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Adaptation versus migration in demographically unstable populations

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Abstract. We analyze a model of the joint population and evolutionary dynamics of a diploid "island" population that receives a recurrent influx of immigrants from a hypothetical continent population. We derive a criterion for the initial spread of a rare allele when the island population dynamics initially are chaotic. By computing a Liapunov exponent, we show that this criterion depends on a generalization of the geometric mean absolute fitness of individuals heterozygous for the rare allele. This criterion applies regardless of whether the initial population is self-sustaining or not.

Key words: Population dynamics – Adaptive evolution – Chaos – Ergodic – Liapunov exponent

1. Introduction

A classic problem in evolutionary biology is understanding how organisms adapt to heterogeneous environments (Haldane, 1930; Endler, 1977; Slatkin, 1987). One of the first theoretical treatments of this problem was the "island model" (Haldane, 1930; Wright, 1931), which considers the genetic evolution of a semi-isolated island population that receives a recurrent influx of immigrants from a hypothetical continent population. Recently (Holt & Gomulkiewicz, 1997;

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Gomulkiewicz et al., 1999), the island model has been extended to examine local adaptation in "sink" populations, which are populations of a species that would be unable to persist deterministically if isolated. These more recent treatments consider both population and evolutionary dynamics, and demonstrate that the conditions for initial increase of a rare allele on the island depend on an absolute fitness criterion if the island population size initially is stable or fluctuates periodically. They also show that absolute fitness criteria arise from the fundamental requirement that persistence is necessary for evolution and are of critical importance to the process of evolution in populations that risk extinction. Here we derive a related absolute fitness criterion for initial spread of a rare allele when the island population dynamics initially are chaotic. We also show that the "sink" assumption is not mathematically necessary; the condition for spread of a rare favorable allele in a sink population also applies when the population persists deterministically.

2. Model description

Consider a diploid discrete-breeding, random mating island population that recurrently receives immigrants from a fixed "continent" population. In particular, assume evolution of the island population has no effect on the continent population. We assume viability selection, immigration, and reproduction occur sequentially each generation from zygote stage to zygote stage. Viability (survival from birth to the arrival of immigrants) on the island is determined by an autosomal locus with two alleles A_1 and A_2 and may depend on N_t, the population density in generation t censused immediately after reproduction. We likewise assume that p_t, the frequency of allele A_1, is censused just after reproduction ($0 \leqq p_t \leqq 1$). The viability of individuals with genotype A_iA_j will be denoted by $V_{ij}(N)$ which emphasizes that viability is density dependent. Given the assumption of random mating, the size of the population immediately before the arrival of immigrants will be $N_t\bar{V}(N_t, p_t)$, where $\bar{V}(N_t, p_t) = p_t^2 V_{11}(N_t) + 2p_t(1 - p_t)V_{12}(N_t) + (1 - p_t)^2 V_{22}(N_t)$ is the mean viability. After viability selection, I immigrants arrive on the island, of which all are homozygous for allele A_2. We assume, for simplicity, that post-immigration viability and *per capita* fecundity are genotype independent but may depend on $N_t^* \equiv N_t\bar{V}(N_t, p_t) + I$, the population density immediately after immigration. The combined post-immigration survival and fecundity can therefore be summarized by a single term, $F(N_t, p_t)$. After reproduction, the population density will be

$$N_{t+1} = N_t^* F(N_t, p_t) = [N_t\bar{V}(N_t, p_t) + I]F(N_t, p_t). \tag{1}$$

Because resident and immigrant adults mate at random, zygote frequencies will return to Hardy–Weinberg proportions. The frequency of A_1 among the new zygotes, p_{t+1}, will be equal to the frequency of A_1 among the parents, $p_t^* = [N_t \bar{V}_1(N_t, p_t)/N_t^*]p_t$, where $\bar{V}_1(N_t, p_t) = p_t V_{11}(N_t) + (1 - p_t)V_{12}(N_t)$ is the marginal viability of A_1. Finally, let $W_{ij}(N_t, p_t) = V_{ij}(N_t)F(N_t, p_t)$ and $\bar{W}_1(N_t, p_t) = p_t W_{11}(N_t, p_t) + (1 - p_t) W_{12}(N_t, p_t) = \bar{V}_1(N_t, p_t)F(N_t, p_t)$, which leads to

$$p_{t+1} = \left(\frac{N_t}{N_{t+1}}\right)\bar{W}_1(N_t, p_t)p_t. \tag{2}$$

The recursions (1) and (2) are identical to those derived in (Gomulkiewicz et al., 1999), except that here, we relax the requirement $W_{22}(N) < 1$ for all N which was needed to ensure that the initial island population is a sink regardless of local densities. (Note that this derivation assumes mutation and drift are negligible compared with other evolutionary forces. It also ignores random changes in population size.)

