63:47–86.
- 447 Ellner, S., and N. G. Hairston Jr. 1994. Role of overlapping generations in maintaining genetic variation in a fluctuating environment. *The American Naturalist* 143:403–417.
- Ewens, W. J. 2004. *Mathematical Population Genetics 1: Theoretical Introduction*, vol. 27.
450 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*
453 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.
- 456 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.
- 459 ———. 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 —
origins, implications and tests, pages 371–393. Springer Netherlands, Dordrecht.

Haldane, J. B. S. 1927. A mathematical theory of natural and artificial selection, part v: selection
and mutation 23:838–844.

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
stressors. *Environmental Toxicology and Chemistry* 24:2267–2277.

Joshi, A., N. Prasad, and M. Shakarad. 2001. K-selection, α -selection, effectiveness, and tolerance
in competition: density-dependent selection revisited. *Journal of Genetics* 80:63–75.

Kuno, E. 1991. Some strange properties of the logistic equation defined with r and K : Inherent
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.

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-*
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.

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
evolution? *Trends in Genetics* 32:408–418.

Metz, J. A., R. M. Nisbet, and S. A. Geritz. 1992. How should we define 'fitness' for general ecological scenarios? *Trends in Ecology & Evolution* 7:198–202.

486 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.

Ram, Y., E. Dellus-Gur, M. Bibi, U. Obolski, J. Berman, and L. Hadany. 2016. Predicting microbial
489 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 regulation in life-history evolution. *Ecology* 83:1509–1520.

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

Shmida, A., and S. Ellner. 1984. Coexistence of plant species with similar niches. *Vegetatio*
495 58:29–55.

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

498 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 polymorphism in temporally and spatially heterogeneous environments. *Theoretical Population Biology* 99:76 – 97.
501

Taylor, D. R., L. W. Aarssen, and C. Loehle. 1990. On the relationship between r/k selection and
504 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.

- 507 ———. 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.
- 510 Uecker, H., and J. Hermisson. 2011. On the fixation process of a beneficial mutation in a variable environment. *Genetics* 188:915–930.
- Violle, C., M.-L. Navas, D. Vile, E. Kazakou, C. Fortunel, I. Hummel, and E. Garnier. 2007. Let
513 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* 199:213–227.
- 516 Winemiller, K. O., D. B. Fitzgerald, L. M. Bower, and E. R. Pianka. 2015. Functional traits, convergent evolution, and periodic tables of niches. *Ecology Letters* 18:737–751.
- Winemiller, K. O., and K. A. Rose. 1992. Patterns of life-history diversification in north american
519 fishes: implications for population regulation. *Canadian Journal of Fisheries and Aquatic Sciences* 49:2196–2218.
- Yampolsky, L. Y., and A. Stoltzfus. 2001. Bias in the introduction of variation as an orienting
522 factor in evolution. *Evolution & Development* 3:73–83.
- 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.

525 **Appendix A: Poisson approximation**

For simplicity of presentation, we have assumed a Poisson distribution for the x_i as our model of dispersal. Strictly speaking, the total number of i propagules $\sum x_i$ (summed over unoccupied
528 territories) is then no longer a constant m_i , but fluctuates between generations for a given mean

m_i , which is more biologically realistic. Nevertheless, since we do not consider the random fluctuations in type abundances here, and for ease of comparison with the classic lottery model, we ignore the fluctuations in m_i . Instead we focus, on Poisson fluctuations in propagule composition in each territory.

In the exact model of random dispersal, the counts of a genotype's propagules across unoccupied territories follows a multinomial distribution with dimension U , total number of trials equal to m_i , and equal probabilities $1/U$ for a propagule to land in a given territory. Thus, the x_i in different territories are not independent random variables. However, for sufficiently large U and m_i , 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 (Arenbaev, 1977, Theorem 1). Since we are ignoring finite population size effects, we effectively have $T \rightarrow \infty$, in which case U can be only be small enough to violate the Poisson approximation if there is vanishing population turnover, and then the dispersal distribution is irrelevant anyway. Likewise, in ignoring stochastic finite population size for the n_i , we have effectively already assumed that m_i is large enough to justify the Poisson approximation (the error scales as $1/\sqrt{m_i}$; Arenbaev 1977).

