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The Effects of Density Dependence and Immigration on Local Adaptation and Niche Evolution in a Black-Hole Sink Environment

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We examine the effects of density dependence and immigration on local adaptation in a ''black-hole sink'' habitat, i.e., a habitat in which isolated populations of a species would tend to extinction but where a population is demographically maintained by recurrent one-way migration from a separate source habitat in which the species persists. Using a diploid, one-locus model of a discrete-generation sink population maintained by immigration from a fixed source population, we show that a locally favored allele will spread when rare in the sink if the absolute fitness (or, in some cases, the geometric-mean absolute fitness) of heterozygotes with the favored allele is above one in the sink habitat. With density dependence, the criterion for spread can depend on the rate of immigration, because immigration affects local densities and, hence, absolute fitness. Given the successful establishment of a locally favored allele, it will be maintained by a migration-selection balance and the resulting polymorphic population will be sustained deterministically with either stable or unstable dynamics. The densities of stable polymorphic populations tend to exceed densities that would be maintained in the absence of the favored allele. With strong density regulation, spread of the favored allele may destabilize population dynamics. Our analyses show that polymorphic populations which form subsequent to the establishment of favorable alleles have the capacity to persist deterministically without immigration. Finally, we examined the probabilistic rate at which new favored alleles arise and become established in a sink population. Our results suggest that favored alleles are established most readily at intermediate levels of immigration.

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INTRODUCTION

Most species live in heterogeneous environments. In many circumstances, the strength and direction of selection vary with location and population size. Indeed, understanding the evolution of adaptation to local environments is a classic problem in evolutionary genetics (Haldane, 1930; Endler, 1977; Slatkin, 1987), as is the study of density dependence in natural selection (Roughgarden, 1971). Density dependence can have direct and indirect effects on evolution (Holt, 1987), both of which must be considered in analyzing adaptation to local environments. The direct effect arises because the relative fitnesses of genotypes can vary with population size, so that spatial variation in abundance can lead to corresponding spatial variation in selection. Studies of density-dependent selection in closed populations (Charlesworth, 1971; Asmussen and Feldman, 1977; Ginzburg, 1977) suggest that genotypic carrying capacity is, under some conditions (see, e.g., Prout, 1980), a reasonable proxy for fitness. Indirect effects on selection arise because density dependence influences population size, and thereby allows non-selective factors to influence local evolution. For instance, a given rate of immigration (number of immigrants per generation) represents a lower rate of gene flow (defined as the fraction of individuals composed of immigrants) in an abundant population than in a scarce population.

One key demographic consequence of spatial heterogeneity is that in low-quality sites ("sink" habitats), a species may persist only because of recurrent immigration from high-quality sites ("source" habitats). One definition of a species' fundamental niche is that set of conditions, resource availabilities, and so on, that permits isolated populations to persist deterministically. Hence, a sink habitat by definition has conditions outside a species' niche (Holt and Gaines, 1992).

Recently, we (Holt and Gomulkiewicz, 1997b) analyzed the evolution of adaptation in sink environments. In particular, we imagined that in a given habitat a population is tending toward extinction (viz., has an absolute fitness less than one, for all densities) but is nonetheless maintained demographically by recurrent immigration from a source, where the species persists. This source, for instance, may be located at the periphery of a species' geographical range. If genetic variation is present, then in principle the population may adapt to the sink environment, such that once it is sufficiently adapted, immigration is no longer required for persistence there.

Analyzing a discrete-generation, haploid model with constant immigration, we found that the spread of a rare locally favored allele depends upon an absolute, rather than relative, fitness criterion. Namely, if a novel allele has absolute fitness greater than unity, it will spread when rare, irrespective of both its fitness relative to the immigrant type and the rate of immigration. This criterion applies in sink populations of stable size whether fitnesses are density dependent or not. Classic population genetic treatments of the interplay between gene flow and selection, by contrast, revolve around the relative strengths of selection (governed by relative fitness) and gene flow. We resolved this seeming contradiction by treating the rate of gene flow not as a parameter, but as a variable that indirectly depends upon the local fitness of the immigrant type, when considering a population sustained stably by immigration.

In this paper, we extend our previous analyses to diploids and examine in some detail the influence of density dependence on the evolution of local adaptation in a sink habitat. Given density dependence, immigration has two potentially important effects: first, it changes genetic composition, and second, it influences population size, and thus fitness. We begin by analyzing the initial increase of a locally favored allele in a diploid population and as before find that an absolute fitness criterion governs the spread of a rare, favored allele in a sink population. We then consider sink populations with both stable and unstable population dynamics and characterize the demographic and genetic properties of populations in which a locally favored allele is maintained by selection. This extends previous work on density-dependent selection by considering the demographic and genetic effects of immigration in a sink population with density regulation. Next, we consider whether a sink population initially sustained by immigration, in which a locally favored allele has spread and is being maintained, could then persist without immigration. That is, we determine whether the sink population has in fact evolved to become a potential source. Finally, we examine the effects of genetic drift and mutation on the probabilistic rate of establishment of new favorable alleles in a sink population sustained by immigration.

MODEL AND ANALYSES

We analyze an "island" model of selection and immigration in a population that is initially a sink. For our purposes in this paper, a sink population is one that will go extinct deterministically unless its numbers are replenished by immigration. We consider a discrete-generation, locally panmictic sink population maintained by recurrent immigration from a separate fixed source population. We assume in particular that evolution

in the sink has a negligible back-effect on the source population; this is the "black-hole sink" scenario discussed by Holt and Gaines (1992). Fitness in the island population is determined by a diploid locus with two alleles, A_1 and A_2. We assume that fitnesses depend on population density in the sink. There are excellent empirical examples of sink populations exhibiting strong density dependence. For instance, Keddy (1981, 1982) showed that populations of the annual *Cakile edentula* on the landward side of sand dunes in Nova Scotia were sinks, dependent upon recurrent seed input for their persistence. Yet, death rates were strongly density dependent.

In the model there are three basic life history events from zygote-to-zygote: selection due to differential mortality, immigration, and reproduction. Population size, N, and the frequency p of allele A_1 on the island are censused immediately after reproduction. For genotype A_iA_j, let $V_{ij}(N)$ denote the (density-dependent) probability of survival from birth to the time of immigration. Just prior to the arrival of immigrants, population size is $N^* = \bar{V}(N) \cdot N$, where $\bar{V}(N) = p^2 V_{11}(N) + 2p(1-p) V_{12}(N) + (1-p)^2 V_{22}(N)$. Every generation, I individuals all homozygous for the locally less fit allele A_2 immigrate to the island. Let N^{**} be the population size immediately after immigration, i.e., $N^{**} = N^* + I$. For simplicity, we assume that post-immigration viability and fecundity are genotype independent; the combined effects of size on post-immigration individual fitness can then be summarized by a single density-dependent term, $F(N^{**})$. After reproduction, population size will be

$$N' = N^{**} \cdot F(N^{**}) = [N\bar{V}(N) + I] \cdot F(N\bar{V}(N) + I), \quad (1)$$

where the prime ($'$) indicates the next generation. Now define $W_{ij}(N) = V_{ij}(N)\, F(N^{**})$ and $\bar{W}_1(N) = pW_{11}(N) + (1-p)\, W_{12}(N)$. Assuming adults mate at random, zygote genotype frequencies will return to Hardy–Weinberg proportions with the frequency of allele A_1 given by

$$p' = \left(\frac{N}{N'}\right) \bar{W}_1(N)\, p. \quad (2)$$

(A similar equation arises if post-immigration fitness components are genotype dependent.) Note that our derivation assumes that mutation and drift are negligible. We relax this assumption below.

Our analysis of the recursions (1) and (2) will proceed as follows. We first characterize population dynamics of the island population, assuming it is fixed for A_2, and then determine the conditions under which a rare, favorable allele (A_1) would spread or be lost in such a population. Second, we describe how population size and gene frequency dynamics are influenced by establishment of a locally favored allele. Third, we examine whether the population could persist if it were suddenly isolated. Finally, we will consider some effects of random genetic drift and mutation on the establishment of a rare, favorable allele in a black-hole sink.