3. Criteria for the spread of A_1

In this section we develop a criterion for the spread of the allele A_1 when it is rare for the model discussed in Sect. 2. This might apply, for example, to the spread of a rare novel allele that has arisen through mutation. Since we are assuming that A_1 is rare we are interested in the dynamics of the system (1) and (2) near $p \equiv 0$. In particular, we wish to determine conditions under which initial conditions with "small" initial values of p (denoted p_0) will increase from $p = 0$ under iteration. Note that if $p_t = 0$ then $p_{t+1} = 0$ and hence the line $p = 0$ is invariant. This suggests the use of a linear stability analysis in determining the desired criteria. Gomulkiewicz et al., (1999) have used this method to develop a criterion for the initial spread of allele A_1 in the case of fixed or periodic population dynamics on $p = 0$. Here we generalize their result to other initial asymptotic population dynamics.

To motivate the techniques used below we will begin by revisiting the results presented in (Gomulkiewicz et al., 1999). Let

$$G_1(N, p) = [N\bar{V}(N, p) + I]F(N, p) \tag{3}$$

and

$$G_2(N, p) = \frac{N}{G_1(N, p)}\bar{W}_1(N, p)p \tag{4}$$

so that the model equations (1) and (2) become

$$\begin{aligned} N_{t+1} &= G_1(N_t, p_t) \\ p_{t+1} &= G_2(N_t, p_t). \end{aligned} \tag{5}$$

In what follows we assume that all functions are $\mathscr{C}^1$. We note in particular that the heterozygote absolute fitness function $W_{12}(N)$ is $\mathscr{C}^1$ and strictly positive.

Theorem 1. *Suppose that there exists a periodic orbit*

$$\{(\hat{N}_0, 0), (\hat{N}_1, 0), \ldots, (\hat{N}_{T-1}, 0), (\hat{N}_0, 0), \ldots\}$$

for (5) *and that*

$$\prod_{t=0}^{T-1} W_{12}(\hat{N}_t) > 1. \tag{6}$$

Then there exists an $\varepsilon > 0$ *such that for every initial condition* (N_0, p_0) *with* $p_0 > 0$ *satisfying* $\sqrt{p_0^2 + (N_0 - \hat{N}_0)^2} < \varepsilon$ *there exists an* $M > 0$ *such that* $p_M > \varepsilon$.

Proof. The stability of periodic orbits is determined by the eigenvalues of the matrix

$$J_T = \prod_{t=0}^{T-1} \begin{pmatrix} \partial G_1/\partial N & \partial G_1/\partial p \\ 0 & N\bar{W}_1(N, p)/G_1(N, p) \end{pmatrix}\Bigg|_{(\hat{N}_t, 0)}. \tag{7}$$

Since each matrix in the above product is upper triangular, the matrix J_T is also upper triangular and the eigenvalues of J_T are the entries of this product on the main diagonal. Moreover, stability transverse to the invariant line $p = 0$ is determined by the eigenvalue λ_p^T in the bottom-right entry of J_T. If $|\lambda_p^T| > 1$ then the periodic orbit is repelling transverse to the line $p = 0$ and the result follows immediately. Using the upper-triangularity of J_T and the periodicity assumption we get

$$\lambda_p^T = \prod_{t=0}^{T-1} \frac{\hat{N}_t}{\hat{N}_{t+1}} \bar{W}_1(\hat{N}_t, 0) = \prod_{t=0}^{T-1} W_{12}(\hat{N}_t) \tag{8}$$

as desired. □

We note that $\prod_{t=0}^{T-1} W_{12}(\hat{N}_t) > 1$ if and only if

$$\left[\prod_{t=0}^{T-1} W_{12}(\hat{N}_t)\right]^{1/T} > 1. \tag{9}$$

Thus, in the case of periodic population dynamics, the rare allele A_1 will initially spread if the geometric mean of the absolute fitness of the heterozygote evaluated along the periodic orbit is greater than one.