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

$$\Delta_u n_i = U l_i e^{-L} = m_i e^{-L}. \quad (10)$$

Competition when rare

555 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
558 is $U p_i(1) P(X_i \geq 1) = b_i n_i e^{-l_i} (1 - e^{-(L-l_i)})$. Thus

$$\Delta_r n_i = m_i e^{-l_i} (1 - e^{-(L-l_i)}) \left\langle \frac{c_i}{c_i + \sum_{j \neq i} c_j x_j} \right\rangle_{\tilde{p}}, \quad (11)$$

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
561 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. (11),

$$\left\langle \frac{c_i}{c_i + \sum_{j \neq i} c_j x_j} \right\rangle_{\tilde{p}} \approx \frac{c_i}{c_i + \sum_{j \neq i} c_j \langle x_j \rangle_{\tilde{p}}}. \quad (12)$$

Below we justify this replacement by arguing that the standard deviation $\sigma_{\tilde{p}}(\sum_{j \neq i} c_j x_j)$ (with
564 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

$$\begin{aligned}\tilde{p}(\mathbf{x}_i) &= p(\mathbf{x}_i | X \geq 2, x_i = 1) \\ &= \frac{P(\mathbf{x}_i, X \geq 2 | x_i = 1)}{P(X \geq 2)} \\ &= \frac{1}{1 - (1 + L)e^{-L}} \sum_{X=2}^{\infty} P(X) p(\mathbf{x}_i | X_i = X - 1),\end{aligned}\tag{13}$$

and so

$$\begin{aligned}\langle x_j \rangle_{\tilde{p}} &= \sum_{\mathbf{x}_i} \tilde{p}(\mathbf{x}_i) x_j \\ &= \frac{1}{1 - (1 + L)e^{-L}} \sum_{X=2}^{\infty} P(X) \sum_{\mathbf{x}_i} p(\mathbf{x}_i | X_i = X - 1) x_j.\end{aligned}\tag{14}$$

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,

$$\begin{aligned}\langle x_j \rangle_{\tilde{p}} &= \frac{l_j}{1 - (1 + L)e^{-L}} \frac{1}{L - l_i} \sum_{k=2}^{\infty} P(X)(X - 1) \\ &= \frac{l_j}{1 - (1 + L)e^{-L}} \frac{L - 1 + e^{-L}}{L - l_i},\end{aligned}\tag{15}$$

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 $\langle x_j \rangle_{\tilde{q}} = \langle x_j \rangle_p / C = l_j / C$,

$$\begin{aligned}
\sigma_{\tilde{q}}^2(x_j) &= \langle x_j^2 \rangle_{\tilde{q}} - \langle x_j \rangle_{\tilde{q}}^2 \\
&= \frac{1}{C} \langle x_j^2 \rangle_p - \frac{l_j^2}{C^2} \\
&= \frac{1}{C} (l_j^2 + l_j) - \frac{l_j^2}{C^2} \\
&= \frac{l_j^2}{C} \left(1 - \frac{1}{C} \right) + \frac{l_j}{C},
\end{aligned} \tag{16}$$

and

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

567 where $C = 1 - e^{-(L-l_i)}$ and $j \neq k$.

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 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 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. (15) correctly predicts that there are on average $\sum_{j \neq i} \langle x_j \rangle_{\tilde{p}} \approx L - 1$ 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

573
576
579

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.

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

$$\sigma_{\tilde{q}}^2(\sum_{j \neq i} c_j x_j) = \sum_{j \neq i} \left[c_j^2 \sigma_{\tilde{q}}^2(x_j) + 2 \sum_{k > j, k \neq i} c_j c_k \sigma_{\tilde{q}}(x_j, x_k) \right], \quad (18)$$

and using the fact that $\sigma_{\tilde{q}}(x_j, x_k)$ and the first term in Eq. (16) are negative because $C < 1$, we obtain an upper bound on the relative fluctuations in $\sum_{j \neq i} c_j x_j$,

$$\frac{\sigma(\sum_{j \neq i} c_j x_j)}{\langle \sum_{j \neq i} c_j x_j \rangle} = C^{1/2} \frac{\left(\sum_{j \neq i} c_j^2 l_j + (1 - 1/C) \left(\sum_{j \neq i} c_j l_j \right)^2 \right)^{1/2}}{\sum_{j \neq i} c_j l_j} < C^{1/2} \frac{\left(\sum_{j \neq i} c_j^2 l_j \right)^{1/2}}{\sum_{j \neq i} c_j l_j}. \quad (19)$$