Population Dynamics in the Absence of a Favorable Allele

To determine the conditions under which a rare locally favored allele, A_1, will increase or be lost when island fitnesses are density dependent, it is necessary to characterize the population dynamics of black-hole sink populations in which such a favorable allele might arise. To ensure that in the absence of A_1 the island is a black-hole sink maintained only by immigration, we assume $W_{22}(N) < 1$ for all N. Like the density-independent case analyzed in Holt and Gomulkiewicz (1997b), the black-hole sink population may be maintained at a stable demographic equilibrium, $\hat{N}_2$. Using (1) with $p = 0$, it can be seen that $\hat{N}_2$ must be a positive solution of

$$\hat{N}_2 = [\hat{N}_2 V_{22}(\hat{N}_2) + I] \cdot F(\hat{N}_2 V_{22}(\hat{N}_2) + I). \quad (3a)$$

Density regulation can potentially produce unstable population dynamics—including limit cycles and chaos—in the absence of A_1, even in a sink population. For example, Fig. 1 illustrates the potential for both stable and unstable population dynamics assuming that all density regulation occurs prior to the arrival of immigrants. The results shown are based on (1) with $p = 0$, $F(N^{**}) \equiv f$ (a constant), and $V_{22}(N) = v_{22} \cdot g(N)$, where v_{22} is a constant that reflects the maximum survival from birth to reproduction of A_2 homozygotes. In Fig. 1, we assume the density-dependent factor $g(N) = 1/(1 + aN^c)$, where c determines the severity of density dependence and a is a scaling factor. (We utilize this form of density dependence in several of the results below; for more information see Maynard Smith and Slatkin, 1973; Bellows, 1981.) The finite per capita rate of population decline (i.e., fitness) in the absence of A_1 is thus $W_{22}(N) = fv_{22}/(1 + aN^c)$. The asymptotic population dynamics depend on the products fv_{22} and fI, and the parameter c, which controls the strength of density regulation. We define $w_{22} = fv_{22}$ and $i = fI$ and assume $c \geq 0$ which ensures that fitness is nonincreasing with density; $c = 0$ corresponds to density independence. The assumption $W_{22}(N) < 1$ for all N implies $w_{22} < 1$. Given sufficiently weak density regulation ($c < 3 + 2\sqrt{2}$), a positive locally stable equilibrium

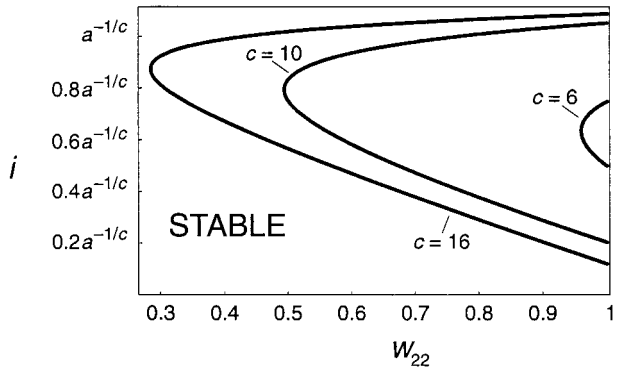


FIG. 1. Parameter values consistent with stable and unstable population dynamics in a black-hole sink population fixed for allele A_2 assuming that density-dependent fitness takes the form $W_{22}(N) = w_{22}/(1 + aN^c)$. The figure shows combinations of w_{22} and $i\,(= fI)$ leading to stable and unstable dynamics for different values of c. Note that i is shown scaled in units of $a^{-1/c}$. The equilibrium sink density $\hat{N}_2$ is stable (unstable) for values of w_{22} and i that lie to the left (right) of a curve that corresponds to a particular value of c. For fixed w_{22}, a, and c, $\hat{N}_2$ is stable for sufficiently large and small values of i.

$\hat{N}_2$ exists and can be computed after substitution into (3a) from

$$a\hat{N}_2^{c+1} - ia\hat{N}_2^c + (1 - w_{22})\,\hat{N}_2 - i = 0; \qquad (3b)$$

numerical results suggest that $\hat{N}_2$ is also globally stable. With stronger density regulation ($c > 3 + 2\sqrt{2}$), the sink population may, depending on w_{22} and i (Fig. 1), approach a limit cycle or exhibit chaotic dynamics, if it does not stabilize. Stable equilibria are always associated with sufficiently small and large values of i (Fig. 1).

Conditions for Increase of a Rare Locally Favored Allele

We now analyze conditions under which a favorable allele A_1 will increase when rare. Assume that the sink population is near fixation for A_2, and is also near an associated stable demographic equilibrium for the recursion (1) with $p = 0$. That is, $N \approx N' \approx \hat{N}_2$, where $\hat{N}_2$ is the equilibrium size in the absence of A_1 (see Eq. (3a)). Then, the recursion (2) for p simplifies to

$$p' \approx \bar{W}_1(\hat{N}_2)\,p \approx pW_{12}(\hat{N}_2). \qquad (4)$$

Thus, allele A_1 will spread when rare if and only if the absolute fitness of the heterozygote at the equilibrium population size for A_2A_2 individuals, $W_{12}(\hat{N}_2)$, exceeds one and otherwise will not—independent of any direct effect of the rate of immigration and independent of $W_{22}(\hat{N}_2)$, which is less than one by assumption. This criterion also applies when fitnesses are density independent, as reported in Holt and Gomulkiewicz (1997b) and when $W_{22}(N) > 1$ for some N (Lofaro and Gomulkiewicz, in press). Immigration may indirectly affect the criterion for spread if $\hat{N}_2$ increases with the immigration rate I since then absolute fitnesses would be depressed at larger immigration rates. In this way, immigration may hamper local adaptation, but via an ecological effect resulting from local density dependence (Holt, 1983) rather than by gene swamping.

Density dependence can further hamper the spread of a locally favored allele in the initial sink population by generating unstable population dynamics, which may occur if density regulation is sufficiently strong. If, say, population sizes are cyclic with period k when A_1 is absent, then an extension of the argument leading to (4) across k generations suggests that A_1 will spread when rare if and only if

$$\left[\prod_{i=1}^{k} W_{12}(\hat{N}_2^{(i)})\right]^{1/k} > 1, \qquad (5)$$

where $\hat{N}_2^{(1)}$, $\hat{N}_2^{(2)}$, ..., $\hat{N}_2^{(k)}$ denote the sink population sizes over one cycle assuming $p = 0$. (This condition can be rigorously justified; Lofaro and Gomulkiewicz, in press.) Asmussen (1979) derived a similar result for a density-dependent selection model without migration. Criterion (5) indicates that the geometric mean of heterozygote fitnesses over one cycle determines the spread or loss of a rare allele. A geometric-mean heterozygote fitness criterion also applies when population dynamics in the absence of A_1 are chaotic (Lofaro and Gomulkiewicz, in press). The geometric-mean fitness criterion (5) for the spread of A_1 implies that unstable population dynamics may limit further the conditions under which a favored allele can spread when rare compared to stable sink populations with the same arithmetic-mean heterozygote fitness, since geometric means are dominated by values near zero.

Polymorphic Equilibria and Unstable Dynamics

Density effects can modulate the influence of immigration on the eventual size and genetic composition of a population following the initial spread of a rare, locally favored allele. A fundamental effect of density dependence is to limit population size which, in turn, bounds the rate of gene flow, I/N', above zero. Combined with our assumption that the immigrant stream consists solely of A_2 alleles, this implies that populations cannot be fixed for the favored allele A_1.

Consider first a population that converges to a stable polymorphic equilibrium after A_1 is successfully introduced. Any equilibrium density $\hat{N}$ and frequency $\hat{p}$ must satisfy

$$\hat{N} = \hat{N}^{**} \cdot F(\hat{N}^{**}), \tag{6}$$

where $\hat{N}^{**} = \hat{N}\hat{\bar{V}}(\hat{N}) + I$ and $\hat{\bar{V}}(\hat{N}) = \hat{p}^2 V_{11}(\hat{N}) + 2\hat{p}(1-\hat{p})\, V_{12}(\hat{N}) + (1-\hat{p})^2\, V_{22}(\hat{N})$. At a polymorphic equilibrium ($\hat{p} > 0$), the equation

$$\hat{\bar{W}}_1(\hat{N}) = [\,\hat{p} V_{11}(\hat{N}) + (1-\hat{p})\, V_{12}(\hat{N})\,]\, F(\hat{N}^{**}) = 1 \tag{7}$$

must also be satisfied (see Eq. (2)). Because the post-immigration density $\hat{N}^{**}$ depends on I, Eqs. (6) and (7) show that both genetic and demographic equilibria will, in general, depend on the immigration rate; i.e., $\hat{N} = \hat{N}(I)$ and $\hat{p} = \hat{p}(I)$.

