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Pulsed Immigration Events Can Facilitate Adaptation to Harsh Sink Environments

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ABSTRACT: In nature, rates of dispersal vary greatly over time, yet most theoretical explorations of ecological and evolutionary dynamics to date have assumed constant movement rates. Here we examine how a particular pattern of temporal variation—periodic pulses of immigration—influences adaptation to a harsh environment, in which a species experiences conditions outside its niche requirements. Using both deterministic models and stochastic individual-based simulations, we show that for many ecological and genetic scenarios, temporally spacing out immigration events increases the probability that local adaptation is sufficient for persistence (i.e., niche evolution). When immigration events are too frequent, gene flow can hamper local adaptation in sexual species, but sufficiently infrequent pulses of immigration allow for repeated opportunities for adaptation with temporary escapes from gene flow during which local selection is unleashed. We develop versions of our models with and without density dependence for three different assumptions about the genetics underlying fitness (haploid, diploid, and quantitative genetic variation) so that our results may be applicable to a wide range of natural systems. Our study adds to a growing body of literature showing that temporal variation in migration rates can have significant effects on local adaptation and is among the first to show how such variation affects niche evolution.

Keywords: niche evolution, variable migration, range expansion, genetic architecture, eco-evolutionary dynamics, propagule pressure.

Introduction

A major constraint on the geographic distribution of a species is its ecological niche—the set of biotic and abiotic conditions permitting persistence without recurrent immigration from external sources (Hutchinson 1957; Holt 2009). However, whether via dispersal, anthropogenic introductions, or environmental change, organisms of all kinds (prokaryotes, plants, animals, fungi, etc.) frequently encounter habitats outside their niches (sink environments; Holt 1985; Pulliam 1988). In some cases, species adapt evolutionarily to these new environments, allowing them to eventually persist in the absence of immigration (which is tantamount to niche evolution). The literature is sprinkled with plausible examples of such niche evolution, sometimes occurring over only tens of generations (Pearman et al. 2008; Carlson et al. 2014). Conversely, there are many cases in which species have remained locally maladapted for long periods of time (i.e., niche conservatism; Holt and Gaines 1992; Webb et al. 2002). Understanding when a species will adapt (and when it will not) to environments outside its niche is a subject that lies at the heart of many questions in ecology, biogeography, evolution, and conservation biology (Holt and Gomulkiewicz 2004; Wiens et al. 2010).

A key determinant of adaptation to a sink environment is immigration, which influences both the likelihood that a species reaches that location in the first place and the local genetics and demography that govern its persistence and evolution thereafter (Holt and Gomulkiewicz 1997; Garant et al. 2007). There are contrasting effects of immigration on local adaptation. Without immigration, a species will not be exposed to a novel environment at all. Moreover, when immigration rates are very low, only a small proportion of the standing variation in source populations will be sampled, and local adaptation may then be limited by a lack of suitable genetic variation (i.e., genetic impoverishment; Bradshaw 1991; Hoffmann et al. 2003; Blows and Hoffmann 2005). Higher immigration rates, in addition to increasing the imported genetic variation, typically boost the population size of recipient populations (Holt 1983), which provides more opportunity for in situ mutation. For both of these reasons, increased immigration rates into a sink habitat could increase local genetic variation and thus facilitate adaptation (Gomulkiewicz et al. 1999; Perron et al. [2007] provide an experimental demonstration of this predicted effect). Additionally, high immigration rates can increase adaptation by counteracting Allee effects (Holt et al. 2004*b*; Courchamp et al. 2008).

Alternatively, adaptation can be inhibited by too much immigration because the recurrent arrival of locally maladapted individuals hampers the establishment of locally

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beneficial alleles. For example, with negative density dependence in the sink, higher immigration rates from a source to the sink increase the sink population size (Holt 1983), which reduces absolute fitness and thus can constrain the emergence of local adaptation (Holt 1996; Gomulkiewicz et al. 1999). Although often there may be negligible negative density dependence in sinks because local maladaptation keeps population sizes low, thereby limiting intraspecific competition, there are some excellent empirical examples of sink populations with strong negative density dependence (e.g., the American sea rocket [*Cakile edentula*] in coastal dunes of Nova Scotia; Keddy 1981, 1982). Beyond this ecological mechanism, when maladaptive immigrants mate with better-adapted residents, locally beneficial combinations of alleles are broken down (i.e., gene flow "swamping selection"; Haldane 1956; Ronce and Kirkpatrick 2001; Kawecki and Holt 2002; Lenormand 2002; Bridle and Vines 2007), thus inhibiting adaptation if immigration is high enough.