Remark 1. *The black-hole sink models considered by Holt and Gomulkiewicz* (1997) *and Gomulkiewicz et al.* (1999) *assume immigrant fitness* ($W_{22}(N)$ *in this paper*) *is less than* 1 *for all* N. *However, this assumption is not necessary for Theorem* 1. *Thus* (9) *is the criterion for spread regardless of whether the initial population is a sink or not.*

We now wish to generalize this result to non-periodic population dynamics on $p = 0$. The standard linear stability analysis arguments used above do not directly apply to non-periodic dynamics. Generalizing the results of Theorem 1 requires the use of **Liapunov exponents**. Liapunov exponents describe the rates of contraction and expansion in the tangent spaces of points along an arbitrary orbit generated by iterating a function f. If f: $\mathbf{R}^n \to \mathbf{R}^n$ then there are n Liapunov exponents. If the orbit is periodic, as in Theorem 1, then the i-th Liapunov exponent is the logarithm of the absolute value of the i-th eigenvalue of the product of the Jacobian matrices evaluated along the periodic orbit divided by the period. The generalization to non-periodic orbits and arbitrary functions f is somewhat technical and will not be presented here. See Guckenheimer and Holmes (1983) or Katok and Hasselblatt (1995) for a detailed discussion of these ideas. Ferriere and Gatto (1995) discuss the application of these ideas in population dynamics.

In our problem the definition of the Liapunov exponent describing stability transverse to the invariant line $p = 0$ is straightforward. As in Theorem 1, transverse stability at $(N, 0)$ is governed by the eigenvalue

$$\lambda_p(N) = \frac{N}{G_1(N, 0)} \bar{W}_1(N, 0). \tag{10}$$

The Liapunov exponent $s_R(\hat{N}_0)$ of the orbit having initial condition $(\hat{N}_0, 0)$ is defined as

$$s_R(\hat{N}_0) = \lim_{T\to\infty} \log\left(\left[\prod_{t=0}^{T-1} \lambda_p(\hat{N}_t)\right]^{1/T}\right) = \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log(\lambda_p(\hat{N}_t)) \tag{11}$$

if this limit exists. This definition and the results of Theorem 1 suggest that

$$s_R(\hat{N}_0) = \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log(W_{12}(\hat{N}_t)) \tag{12}$$

should determine the spread of allele A_1 when the asymptotic population dynamics are not periodic. This is in fact the case as we will prove below.

Ergodic theory guarantees that $s_R(\hat{N}_0)$ exists and *is independent of* $\hat{N}_0$ almost everywhere. For a more complete treatment of ergodic theory and its applications see Ferriere & Gatto (1995); Guckenheimer

and Holmes (1983); Katok & Hasselblatt (1995). We will briefly present the necessary concepts from ergodic theory as they not only justify the use of (12) but also shed some light on the factors that influence invasion.

Let f: $X \to X$ define a discrete dynamical system on a metric space X. A measure μ is **f-invariant** if given a measurable set A, $\mu(A) = \mu(f^{-1}(A))$. A function f having this property is said to be **μ-preserving**. In this notation $f^{-1}(A)$ denotes the set of preimages of A and does not imply that f is necessarily invertible. A measure μ is a **probability** measure on X if $\mu(X) = 1$. An invariant measure μ is **ergodic** if every f-invariant set (i.e. $A = f(A)$) has either measure zero or measure one. When a function f has an ergodic measure μ it is often called ergodic as well. Every continuous function on a compact invariant metric space has at least one ergodic invariant probability measure. We are now ready to discuss the existence of the Liapunov exponent given in (12).

Definition 1. *Let* f: $\mathbf{R}^n \to \mathbf{R}^n$ *define a discrete dynamical system and let* ϕ: $\mathbf{R}^n \to \mathbf{R}$ *be a real-valued function. The* **time average** *of* ϕ along the trajectory of x is

$$\lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \phi(f^t(x)), \tag{13}$$

if this limit exists. Here $f^t(x)$ *denotes the n-fold composition of* f *applied to* x.