For the remainder of this section, we will assume that $F(N^{**}) \equiv f$ and $V_{ij}(N) = v_{ij}/(1 + aN^c)$, where v_{ij} is a genotype-specific constant and the parameters a and c are positive. That is, we assume that density regulation is genotype independent and occurs prior to the arrival of immigrants. Note that *relative* fitnesses are density independent (Prout, 1980). These more specific assumptions will allow us to visualize equilibria and analyze their stability more easily. The general conclusions apply to models with alternative forms of density dependence (e.g., discrete logistic and exponential logistic; see Holt, 1983a) or post-immigration density regulation (provided that the population is censused immediately after immigration). As discussed above, the density-dependence factor $1/(1 + aN^c)$ can lead to stable or unstable population dynamics. With $w_{ij} = v_{ij} f$, $i = fI$, and $\hat{\bar{w}} = \hat{p}^2 w_{11} + 2\hat{p}(1-\hat{p})\, w_{12} + (1-\hat{p})^2\, w_{22}$, Eq. (6) becomes

$$\hat{N} = \frac{\hat{N}\hat{\bar{w}}}{1 + a\hat{N}^c} + i. \tag{8}$$

If $\hat{p} > 0$, the second equilibrium condition (7) reduces to

$$\frac{\hat{\bar{w}}_1}{1 + a\hat{N}^c} = 1, \tag{9}$$

where $\hat{\bar{w}}_1 = \hat{p} w_{11} + (1-\hat{p})\, w_{12}$.

Figure 2 illustrates how $\hat{N}$ and $\hat{p}$ depend on i for directional selection, $w_{11} \geqslant w_{12} \geqslant w_{22}$, such that $w_{22} < 1$. (Recall that the latter condition ensures that a population fixed for A_2 is a sink.) In the special case of dominance ($w_{11} = w_{12}$), one can show by combining (8) and (9) that $\hat{N} = [(w_{11} - 1)/a]^{1/c}$ and $\hat{p} = 1 - \sqrt{w_{11} i a^{1/c} / [(w_{11} - w_{22})(w_{11} - 1)^{1/c}]}$ provided that $w_{11} > 1$, which indicates that $\hat{p}$ declines with i and $\hat{N}$ is independent of i and w_{22} (Fig. 2a). If A_1 is incompletely dominant (Figs. 2b–2c), the equilibria are defined only implicitly by (8) and (9).

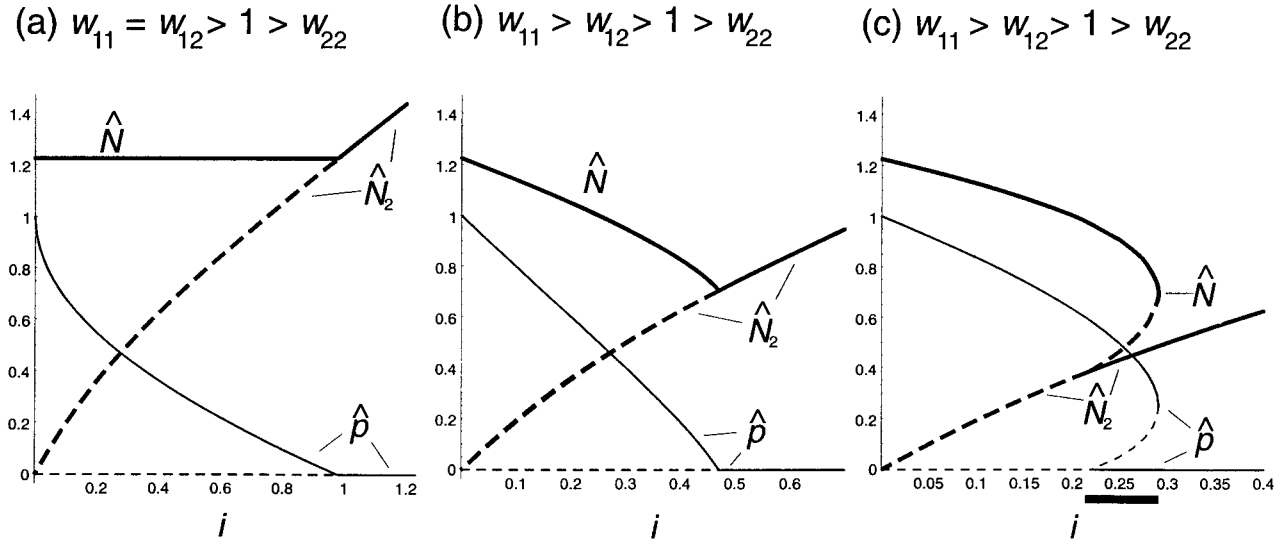
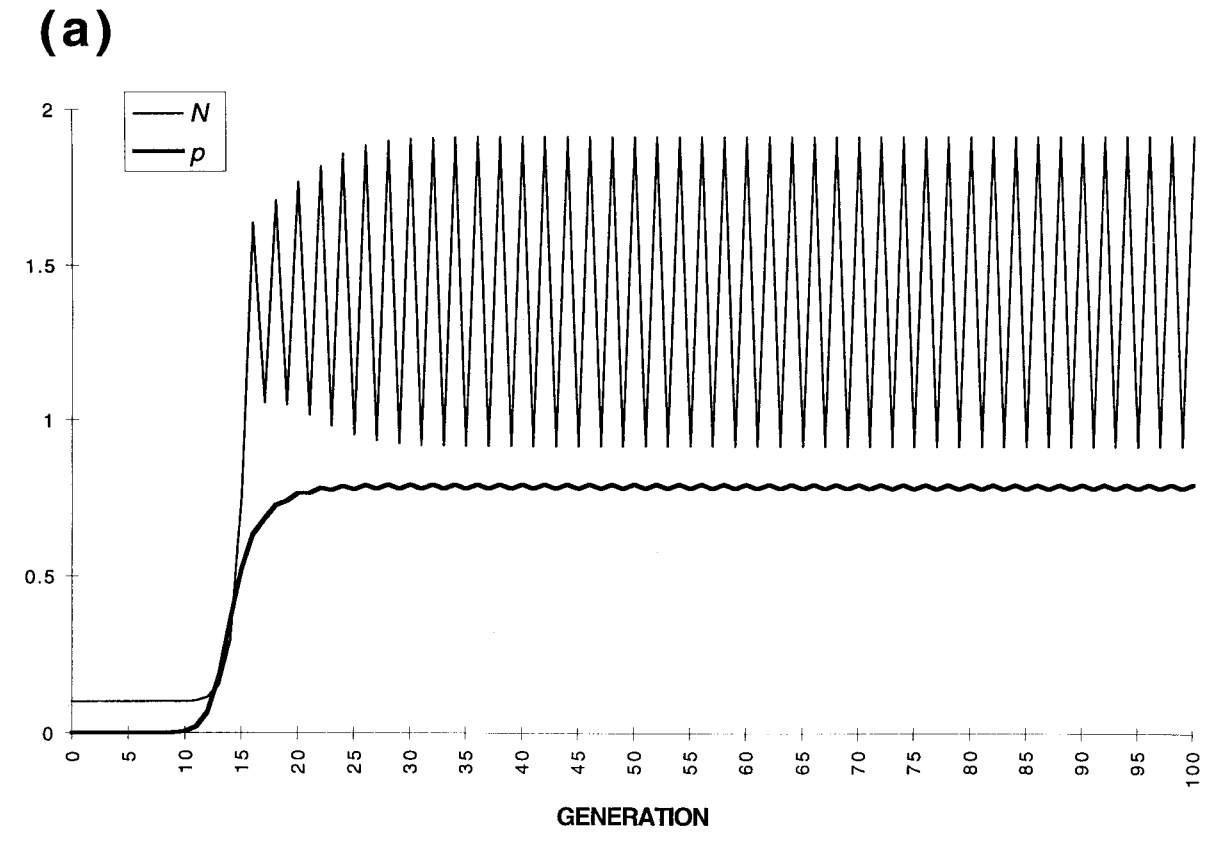