585 Suppose that the c_j are all of similar magnitude (their ratios are of order one). Then Eq. (19) 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 $1/\sqrt{L - l_i}$).
 588 The worst case scenario occurs when $L - l_i$ is of order one. Then Eq. (19) 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. 6) confirm that the relative errors are indeed
 591 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. (19) can be large and we
 594 cannot make the replacement Eq. (12).

Substituting Eqs. (12) and (15) into Eq. (11), we obtain

$$\Delta_r n_i \approx m_i R_i \frac{c_i}{\bar{c}}, \quad (20)$$

597 where R_i is defined in Eq. (5).

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. (11), we have

$$\Delta_a n_i = U(1 - (1 + l_i)e^{l_i}) \left\langle \frac{c_i x_i}{\sum_j c_j x_j} \right\rangle_{\hat{p}} \quad (21)$$

where \hat{p} is the probability distribution of both focal and nonfocal propagule 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 to \hat{p}), so that,

$$\left\langle \frac{c_i x_i}{\sum_j c_j x_j} \right\rangle_{\hat{p}} \approx \frac{c_i \langle x_i \rangle_{\hat{p}}}{\sum_j c_j \langle x_j \rangle_{\hat{p}}}. \quad (22)$$

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,

$$\begin{aligned} \langle x_i \rangle_{\hat{p}} &= \sum_{\mathbf{x}} x_i p(\mathbf{x} | x_i \geq 2) \\ &= \sum_{x_i} x_i p(x_i | x_i \geq 2) \\ &= \frac{1}{1 - (1 + l_i)e^{-l_i}} \sum_{x_i \geq 2} p(x_i) x_i \\ &= l_i \frac{1 - e^{-l_i}}{1 - (1 + l_i)e^{-l_i}}. \end{aligned} \quad (23)$$

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

$$\begin{aligned}
\langle x_j \rangle_{\hat{p}} &= \sum_{\mathbf{x}} x_j p(\mathbf{x} | x_i \geq 2) \\
&= \sum_X P(X | x_i \geq 2) \sum_{\mathbf{x}} x_j p(\mathbf{x} | x_i \geq 2, X) \\
&= \sum_X P(X | x_i \geq 2) \sum_{x_i} p(x_i | x_i \geq 2, X) \sum_{\mathbf{x}_i} x_j p(\mathbf{x}_i | X_i = X - x_i) \\
&= \sum_X P(X | x_i \geq 2) \sum_{x_i} p(x_i | x_i \geq 2, X) \frac{l_j(X - x_i)}{L - l_i} \\
&= \frac{l_j}{L - l_i} \left[\sum_X P(X | x_i \geq 2) X - \sum_{x_i} p(x_i | x_i \geq 2) x_i \right] \\
&= \frac{l_j}{L - l_i} \left(L \frac{1 - e^{-L}}{1 - (1 + L)e^{-L}} - l_i \frac{1 - e^{-l_i}}{1 - (1 + l_i)e^{-l_i}} \right). \tag{24}
\end{aligned}$$

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

$$\sigma_{\hat{q}}^2(x_i) = \frac{l_i}{D} \left(l_i + 1 - e^{-l_i} - \frac{l_i}{D} (1 - e^{-l_i})^2 \right), \tag{25}$$

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

$$\frac{\sigma_{\hat{q}}(\sum c_j x_j)}{\langle \sum c_j x_j \rangle} = \frac{\left(\sum_{j \neq i} c_j^2 l_j + c_i^2 \sigma_{\hat{q}}^2(x_i) \right)^{1/2}}{\sum_{j \neq i} c_j l_j + c_i l_i (1 - e^{-l_i}) / D}. \tag{26}$$

Similarly to Eq. (19), the RHS of Eq. (26) 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. (26) is around 35%, which is again where the \hat{q} approximation produces the biggest overestimate of the fluctuations in \mathbf{x} . Similarly

615 to Eq. (19), the RHS of (26) will not be $\ll 1$ in the presence of a rare, extremely strong competitor.