Theorem 2 (Birkhoff Ergodic Theorem). *If* f: $X \to X$ *is an ergodic* μ*-preserving transformation,* $\mu(X) = 1$, *and* $\phi \in L_1(X, \mu)$ *then for every* x *outside a set of measure zero*

$$\lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \phi(f^t(x)) = \int_X \phi d\mu. \tag{14}$$

This remarkable theorem says that the time average of ϕ along almost every orbit is equal to the spatial average of ϕ with respect to the measure μ. In other words, given almost any initial condition x and a measurable set A, the orbit of x visits A with frequency $\mu(A)$. This can fail if, for example, the dynamics on X are chaotic, but x lies on a periodic orbit in X. This explains why the equality in the Birkhoff Ergodic Theorem does not hold for a set of points of measure zero. A property that holds everywhere except on a set of measure zero is said to hold *almost everywhere*. If we apply this result to (12) we get the following result.

Theorem 3. *Suppose that there exists a compact invariant set* X *on the line* $p = 0$ *for equations* (5). *Then there exists an ergodic invariant*

probability measure μ on X and the Liapunov exponent describing transverse stability

$$s_R(\hat{N}_0) = \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log(W_{12}(\hat{N}_t)) \tag{15}$$

exists and is constant for almost every $\hat{N}_0 \in X$ with respect to the measure μ.

Moreover, if $s_R(\hat{N}_0) > 0$ then there exists $\varepsilon > 0$ such that if $p_0 > 0$ and the distance between (N_0, p_0) and X is less than ε then there exists an $M > 0$ such that the distance between p_M and X is greater than ε.

Proof. The existence of an ergodic probability measure follows from the continuity of the model equations and the compactness assumption. Let $(\hat{N}_0, 0)$ be an initial condition in X. As in Theorem 1, the Liapunov exponent describing transverse stability depends only on the product of the eigenvalues $\lambda_p(\hat{N}_t)$ of the Jacobian matrix $J(\hat{N}_t, 0)$ and is given by

$$s_R(\hat{N}_t) = \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log\left(\frac{\hat{N}_t}{\hat{N}_{t+1}} W_{12}(\hat{N}_t)\right). \tag{16}$$

By the Birkhoff Ergodic Theorem,

$$\lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log(\hat{N}_t) = \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log\left(\hat{N}_{t+1}\right) = \int_X \log(N)\, d\mu \tag{17}$$

almost everywhere. Since the logarithm of a quotient is the difference of logarithms we get that

$$s_R(\hat{N}_0) = \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log(W_{12}(\hat{N}_t)) \tag{18}$$

almost everywhere as desired. This limit exists if $\log(W_{12}(N)) \in L_1(X, \mu)$, a biologically inert assumption.

The second part of the proof follows immediately from the definition of the Liapunov exponent. □

Remark 2. *An ergodic dynamical system may have multiple invariant ergodic measures and thus the Liapunov exponent may be different for each of these measures. However, with only minor modifications to the above proof, Theorem* 3 *can be strengthened to handle this complication. In particular, there exists a Borel set $U \subset X$ of total probability (i.e. $\mu(U) = 1$ for every ergodic measure μ with support in X) such that $s_R(\hat{N}_0)$ exists and is strictly positive for all $\hat{N}_0 \in U$ and all μ.* (*Schreiber*, 1998).

Note that if the invariant set X is a periodic orbit then Theorem 3 is equivalent to Theorem 1. The criterion for the initial spread of allele

A_1 given in Theorem 3 is, in some sense, a geometric mean criterion where the geometric mean of $W_{12}(N)$ is taken over an infinite set of population sizes. In the next section, we examine some of the biological consequences of this criterion.

4. Numerical results

In both Theorems 1 and 3, the initial spread of the rare allele depends only on the absolute fitness of the heterozygote and the population dynamics. The immigration rate and fitness of the A_2A_2 genotype do not directly affect the spread of A_1. However, these factors do play a role in determining population dynamics and hence indirectly affect adaptation.

To illustrate the indirect dependence of the spread criterion consider the following example. Let the absolute fitness of genotype A_iA_j be given by

$$W_{ij}(N) = \frac{w_{ij}}{1 + N^c}, \tag{19}$$

which incorporates a form of genotype-independent density dependence suggested by Maynard Smith and Slatkin (1973). Note that this form assumes scaled densities. When $p = 0$ the population dynamics are governed by the recursion

$$N_{t+1} = N_t W_{22}(N_t) + I. \tag{20}$$

Let $c = 4.5$, $w_{12} = 3.75$ and $I = 0.05$. We will consider two different values of w_{22} both of which lead to chaotic population dynamics. However, the first will lead to spread of the favorable allele while the second will not.