FIG. 2. Stable and unstable equilibrium values of population size and frequency of A_1 vs $i = fI$ assuming absolute density-dependent fitnesses of the form $W_{ij}(N) = w_{ij}/(1 + aN^2)$ with $a = 1$. Note that setting $a = 1$ is equivalent to scaling i and N in units of size $a^{-1/2}$ if $a \neq 1$. Thick curves: equilibrium densities for a polymorphic population ($\hat{N}$) or a population fixed for allele A_2 ($\hat{N}_2$). Thin curves: equilibrium frequencies of A_1 ($\hat{p}$). Solid curves indicate stable equilibria, and dashed curves indicate unstable equilibria. Stability was determined by iterating (1) and (2) near equilibria. (a) A_1 dominant: $w_{11} = w_{12} = 2.5$, $w_{22} = 0.5$. (b) $w_{11} = 2.5$, $w_{12} = 1.5$, $w_{22} = 0.5$. (c) $w_{11} = 2.5$, $w_{12} = 1.15$, $w_{22} = 0.5$. The thick bar below the horizontal axis indicates the range of i over which polymorphic and monomorphic A_2 equilibria are both stable.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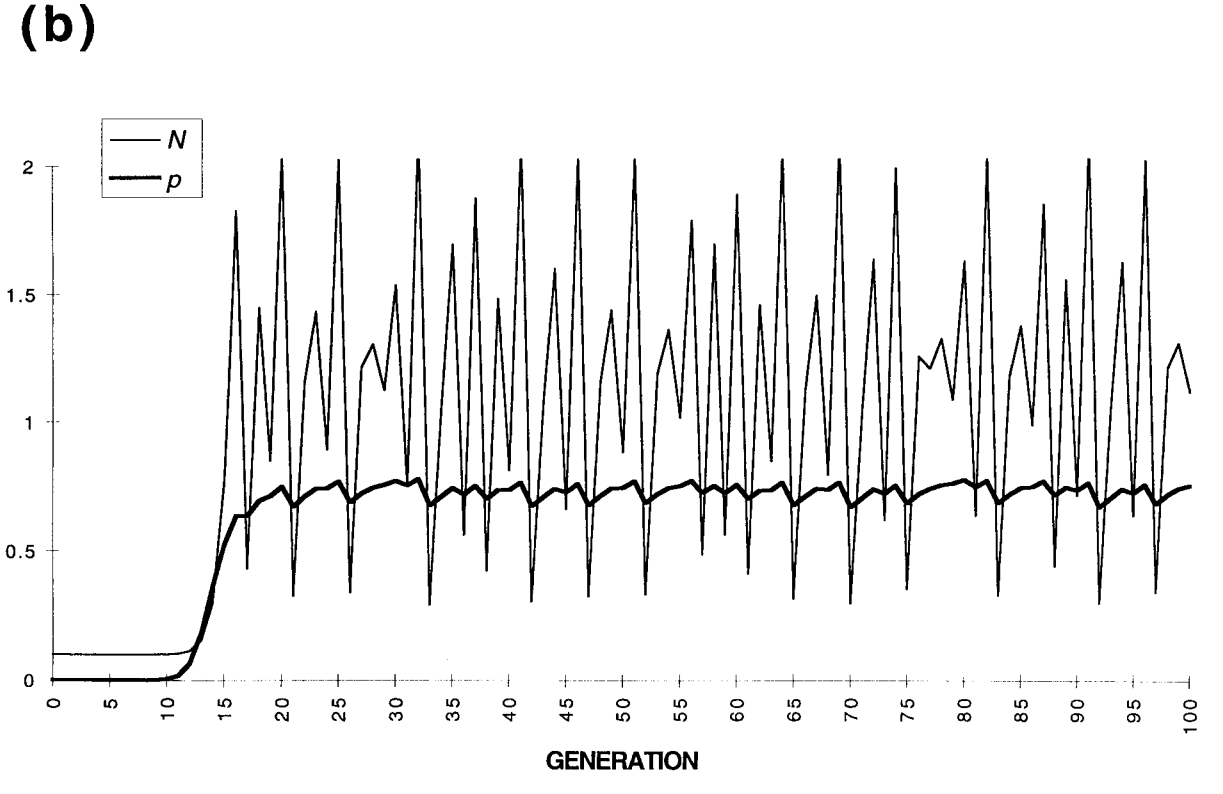
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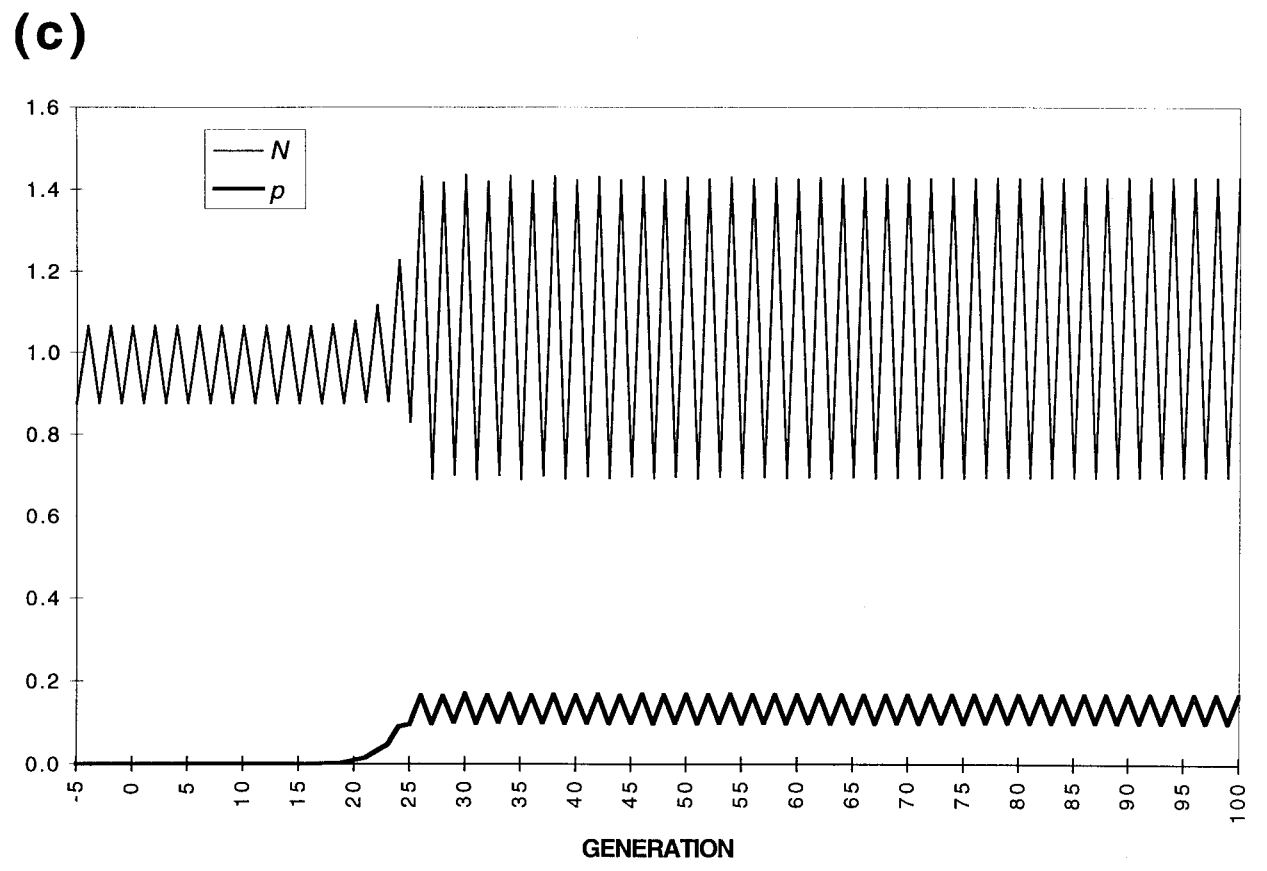

FIG. 3. Dynamics of initial spread and maintenance of a locally favored allele assuming absolute fitnesses of the form $W_{ij}(N) = w_{ij}/(1 + N^c)$. This assumes the scaling parameter $a = 1$, which is equivalent to scaling i and N in units of size $a^{-1/c}$ if $a \neq 1$. (a) Periodic asymptotic dynamics; $c = 3$, $w_{11} = w_{12} = 3.75$, $w_{22} = 0.5$, and $i = 0.05$. (b) Chaotic asymptotic dynamics; $c = 4.5$, $w_{11} = w_{12} = 3.75$, $w_{22} = 0.5$, and $i = 0.05$. (c) Periodic initial and asymptotic dynamics. A_1 is introduced at low frequency in generation 0. $c = 7$, $w_{11} = w_{12} = 3.75$, $w_{22} = 0.9$, and $i = 0.5$.