Because of these contrasting effects of immigration, in some cases intermediate immigration rates might be optimal for local adaptation (Garant et al. 2007). Indeed, several theoretical studies and laboratory experiments have shown this pattern (Gomulkiewicz et al. 1999; Ronce and Kirkpatrick 2001; Alleaume-Benharira et al. 2006; Lopez et al. 2009; Ching et al. 2013; Barton and Etheridge 2018), and recent work on the invasive Asian tiger mosquito (*Aedes albopictus*) provides evidence that local adaptation at the invasion front peaks with an intermediate immigration rate (Medley 2012).

Most theoretical studies of the interplay of gene flow and natural selection in marginal populations (including those cited in the previous paragraph) have assumed a constant immigration rate, but in nature immigration into a focal habitat is often highly variable over time. Conceptually, this can arise in many ways. For example, in wind-dispersed organisms temporal variation in wind strength and direction can lead to variable immigration rates (Rodríguez-Riaño et al. 2017). Similarly, many marine taxa have episodic immigration events, driven by fluctuating oceanographic conditions (Reed et al. 1988; Hedgecock and Pudovkin 2011). In many animals, variation in temperature can lead to variation in movement and thus dispersal rates (O'Connor et al. 2007; Pärn and Sæther 2012). Even if abiotic conditions are constant, immigration rates can vary due to changes in population abundances, sex ratios, behaviors, or community dynamics (Nager et al. 1996; Matthysen 2005; Schtickzelle et al. 2012; Canham et al. 2014). By ignoring variation in immigration rates, we may have overlooked emergent evolutionary processes at the intersection of gene flow and adaptation. Furthermore, studying these types of more dynamic systems may be increasingly important given changing environmental conditions and increasing environmental variability in the Anthropocene.

A handful of theoretical studies have examined temporally variable migration. In models without selection, increased variation in migration rates increases differentiation among populations (Nagylaki 1979; Whitlock 1992; Gaggiotti 1996; Gaggiotti and Smouse 1996). However, it is less clear how variable migration rates affect local adaptation. Nagylaki (1979) showed (assuming an infinite population size) that variation in migration rates hinders the establishment of rare beneficial alleles. However, more recent studies have shown that with finite population sizes, increased variance in migration rates can reduce the impact of gene flow relative to selection due to an increased covariance between migration and population size; this lowers the effective migration rate and facilitates local adaptation (Rice and Papadopoulos 2009; Rice et al. 2011). In this article, we extend the literature on the evolutionary effects of variable migration and investigate how temporal variation in immigration rates influences adaptation to environments outside a species' niche (i.e., sink environments).

We focus on the specific case in which immigration occurs via pulsed events, such that there are short bouts of immigration between which there are periods without immigration, which might be short or long. Pulsed immigration may be relevant to many empirical systems, perhaps most pertinently in passively dispersed organisms, because natural transport processes often occur as episodic discrete events (Reiners and Driese 2004). We define immigration events as pulsed if there is usually a gap of more than one generation between immigration events. A given amount of time between immigration events might be considered pulsed for one taxon and continuous for others due to differences in generation times. For example, waterfowl often inadvertently disperse aquatic invertebrates (e.g., rotifers, copepods, and ostracods; Frisch et al. 2007). If individual birds arrive annually to a sink environment, immigration might be considered continuous for the birds but pulsed for the invertebrates because of their shorter generation times. Pulsed immigration is a simple pattern of variation in immigration rates, an understanding of which provides a springboard for assessing the implications of more complex patterns of temporal variation in dispersal for adaptation to new environments.

Local adaptation can also be influenced by the genetic basis of fitness (Gomulkiewicz et al. 2010). In some organisms fitness is determined by a few major loci (e.g., metal tolerance in plants; Macnair 1987), while in others it is influenced by many loci, each of small effect (e.g., saline tolerance in yeast; Warringer et al. 2003). The details of different genetic architectures can alter evolutionary outcomes (Lenormand 2002; Gomulkiewicz et al. 2010; Gilbert and Whitlock 2017). For instance, gene flow may have a relatively stronger effect on local adaptation when fitness is determined by many small-effect alleles compared with a few large-effect ones (Yeaman 2015). This is because local

adaptation requires a positive association between locally beneficial alleles (i.e., positive linkage disequilibrium), and these associations are more likely to be broken down by recombination when more alleles are involved (Lenormand 2002).