For the first case let $w_{22} = 3.25$. We numerically approximated the Liapunov exponent to be $s_R = 0.087$ using (12) with a random initial population density and $T = 2000$. Since $s_R > 0$ Theorem 3 implies that A_1 will initially spread when rare. For the second case we let $w_{22} = 4.25$ and using the same method calculated $s_R = -0.204$ implying that A_1 will not spread from rarity. Figure 1 shows typical time series data for N_t in these two cases (case 1 in black and case 2 in gray). Note that both appear to be chaotic and that the only discernible difference in the two trends is that the time series associated with the non-invasion case appears to take on values in a wider interval.

Since the absolute fitness function $W_{12}(N)$ is identical in these two cases, the cause of the different Liapunov exponents must be the invariant measures associated with the two different population

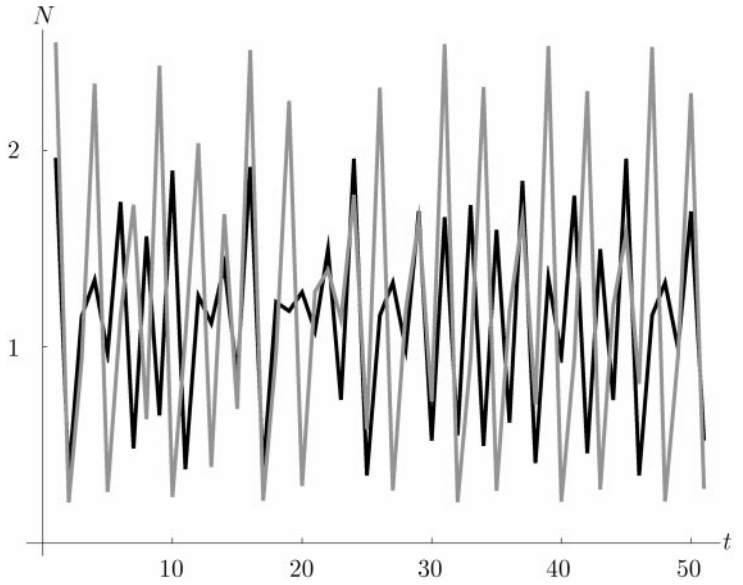

Fig. 1. Plots of population density versus time (equation 1) when $p = 0$ for two different fitnesses $W_{22}(N) = w_{22}/(1 + N^c)$ with $c = 4.5$. The black time series corresponds to case 1 where $w_{22} = 3.25$ and the gray time series corresponds to case 2 where $w_{22} = 4.25$. Note that larger populations occur more frequently in case 2 than in case 1.

dynamics. Figure 2A shows an approximation of the two invariant measures. These were computed by dividing the union of the two invariant intervals into 60 bins, choosing a random initial population size, iterating equation (20) 2000 times and recording the percentage of visits to each bin. This percentage was plotted against the midpoint of each bin. Using this data in juxtaposition with the graph of the logarithm of the absolute fitness function $W_{12}(N)$ shown in Fig. 2B we can see why the Liapunov exponent is greater in case 1 than in case 2. In case one (black in Fig. 2A), a greater percentage of iterates fall in the region where the logarithm of absolute fitness is positive. On the other hand, in case 2 (gray in Fig. 2A), a significant percentage of iterates lie in the region where the logarithm of absolute fitness is negative. Since the Liapunov exponent is approximated by the arithmetic mean of $\log(W_{12}(N))$ over any orbit, these population trends and the form of the absolute fitness determine the value of the Liapunov exponent. In particular, the higher percentage of large population sizes at which the absolute fitness of the heterozygotes is small results in a negative Liapunov exponent and no spread of the favorable allele.