Polymorphic equilibria of this model need not be stable. We studied the stability of polymorphic equilibria using local stability analysis and by numerically iterating Eqs. (1) and (2). The cases we have examined share a number of features (see Fig. 2):

1. At sufficiently high rates of immigration, the locally favored allele is bound to be lost, making $\hat{p} = 0$ the sole "genetically stable" equilibrium. By this we mean that A_1 cannot spread when rare whether the initial sink population is demographically stable or not.

2. At sufficiently low immigration rates, $\hat{p} = 0$ will be unstable. Polymorphic equilibria will exist, but may be stable (Fig. 2; see below) or, if density regulation is sufficiently strong, unstable in p and N (e.g., Fig. 3).

3. There may be an intermediate range of i for which monomorphic and polymorphic equilibria are both stable (Fig. 2c). In such cases, $\hat{p} = 0$ is approached by populations for which A_1 is rare initially. The alternative stable polymorphic equilibrium will be approached only by populations for which A_1 is sufficiently frequent to begin with.

4. The frequency of A_1 at stable polymorphic equilibria declines with increasing i (Fig. 2). This reflects the increased swamping effect of gene flow at higher immigration rates.

5. For a given immigration rate, the population density at a stable polymorphic equilibrium, $\hat{N}$, exceeds the density $\hat{N}_2$ that would be maintained at the same immigration rate in a population monomorphic for A_2 (Fig. 2). Unless A_1 is dominant, equilibrium densities of stable polymorphic populations tend to decline with increasing i, opposite the effect of i on monomorphic populations.

These five features continue to hold qualitatively if density regulation occurs after immigration, provided one assumes that population density and allele frequencies

are censused immediately after immigration rather than reproduction.

The spread and maintenance of A_1 may lead to unstable dynamics in both N and p. Figure 3 illustrates three possible scenarios. In Fig. 3a, population size and allele frequency dynamics both approach limit cycles after A_1 spreads from its initial rarity. In Fig. 3b, the long-term dynamics are chaotic. For both cases, the initial increase of p is smooth even though A_1 is ultimately maintained with unstable dynamics. Simulations suggest that this smooth initial increase is a general feature of the spread of rare, locally favored alleles of sufficiently high absolute fitness as predicted from our analysis above (see Eqs. (4) and (5)). Figure 3c shows a case in which A_1 is introduced at low frequency into a demographically unstable population that oscillates between two sizes. After A_1 becomes established, population size continues to exhibit a period 2 oscillation, but with increased amplitude. The cases in Fig. 3, taken together, illustrate another general feature suggested by our simulation results, viz., that the spread of A_1 tends to destabilize population dynamics, given strong density dependence in the sink habitat. By this we mean, for example, that a sink population that is demographically stable in the absence of A_1 may become cyclic or chaotic after A_1 spreads.

Niche Evolution

The analyses above address the spread and maintenance of a locally favored allele in a black-hole sink population but not whether the population has evolved to become a potentially self-sustaining population capable of persisting deterministically without recurrent immigration. We therefore analyzed the fate, following sudden isolation, of populations in which A_1 had successfully spread. This was accomplished numerically by setting $I=0$ in recursions (1) and (2) after a population had reached a stable equilibrium or asymptotic non-equilibrium polymorphic state. An example is shown in Fig. 4. In general, A_1 spreads rapidly to fixation after isolation because it is both favored by selection (by assumption) and no longer diluted by an influx of A_2 alleles. Once p is sufficiently near unity, the population dynamics are governed by $N' = NW_{11}(N)$. Such population

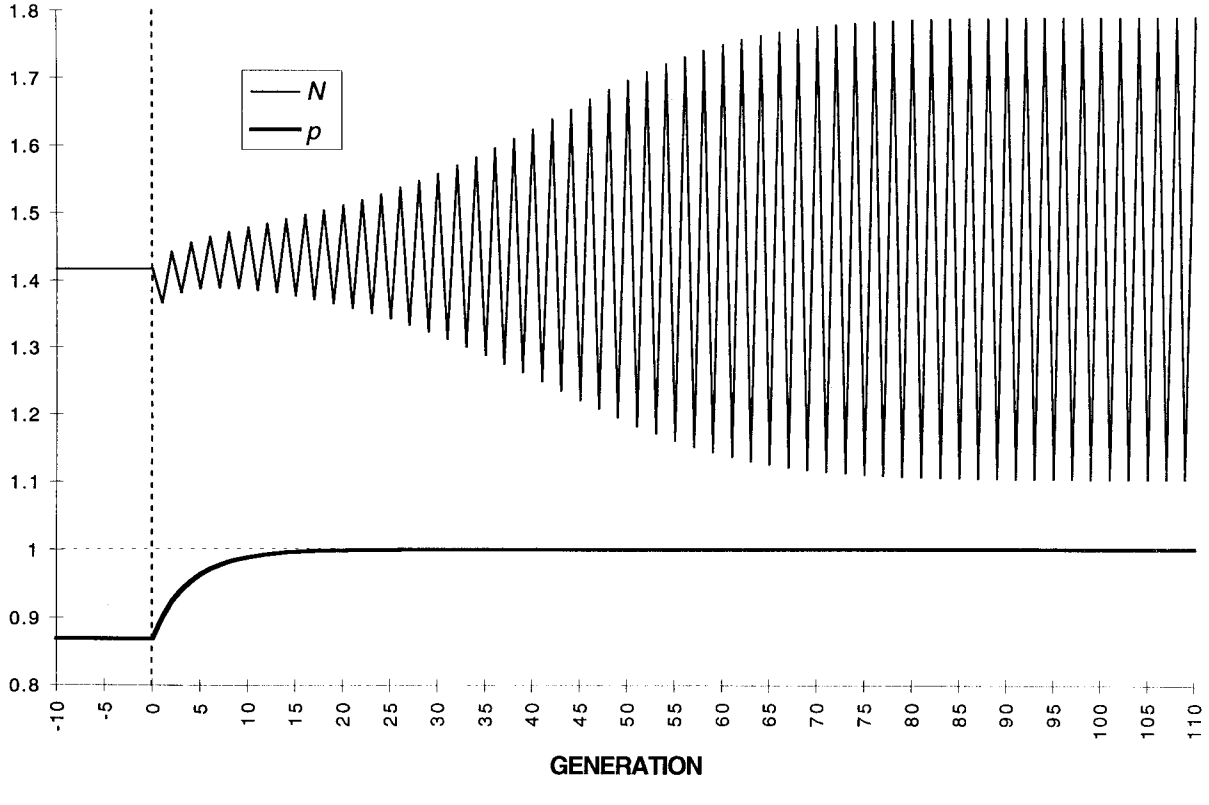

FIG. 4. Population and evolutionary dynamics of an initially stable polymorphic population that is abruptly isolated. Isolation occurs at generation 0 (vertical dashed line). Fitnesses are of the form $W_{ij}(N) = w_{ij}/(1+N^c)$ with $w_{11}=3.75$, $w_{12}=3.0$, $w_{22}=0.5$, and $c=2.8$. The preisolation (scaled) immigration rate is $i=0.05$. Note that the scaling parameter $a=1$ or, equivalently, i and N are scaled in units of $a^{-1/c}$.

dynamics automatically ensure that the population would persist deterministically (perhaps unstably) because, by assumption, $W_{11}(N) \geqslant W_{12}(N)$ for every N and furthermore, $W_{12}(N) > 1$ for N sufficiently small since otherwise A_1 could not spread when rare (see Eqs. (4) and (5)). This suggests that the condition for spread of a rare allele is also the necessary and sufficient condition for niche evolution in a black-hole sink.