Combining Eqs. (21) and (22), we obtain

$$\Delta_a n_i = m_i A_i \frac{c_i}{\bar{c}}, \quad (27)$$

where A_i is defined in Eq. (6).

618 **Comparison with simulations**

Fig. 6 shows that Eq. (4) and its components closely approximate our density-dependent lottery model over a wide range of propagule densities (the latter is evaluated by direct simulations of
621 uniform random dispersal and lottery competition). Two genotypes are present, one of which 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 $A_i c_i / \bar{c}$
624 if l_1 is large enough (Fig. 6b). 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. 6d). Fig. 3 also shows the breakdown of the classic lottery model at low propagule densities.

627 **Appendix C: Direction of evolution under different environmental extremes**

In this Appendix we consider the invasion of mutants into a population with a single resident
630 type i in equilibrium under the different environments from section “Primary strategies and Grime’s triangle”. We first evaluate the ideal and stressful environments (the treatment of mortality is different for disturbed environments).

633 Summing Eq. (2) over genotypes, the change in N is given by

$$\Delta N = U(1 - e^{-L}) - \sum_i d_i n_i, \quad (28)$$

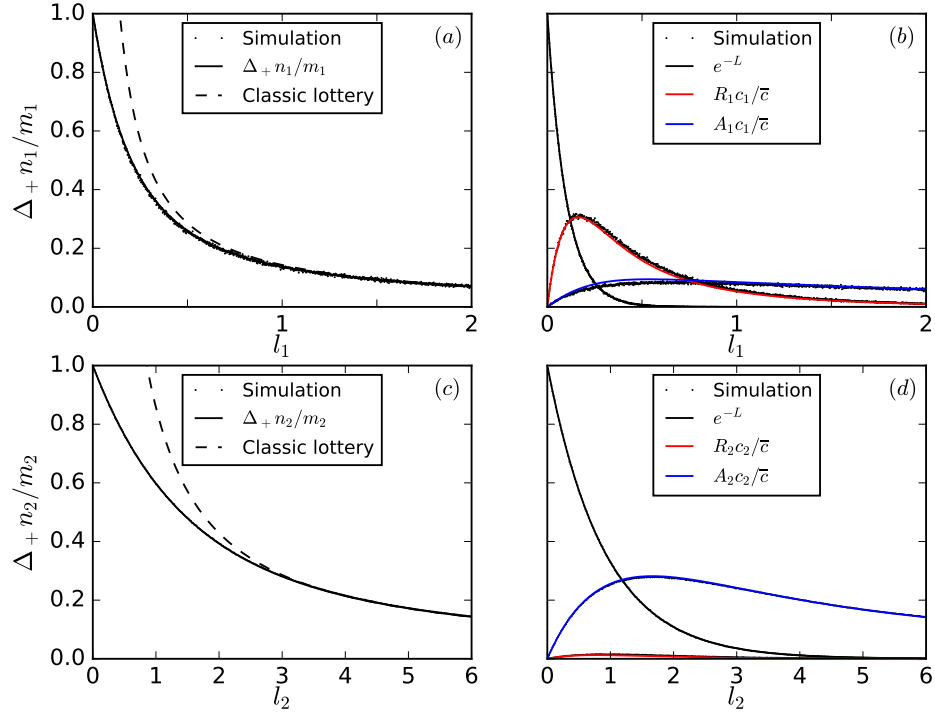


Figure 6: The change in genotype abundances in a density dependent lottery model is closely approximated by Eq. (4). $\Delta_+ n_i / m_i$ from Eq. (4) (and its separate components) are shown, along with direct simulations of random dispersal and lottery competition over one iteration over a range of propagule densities (U is varied between 5×10^3 and 10^6 with $m_1 = 10^4$ and $m_2 = 9 \times 10^4$). Two genotypes are present. (a) and (b) show the low-frequency genotype with c -advantage ($c_1 = 1.5$), (c) and (d) show the high-frequency predominant genotype ($c_2 = 1$). Simulation points are almost invisible in (c) and (d) due to near exact agreement with Eq. (4). Dashed lines in (a) and (c) show the breakdown of the classic lottery model.