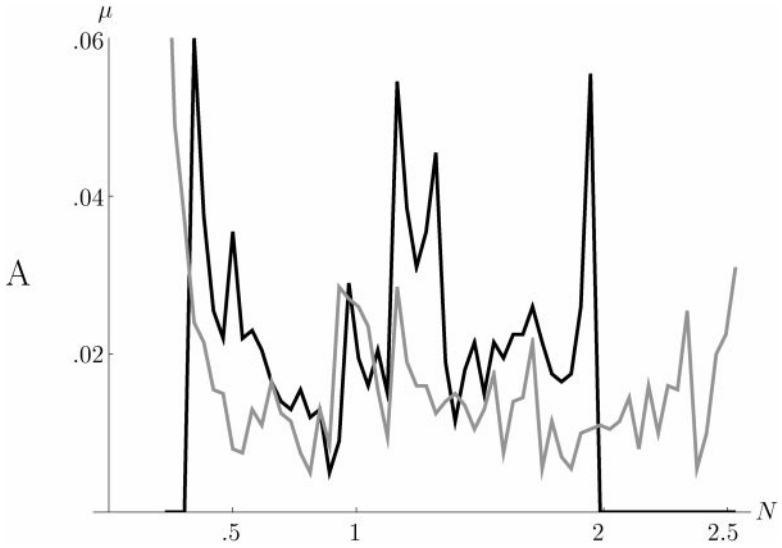

Fig. 2A. Plot of invariant measures for case 1 (black) and case 2 (gray). Using 60 bins and 2000 iterates we computed the frequency of visits to each bin and plotted this frequency against the midpoint of each bin. This is an estimate of the measure of each bin. For a given measure, the asymptotic frequency of visits to a given interval $[a, b]$ is approximately the area under the graph of that measure between a and b. Note that in case 1, population size is bounded above by 2. By contrast, a significant percentage of population sizes greater than 2 appears in case 2.

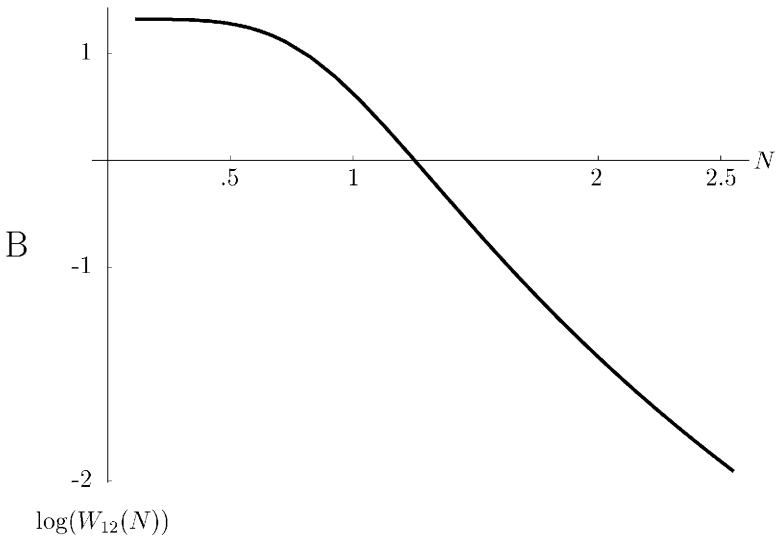

Fig. 2B. The graph of $\log(W_{12}(N)) = \log(w_{12}/(1 + N^c))$ with $w_{12} = 3.75$ and $c = 4.5$. Juxtaposing this graph with the numerically computed invariant measures of case 1 and 2 we see that the large population sizes that occur in case 2 correspond to log absolute fitnesses less than zero and ultimately a negative Liapunov exponent.

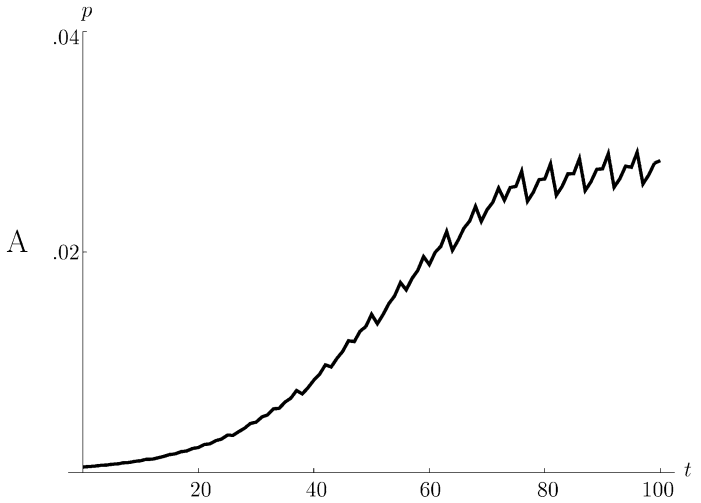

Fig. 3A. A plot of p_t versus t for case 1 generated using the full two-dimensional model. In this example $p_0 = 0.005$ and we see that the frequency of allele A_1 iterates away from zero as predicted by the positive Liapunov exponent computed for this example.