Mutation and Drift

The above analyses ignore the effects of mutation and genetic drift. These effects are important in determining the rate of appearance and probabilistic fate of rare novel alleles. For example, it is well known from standard population genetics theory that advantageous alleles may easily be lost when rare due to genetic drift (Haldane, 1927; Fisher, 1930b). By ignoring such stochastic details, our deterministic analyses are overly optimistic regarding the scope for local adaptation and niche evolution in a black-hole sink.

In the last section it was shown that the (deterministic) spread and establishment of a rare allele is required for niche evolution. We will now consider some of the influences of mutation and drift on the establishment rate of novel favored alleles in sink habitats. By "establishment" we mean simply that an allele is present at some specified time in the future. To become established, an allele must appear initially and then persist in the face of genetic drift. We first examine the probability that a newly arisen allele will persist and then consider the rate that novel alleles appear in a black-hole sink population.

Suppose that prior to the appearance of the novel allele A_1 the sink population is in equilibrium at size $\hat{N}_2$ (see Eq. (3)). If the novel allele is unique then, following the approach of Haldane (1927), Fisher (1930a,b), and others, we can approximate genetic drift by a branching process. We will assume that offspring numbers are Poisson distributed and that, initially, the expected number of descendent alleles per A_1 allele is $W_{12}(\hat{N}_2)$. If $W_{12}(\hat{N}_2)$ is close to unity and $|dW_{12}(\hat{N}_2)/dN|$ is small, then the mean number of descendants per A_1 allele should be approximately $W_{12}(\hat{N}_2)$ for several generations.

Using generating functions, it is straightforward to compute the probability that at least one A_1 allele is present t generations after it first appears (e.g., Karlin and Taylor, 1975). However, A_1 is most likely to be lost in the first few generations so the probability that at least one copy of the allele is present in later generations is nearly the same as the asymptotic probability as $t \to \infty$, which we denote by $\varepsilon_{\mathbf{B}}$. Using the above approximations and following standard arguments (e.g., Haldane, 1927), it can be shown that $\varepsilon_{\mathbf{B}}$ is the largest root less than one of the equation

$$1 - \varepsilon_{\mathbf{B}} = e^{-\varepsilon_{\mathbf{B}} W_{12}(\hat{N}_2)} \tag{10a}$$

if $W_{12}(\hat{N}_2) > 1$ and is zero otherwise. Using Taylor series in (10a) leads to the approximation

$$\varepsilon_{\mathbf{B}} \approx 2x - \tfrac{5}{3}x^2 + \tfrac{7}{9}x^3 - \tfrac{131}{540}x^4, \tag{10b}$$

where $x = \ln W_{12}(\hat{N}_2)$ (Fisher, 1930a; see also Gale, 1990, p. 134). If $W_{12}(\hat{N}_2) \approx 1$, then $\ln W_{12}(\hat{N}_2) \approx W_{12}(\hat{N}_2) - 1$ and the first term of (10b) yields the classic approximation $\varepsilon_{\mathbf{B}} \approx 2[\, W_{12}(\hat{N}_2) - 1\,]$ (Haldane, 1927).

Diffusion methods provide a second way to approximate the probability that A_1 is present long after its initial appearance. Letting $\varepsilon_{\mathbf{D}}$ denote this approximation and using the same assumptions regarding $W_{12}(\hat{N}_2)$ as for the branching process, we have (Kimura, 1957)

$$\varepsilon_{\mathbf{D}} = \frac{1 - e^{-2\alpha}}{1 - e^{-4\hat{N}_2\alpha}}, \tag{10c}$$

where $\alpha = W_{12}(\hat{N}_2) - 1$. Note that rather than relative fitness, (10c) uses the total rate of replacement (including immigration) of A_2A_2 individuals, which must equal one in the initial equilibrium sink population.

Technically, $\varepsilon_{\mathbf{B}}$ and $\varepsilon_{\mathbf{D}}$ give the probability of permanent establishment of A_1 for their respective stochastic processes. However, if the number of A_1 alleles on the island grows substantially, then the approximations (10) may no longer apply since actual population sizes will deviate strongly from $\hat{N}_2$ (thereby altering fitnesses because of density dependence) and the number of A_1A_1 homozygotes will become appreciable. Because density regulation will confine the island population to finite sizes and only A_2 alleles arrive in the immigrant stream, A_1 alleles will be lost with probability one, although loss might not occur for a long time if a sizable number of A_1 alleles accumulate. Thus, in the present context $\varepsilon_{\mathbf{B}}$ and $\varepsilon_{\mathbf{D}}$ should be interpreted as approximating the probability that A_1 alleles are present for a substantial number of generations after the allele is introduced in the black-hole sink.

To explore the interpretation and accuracy of the approximations (10), we simulated the evolution of a single A_1 allele in a black-hole sink using a stochastic version of the deterministic model described above. Specifically, we assumed that the initial sink population size was $\hat{N}_2$ (to the closest integer), so that the initial

frequency of A_1 is $1/(2\hat{N}_2)$. Now suppose that t generations later the population size and number of A_1 alleles are N and k, respectively (i.e., the frequency of A_1 is $p = k/(2N)$). In the next generation the population size, N', is generated from a Poisson distribution with expected value $N\bar{W} + i$ (assuming that fecundity is constant at f and $i = fI$) and the number of A_1 alleles (k') is randomly sampled from a binomial distribution with parameters $2N'$ and $p^* = N\bar{W}_1 p/(N\bar{W} + i)$ (see Eqs. (1) and (2)), giving $p' = k'/(2N')$. This is essentially the standard Wright–Fisher model with selection and immigration modified so that population size fluctuates according to a time-inhomogeneous probability distribution.

As expected, the simulations showed that the probability that A_1 is present in at least one copy drops rapidly initially and then declines slowly (Fig. 5). This suggests that A_1 is most likely to be lost in the first few generations. Given that the allele manages to persist through the early generations, the probability it persists for an extremely long time is high. These qualitative features support the rationale for using asymptotic fixation values (10).

Table I compares the approximations (10) with the simulated probability of A_1 alleles persisting at least $10\hat{N}_2$ generations (denoted by $P_{10\hat{N}_2}$) for several parameter sets assuming absolute fitnesses of the form $W_{ij}(N) = w_{ij}/(1 + aN^c)$. The results show that the approximations (10) are fairly close to $P_{10\hat{N}_2}$ for immigration rates $i \geqslant 1$ and perform best at the highest immigration rates. The approximations are more accurate in an absolute sense for weaker selection (top vs bottom cases). They are also better with additive fitnesses compared to dominant fitnesses (left- vs right-hand side). Only the branching process approximation ε_B resembles $P_{10\hat{N}_2}$ when $\hat{N}_2$ is much less than 10. The relationship between the ε's and $P_{10\hat{N}_2}$ is less consistent for dominant fitnesses, although they still tend to indicate the probability of A_1 alleles persisting for a time period on the order of $\hat{N}_2$ generations after introduction when $i \geqslant 1$ (results not shown). Finally, both approximations were sometimes poor for values of c above that assumed in Table I; this is consistent with heightened sensitivity of $W_{12}(N)$ to changes in N when c is large. In summary, the simulations suggest that the approximations (10)—especially ε_B—accurately reflect the probability that A_1 alleles persist for a substantial length of time ($> 10\hat{N}_2$ generations) after introduction in a black-hole sink, particularly for weak selection and weak density dependence, provided that immigration rates are at least moderately large.