where $U = \sum_i b_i n_i / L$. In equilibrium ($\Delta N = 0$), this implies $L/(1 - e^{-L}) = b_i/d_i$. The fitness of a low frequency invader j is

$$\Delta n_j / n_j \approx b_j (e^{-L} + R_j c_j) - d_j \quad (29)$$

636 where $R_j \approx (1 - e^{-L}) / (c_j + \frac{L-1-e^{-L}}{1-(1+L)e^{-L}})$, and we set the reference competitiveness at $c_i = 1$ without loss of generality.

According our representation of Grime's stressful environment, $b \ll 1$, $d \approx b$ and thus
 639 $L \ll 1$. Then Eq. (29) becomes $\Delta n_j / n_j \approx b_j - d_j$. Mutations which improve b or d by the same fraction ϵ (such that $b_j = (1 + \epsilon)b_i$ or $d_j = (1 - \epsilon)d_i$) will therefore yield identical fitness benefits. Stressful environments will therefore favor d -specialist "tolerators", which bring their mortality
 642 rates down enough to enable survival in the face of low b , only if stress makes it harder to increase b than lower d , such that adaptive mutations of a given effect size ϵ occur more often for d .

645 In Grime's ideal environment, d is much smaller than 1 whereas b is not. Thus, $1 \ll b_i/d_i = L/(1 - e^{-L}) \approx L$, and Eq. (29) becomes

$$\Delta n_j / n_j \approx \frac{b_j c_j}{c_j + L - 1} - d_j \approx \frac{b_j c_j}{L} - d_j, \quad (30)$$

where the last approximation follows from the fact that c_j will be of similar magnitude to $c_i = 1$
 648 (ignoring mutations with very large effect sizes; compare with Eq. (1), where the same expression comes from assuming that L is effectively infinite). It is easily verified from Eq. (30) that improving b , c , or d by a fraction ϵ yields identical fitness benefits. This may seem counter-
 651 intuitive in that reducing an already tiny mortality rate by a fraction ϵ seems to yield marginal benefits compared to increasing a potentially large propagule production b by the same fraction. However, the relative magnitudes of b and d are irrelevant, because the ancestral resident type's
 654 density-regulated birth rate $b_i c_i / L$ exactly balances its mortality d_i in equilibrium.

Biases in the direction of evolution of b , c or d must again follow from biases in mutation availability or trade-offs/pleiotropy. The same argument applies for high- L interpretation of the

657 stress regime.

To represent Grime's disturbed environment, we account for juvenile mortality due to disturbance by replacing $\Delta_+ n_i$ with $(1 - d_i)\Delta_+ n_i$. For a single type i in equilibrium, Eq. (28) becomes

$$\Delta N = (1 - d_i)U(1 - e^{-L}) - d_i N, \quad (31)$$

660 which reaches equilibrium when $L/(1 - e^{-L}) = (1 - d_i)b_i/d_i$ (note that population persistence now requires $(1 - d_i)b_i > d_i$). We represent Grime's disturbed environment by $d_i \approx 1$ and $(1 - d_i)b_i \approx d_i$, so that $L \ll 1$. A mutant's invasion fitness is then given by

$$\Delta n_j/n_j = (1 - d_j)b_j - d_j. \quad (32)$$

663 The effects of improving b and d are now no longer identical. Increasing b by a fraction ϵ gives $\Delta n_j/n_j = \epsilon(1 - d_i)b_i$, whereas reducing d by the same fraction gives $\Delta n_j/n_j = \epsilon b_i$, a much greater advantage given that $d_i \approx 1$. Thus, selection intrinsically favors reducing d in disturbed
666 environments.