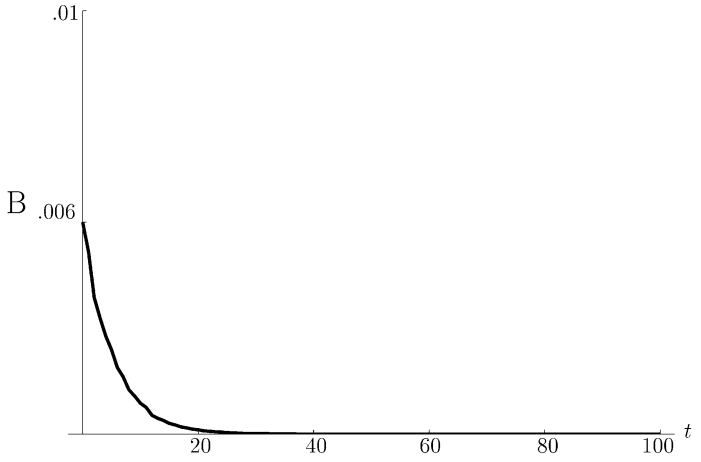

Fig. 3B. A plot of p_t versus t for case 2 generated using the full two-dimensional model. In this example $p_0 = 0.005$ and we see that the frequency of allele A_1 tends to zero as predicted by the negative Liapunov exponent computed for this example.

Figure 3 shows the time course of the frequency of A_1 in the population (p_t) using the full model (5) with the parameters given above and $p_0 = 0.005$. Figure 3A illustrates case 1 where the favorable allele spreads and the time course of p_t can be seen tending away from $p = 0$.

Figure 3B illustrates case 2 and the values of p_t remain small and in fact tend to zero.

One might think that a different initial population size would lead to a different distribution of population sizes over the given orbit and hence a different Liapunov exponent. However, this is not the case. The existence of an *invariant* measure guarantees that this distribution is identical for almost every (in the sense of measure) initial population and hence given almost any initial population density the Liapunov exponent will be the same.

As the above example illustrates, the conditions for spread of the favorable allele do not directly depend on the absolute fitness of the homozygote A_2A_2 or on the immigration rate I. However, these factors do affect the population dynamics, thereby affecting the invariant measure and ultimately the sign of the Liapunov exponent and the eventual spread or decline of the rare allele.

Classical results concerning adaptation versus migration (for example see (Nagylaki, 1992)) suggest that the key criterion for spread of a rare allele depends on *relative fitness* which in our example is $W_{12}(N)/W_{22}(N) = w_{12}/w_{22}$. In particular, it was believed that if the favorable allele did not spread at a given relative fitness then the allele would not spread for all smaller relative fitnesses. However, this model provides a counterexample to this idea. Fix the parameter $c = 0.2$ and the immigration rate $I = 0.05$. Let $w_{12}^1 = 1.43$, $w_{22}^1 = 1$, $w_{12}^2 = 2$, and $w_{22}^2 = 1.5$ so that the relative fitness in case 1 is $w_{12}^1/w_{22}^1 = 1.43$ and the relative fitness in case 2 is $w_{12}^2/w_{22}^2 = 1.33$. In both examples, population size equilibrates when $p = 0$. In case 1 the equilibrium population density is $\hat{N}^1 = 0.1256$ and in case 2 the equilibrium population density is $\hat{N}^2 = 0.3083$. In both cases the Liapunov exponent is given by

$$s_R(\hat{N}^i) = \log(W_{12}(\hat{N}^i)) \tag{21}$$

since the population dynamics approach an equilibrium when $p = 0$. In case 1, $s_R(\hat{N}^1) = -0.15$ implying that the favorable allele does not spread. However, in case 2, which has the smaller relative fitness, $s_R(\hat{N}^2) = 0.11$ implying that the favorable allele will spread. Numerical simulations on the full model confirm the validity of these calculations.

5. Discussion

We have rigorously derived the conditions under which a rare allele will spread initially in a demographically unstable, semi-isolated population that receives a recurrent influx of less favorable alleles. Our model assumes that absolute fitnesses are density dependent and

hence, fitnesses will fluctuate with changing population sizes. In the case of a demographically unstable population, we prove that the criterion for a rare allele to increase in frequency is that the geometric mean absolute fitness of individuals heterozygous for the allele exceeds unity, otherwise the allele will be lost.