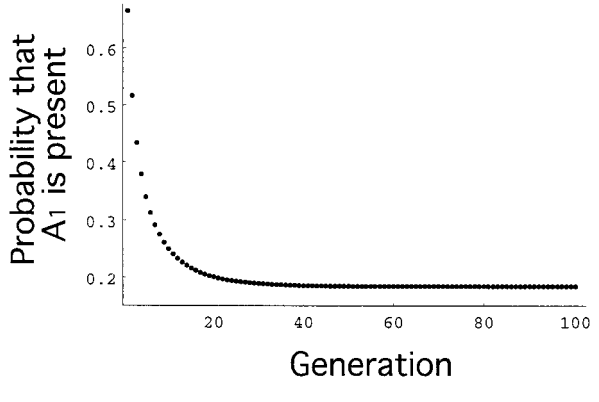

FIG. 5. Probabilities that at least one copy of a new favorable allele is present at various times after its initial introduction. Results are based on 100,000 simulations of the model described in the text assuming absolute fitnesses of the form $W_{ij}(N) = w_{ij}/(1 + 10^{-5}N)$, with $w_{11} = 1.3$, $w_{12} = 1.1$, $w_{22} = 0.9$, and (unscaled) immigration rate $i = 4$. With these parameters, the (unscaled) population size before introduction of A_1 is $\hat{N}_2 = 40$.

(*Aside*: Table I shows that the branching process approximation ε_B is consistently below the diffusion approximation ε_D, although ε_B and ε_D are almost indistinguishable when $\hat{N}_2$ is on the order of 100 or more. The tendency for ε_D to severely overestimate $P_{10\hat{N}_2}$ when $\hat{N}_2 < 10$ may stem from the diffusion approach's built-in assumption that A_1 cannot be lost once it becomes fixed, even if the allele is present in only a few copies. In contrast, A_1 can be lost with positive probability in any generation of the branching process regardless of the allele's relative frequency or copy number. This difference may explain the superior performance of ε_B relative to ε_D when $\hat{N}_2 < 10$, since A_1 will eventually be lost in the simulation model, and also suggests why ε_B tends to be less than ε_D. The approximations converge as $\hat{N}_2$ becomes larger because a high relative frequency of A_1 would then be associated with greater absolute numbers of A_1 alleles. Consequently, the probability that A_1 is lost when at high relative frequency in the branching process would be near zero, much like in the diffusion.)

We now consider the rate of origin of A_1 alleles with heterozygote fitness exceeding one in the initial sink population. Such an allele can arise in one of two ways: through a mutation in the local population or via migration. Of course, we have been ignoring mutation and assuming that the immigrant stream is free of A_1 alleles; we now relax both assumptions. First, let v denote the mutation rate from A_2 to A_1. In an equilibrium sink population, the expected number of A_1 alleles arising through mutation per generation is then $2\hat{N}_2 v$. Second, assume that A_1 is maintained in the continent (source) population at a constant frequency p_c. Then the expected number of A_1 alleles appearing on the island per generation via the immigrant stream is $2ip_c$ at census time. The

TABLE I

Approximate versus Simulated Probabilities of Long-Term Persistence.

i	$\hat{N}_2$	ε_B	ε_D	$P_{10\hat{N}_2}$	i	$\hat{N}_2$	ε_B	ε_D	$P_{10\hat{N}_2}$
		$w_{11}=1.03,\ w_{12}=1.01,\ w_{22}=0.99$					$w_{11}=w_{12}=1.03,\ w_{22}=0.97$		
0.1	10	0.01955	0.06044	0.03080	0.1	3	0.05762	0.17677	0.06330
1	92	0.01794	0.01865	0.02065	1	33	0.05706	0.05877	0.04782
2	171	0.01637	0.01647	0.01746	2	65	0.05644	0.05700	0.05045
4	307	0.01370	0.01373	0.01450	4	128	0.05524	0.05575	0.05060
6	423	0.01140	0.01142	0.01258	6	189	0.05409	0.05458	0.05097
8	527	0.00936	0.00937	0.00966	8	247	0.05298	0.05344	0.05098
10	621	0.00750	0.00751	0.00808	10	304	0.05190	0.05234	0.05128
		$w_{11}=1.3,\ w_{12}=1.1,\ w_{22}=0.9$					$w_{11}=w_{12}=1.1,\ w_{22}=0.9$		
0.1	1	0.17612	0.54987	0.25050	0.1	1	0.17612	0.54987	0.16646
1	10	0.17596	0.18450	0.19651	1	10	0.17596	0.18450	0.14935
2	20	0.17580	0.18097	0.18579	2	20	0.17580	0.18097	0.15858
4	40	0.17545	0.18055	0.18258	4	40	0.17546	0.18055	0.16546
6	60	0.17512	0.18019	0.18178	6	60	0.17512	0.18019	0.16859
8	79	0.17478	0.17984	0.17928	8	79	0.17478	0.17984	0.16775
10	99	0.17445	0.17948	0.17746	10	99	0.17445	0.17948	0.16889

Note. Assumes absolute fitnesses of the form $W_{ij}(N)=w_{ij}/(1+10^{-5}N)$. Equilibrium sizes, $\hat{N}_2$, are computed from Eq. (3b) with $c=1$. Approximations ε_B and ε_D are from Eqs. (10a) and (10c), respectively. ε_B was computed numerically although the approximation (10b) gave nearly identical values. $P_{10\hat{N}_2}$ is the simulated probability that A_1 alleles persist for at least $10\hat{N}_2$ generations for a given parameter set based on 100,000 runs of the simulation model described in the text.

importance of mutation versus immigration as a source of new A_1 alleles depends on the relative sizes of $\hat{N}_2 v$ and ip_c. For example, if the relative fitnesses of A_1A_1, A_1A_2, and A_2A_2 on the continent are, respectively, $1-s$, $1-hs$, and 1, then at mutation-selection balance, $p_c \approx v/(hs)$ assuming h, $s>0$ (e.g., Crow and Kimura, 1970). The ratio of mutation to immigration sources of A_1 alleles in the sink would thus be $hs\hat{N}_2/i$, which is independent of the mutation rate. Immigration will be a more important source of new A_1 alleles in the island population when hs is small than when hs is large.

With expressions for both the rate of origin and the probability of persistence, we can now estimate the overall rate at which A_1 alleles become established in the sink. Considering mutation and immigration together, the total number of new A_1 alleles appearing in the sink per generation is

$$2(\hat{N}_2 v + ip_c). \tag{11}$$

Since $\hat{N}_2$ increases with i, enhanced immigration furthers local adaptation by increasing the rate at which new A_1 alleles appear through both local mutation and immigration (Eq. (11); Fig. 6a). In contrast, the approximations (10) imply that the probability that descendants of a new A_1 allele will be present for a significant period declines with i and is zero for i sufficiently large (Fig. 6b). Thus, increased immigration impedes the ability of A_1 alleles to persist via its effects on density and local fitness. Combining these opposing components gives the approximate rate of establishment

$$2\varepsilon_B(\hat{N}_2 v + ip_c), \tag{12}$$

assuming that the branching process approximation (10a) is used.

In general, the establishment rate (12) is maximal at an intermediate—but potentially large—immigration rate (Fig. 6c). This may have interesting consequences for niche evolution as follows. Suppose we assume that the presence of A_1 alleles would allow the island population to persist deterministically without immigration. Then the establishment rate (12) can be thought of as the per generation probability of niche evolution. Within this context, our results suggest that sufficiently high immigration will hinder or completely prevent niche evolution. However, a reduction in immigration need not enhance the rate of niche evolution, particularly if immigration rates are already low. Moreover, the immigration rate at which niche evolution is maximal decreases with increasing strength of density dependence in the sink (as occurs with increasing values of a in Fig. 6c).