Our result extends previous analyses (Holt & Gomulkiewicz, 1997; Gomulkiewicz et al., 1999) which considered local adaptation in demographically stable or cyclic populations. Consistent with those analyses, the criterion for spread we derived depends only indirectly on the rate of immigration, I, and fitness of the immigrant genotype, A_2A_2.

It is interesting to compare our results with classical population genetics treatments of the island model (see for example Haldane (1930); Wright (1931)) which ignore population dynamics and incorporate immigration through a gene flow parameter, which is defined as the proportion of the local population consisting of immigrants. In the classical approach the typical criterion for spread of a rare favorable allele (e.g., (Nagylaki, 1992)) depends on the *relative* fitness of heterozygotes compared to the fitness of immigrant homozygotes in such a way that an allele with higher relative heterozygote fitness will spread under a broader range of conditions than an allele with lower heterozygote fitness. In contrast, our analyses easily revealed conditions under which a rare allele with higher relative heterozygote fitness is lost whereas an allele with lower relative fitness will spread. Apparently, the indirect effects of immigration rate and immigrant fitness on the criterion for spread of a rare allele overlaps only partially with the classical criterion.

Asmussen (1979) derived a geometric mean fitness criterion for the spread of a rare allele in an isolated, cyclic population. Since our results continue to hold when $I = 0$, we have in essence extended Assmussen's results to isolated, chaotic populations.

Unlike the "black-hole sink" scenario analyzed in (Holt and Gomulkiewicz, 1997) and (Gomulkiewicz et al., 1999), our analyses did not assume that the absolute fitness of the immigrant genotype is less than unity for all population sizes N. Thus the absolute fitness criterion for spread is relevant for self-sustaining "source" populations as well as sink populations that persist only by dint of immigration.

In essence, the absolute fitness criterion states the obvious: a rare novel allele can spread only if it allows its possessors to persist. In the context considered here of fluctuating, density-dependent absolute fitnesses, we have proven that persistence of a rare allele is determined by whether or not its geometric mean fitness in heterozygotes exceeds unity. Persistence is generally a moot issue in standard population

genetics, which is concerned by and large with evolution in extant populations. However, our results also apply when considering evolution in populations that face a risk of extinction (a situation that can arise in subdivided populations, including metapopulations, that occupy source and sink habitat). In this situation, the question of persistence in local adaptation is paramount. As stated previously (Holt and Gomulkiewicz 1997; Gomulkiewicz et al., 1999), an absolute fitness criterion for spread implies, among other things, that adaptation in sink environments will be limited by the appearance of alleles that permit persistence. Such alleles are likely to arise only rarely, particularly in harsh environments. Our results indicate that the absolute fitness barrier is a robust constraint on evolution in demographically stable and unstable populations.

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References

Asmussen, M. A. (1979). Regular and chaotic cycling in models of ecological genetics. Theoretical Population Biology, **16**, 172–190

Endler, J. A. (1977). Geographic variation, speciation, and clines. Princeton: Princeton University Press

Ferriere, R., & Gatto, M. (1995). Lyapunov exponents and the mathematics of invasion in oscillatory or chaotic populations. Theoretical Population Biology, **48**, 126–171

Gomulkiewicz, R., Holt, R. D., & Barfield, M. (1999). The effects of density dependence and immigration on local adaptation and niche evolution in a black-hole sink environment. Theoretical Population Biology, to appear

Guckenheimer, J., & Holmes, P. (1983). Nonlinear Oscillations, Dynamical Systems, and Bifurcations of Vector Fields. Applied Mathematical Sciences, vol. 42. New York: Springer-Verlag

Haldane, J. B. S. (1930). A mathematical theory of natural and artificial selection, Proceedings of the Cambridge Philosophical Society, **26**, 220–230

Holt, R. D., & Gomulkiewicz, R. (1997). How does immigration influence local adaptation? A reexamination of a familiar paradigm. American Naturalist, **149**, 563–572

Katok, A., & Hasselblatt, B. (1995). Introduction to the Modern Theory of Dynamical Systems. Cambridge: Cambridge University Press

Maynard Smith, J. & Slatkin, M. (1973) The stability of predator-prey systems, Ecology, **54**, 384-391

Nagylaki, T. (1992). Introduction to theoretical population genetics, New York: Springer-Verlag

Schreiber, S. (1998). Personal Communication

Slatkin, M. (1987). Gene flow and the geographic structure of natural populations. Science, **236**, 787–792

Wright, S. (1931). Evolution in Mendelian populations. Genetics, **16**, 97–159