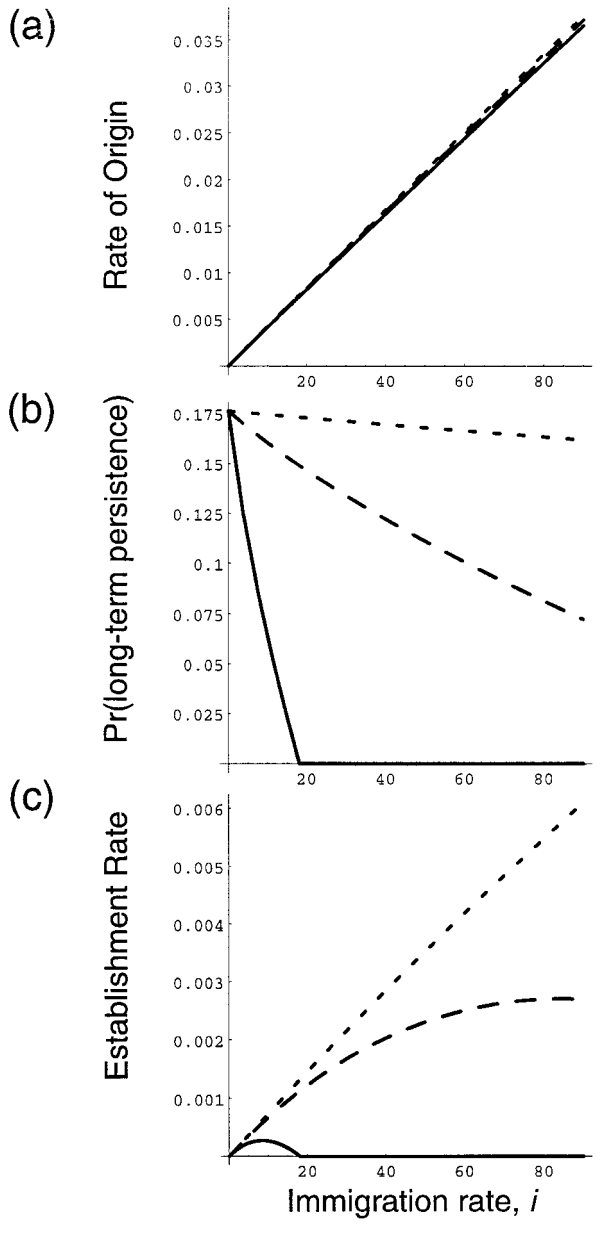

FIG. 6. Components of the rate of establishment of new favorable alleles in an equilibrium black-hole sink vs the rate of immigration. Results are for absolute fitnesses of the form $W_{ij}(N) = w_{ij}/(1 + aN)$, with $w_{11} = 1.3$, $w_{12} = 1.1$, and $w_{22} = 0.9$. Solid lines: $a = 10^{-3}$; dashed lines: $a = 10^{-4}$; dotted lines: $a = 10^{-5}$. (a) Per generation rate of the origin of new favorable alleles through mutation and immigration (Eq. (11)), assuming $v = 10^{-6}$ and $p_c = 2 \times 10^{-4}$ (which if p_c were determined by mutation-selection balance would correspond to, say, $s = 0.01$ and $h = 0.5$). Values of $\hat{N}_2$ were computed from Eq. (3b) with $c = 1$. With these parameter values, it is not hard to show that the ratio of new A_1 alleles expected to appear in the immigrant stream versus from mutation within the sink, $2ip_c/(2\hat{N}_2 v) = 200i/\hat{N}_2$, increases monotonically from $200(1 - w_{22}) = 20$ to 200 with increasing i; immigration is thus the far more likely source of a new A_1 allele in the black-hole sink. (b) Probability of long-term persistence of a new favorable allele (approximation (10b)). (c) Per generation rate of establishment (Eq. (12)).

DISCUSSION

The extent of a species' fundamental niche depends on the absolute fitnesses of the organisms that compose the species. The genetic basis of absolute fitness is likely to vary broadly across species (Orr and Coyne, 1992). Here, we have examined the effects of diploid genetics (including drift and mutation) and density dependence on the spread and maintenance of novel alleles in a black-hole sink population. We also analyzed the conditions for niche evolution in the black-hole sink. Analogous to our deterministic analysis of an asexual population (Holt and Gomulkiewicz, 1997b), we found that local adaptation in diploids depends on an absolute fitness criterion: a locally favored allele will increase in frequency when rare if heterozygote absolute fitness exceeds one. This absolute fitness criterion is completely independent of the rate of immigration and fitnesses of other genotypes provided that there is no density dependence. With density dependence, the criterion for local adaptation may depend on those parameters, but only through their ecological effects on individual fitness; the same criterion holds regardless of which life history stages are density regulated. The reason for an absolute, rather than relative, fitness criterion for local adaptation in a black-hole sink lies in the basic requirement that an allele must be able to persist in order to spread.

Density regulation has a number of potentially important effects on local adaptation in a black-hole sink. First, density dependence causes local fitnesses in the sink to depend on the immigration rate. In the absence of a locally favored allele and given stable dynamics, increasing immigration increases the equilibrium sink size. This demographic effect in turn depresses local absolute fitness, including the fitness of individuals that carry the favored allele. It is through this ecological effect that immigration impedes local adaptation rather than because migration swamps out local adaptation, as is assumed in standard treatments. A second way that sufficiently strong density regulation can hamper the spread of locally favored alleles is by generating nonequilibrium population dynamics. When a novel allele is introduced into a fluctuating sink population, the criterion for its spread depends on a geometric-mean absolute fitness. Geometric-mean fitness strongly reflects low fitnesses and thereby places additional restrictions on the absolute fitnesses necessary for a locally favored allele to invade (Holt and Gomulkiewicz, 1997a). A similar criterion would likely apply if fluctuations in population size were driven by stochastic environmental variation in population growth rates rather than by deterministic density dependence.

Density dependence can also influence the demographic and genetic characteristics of polymorphic populations following the spread of a new allele. Higher rates of immigration will result in lower frequencies of the favored allele as well as lower densities at a stable balance between selection and immigration. However, even the smallest polymorphic densities are above the corresponding densities that would be maintained by the same rates of immigration in a sink population fixed for the deleterious allele (Fig. 2). This suggests that one effect of successful invasion by a locally favored allele in a sink population is to enhance local equilibrium abundances. This result broadens the domain of applicability of the well-known "rule-of-thumb" that density-dependent natural selection tends to increase total population size (Roughgarden, 1971).

Density regulation may lead to permanent fluctuations in the densities of a polymorphic population. Allele frequencies maintained in such a population will also fluctuate in response to changes in density-dependent fitnesses (Asmussen, 1979). In general, the spread of an advantageous allele tends to destabilize sink population dynamics if density regulation is sufficiently strong (Fig. 3). Although populations persist by default in our models, demographic instabilities would likely cause crashes in real populations, with concomitant loss of any locally favored alleles that may have been segregating. Recurrent immigration would renew any population that crashed, but the favored allele will be missing (unless it happened to be present in the immigrant stream). Thus, the interplay of immigration and density dependence could indirectly affect the persistence of a locally favored allele by generating unstable population dynamics with excursions to low abundances. We have assumed in much of the text that absolute fitnesses show negative density dependence, so that $dW_{ij}/dN < 0$. If there is a range of N for which $dW_{ij}/dN > 0$ ("Allee effects") then increasing immigration enhances local fitnesses and may thereby facilitate the evolution of local adaptation. We note, however, that our derivations of criteria for spread (see Eqs. (4) and (5)) nowhere depend upon the sign of dW_{12}/dN.

Immigration, like density dependence, can have a mixture of positive and negative effects on population and evolutionary dynamics in a black-hole sink. The primary benefit of recurrent immigration is to allow a population to persist outside its species' fundamental niche and thereby provide the opportunity for adaptation to unfavorable environments. Moreover, a larger immigration rate implies a larger sink population capable of generating new beneficial mutations. In addition, a higher migration rate will sample more of the genes available in a source population. If alleles that would be favored in the sink occur at low frequencies in the source, then a larger number of immigrants is more likely to sample such alleles, thereby promoting local adaptation. However, if local fitnesses are negatively density dependent, immigration can hamper local adaptation because higher migration rates support larger sink populations, which lead in turn to lower absolute fitnesses. Our analyses confirm that the probabilistic establishment rate of novel alleles in the sink is greatest for intermediate levels of immigration in sink habitats with density-dependent fitnesses. To the extent that the spread and maintenance of new locally favored alleles is required for niche evolution, the results in this paper suggest that when local fitness is density dependent the scope for niche evolution in a black-hole sink will be greatest for moderate—rather than minimal—rates of immigration.

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