In this article, we address how the frequency of immigration pulses influences adaptation to sink environments. Under the broad umbrella of this question, we will also examine how density dependence and genetic architecture modulate the impact of pulsed immigration on local adaptation. We conjectured that changing the frequency of immigration could alter the relative impacts of the opposing positive (e.g., increased genetic variance) and negative (e.g., maladaptive gene flow) effects of immigration sketched above. To answer this question, we used both deterministic models and stochastic individual-based simulations. We developed models with and without density dependence for three different assumptions about the genetic architecture underlying fitness: single-locus haploid, single-locus diploid, and multilocus quantitative genetic variation. We predicted that in the diploid and quantitative genetic models spacing out of immigration events could facilitate adaptation to sink environments because gaps between immigration events could serve as temporary escapes from the negative effects of gene flow. We did not predict this pattern in the haploid model because this model assumes clonal reproduction, and thus immigrant genes do not directly interact with resident ones (e.g., many prokaryote systems). Furthermore, we expected that the type of density dependence could alter the effects of the frequency of immigration on adaptation. By considering a wide range of ecological and genetic assumptions, we hope that our models will be relevant to many natural systems, ranging from single-cell prokaryotes to complex multicellular eukaryotes. Our haploid model could be best applied to organisms that reproduce clonally, such as many microbes, plants, and some animals (e.g., *Hydra*), while our other models apply to organisms with sexual reproduction. The diploid model applies in cases in which a trait is mostly determined by a single gene, whereas the quantitative genetic model applies to a trait determined by many genes. We conclude with a discussion of how our results could bear on long-standing issues in evolutionary ecology, such as niche conservatism.

Methods

All our models assume discrete generations and one-way immigration from a single source habitat to a second habitat that is initially a sink for immigrants from the source (namely, a "black hole" sink; Holt and Gaines 1992). In all models, we assume viability selection (i.e., an individual's phenotype—or genotype—affected only its probability of survival to adulthood). Therefore, the organism's fitness was the product of a phenotype-dependent probability of survival until adulthood and a phenotype-independent fixed fecundity. In each model, unless otherwise specified we assume that the immigrants arrive as adults (all at the same time), mate in the sink (for the sexual cases), and then their offspring undergo viability selection. We also assume that immigration is periodic, consisting of regularly spaced generations with immigration between which there were generations with zero immigration. Let T denote the period of the immigration cycle, which consisted of one generation of immigration followed by $T - 1$ without (the frequency was then $1/T$). To tease out the effects of the spacing of immigration events (pulses) from the effects of the total number of immigrants, we primarily focus on the situation where the total number of immigrants over the length of the simulation (1,000 generations) is fixed, while we varied the period T. We denote the number of immigrants per generation with constant immigration ($T = 1$) as I_1. For other periods, the number of immigrants in generations when immigration occurs is $I_T = I_1 T$ (because there was always immigration on the first generation, there were slightly more total immigrants when the number of generations did not include an integer number of cycles). After exploring these models, we constructed models that relax some of these assumptions. We analyzed all models for a range of immigration frequencies from immigration every generation to immigration once every 50 generations. We considered a population "adapted" when it could persist in the absence of further immigration (see "Model Analysis" below for more detail on adaptation criteria).

Deterministic Single-Locus Haploid Model

Our simplest model (adapted from Holt and Gomulkiewicz 1997) assumes that a single-locus haploid trait determines fitness in the sink for a species with clonal reproduction. There are two genotypes, A_1 and A_2, with respective adult abundances N_1 and N_2 (censused after immigration); total population size is $N = N_1 + N_2$. In all deterministic models, population abundances are continuous variables and demographic stochasticity is ignored. The term $W_i(N)$ is the density-dependent absolute fitness of genotype A_i ($i = 1, 2$) in the sink with total population density N. Absolute fitness is the expected value of the number of offspring produced by each individual of a given genotype in its lifetime (which is the product of its probability of survival to adulthood, $V_i(N)$, and fecundity, B). We assume that A_1 has a higher fitness than A_2 for all densities in the sink. We also assume that all individuals in the source (and hence all immigrants) are A_2, which is a realistic approximation if A_1 individuals are maladapted in the source habitat and therefore at very low frequency there. According to our definition of a sink, $W_2(N) < 1$ for all N. For adaptation to be possible, it is necessary that $W_1(N) > 1$ for some N. Because the initial sink population is generated by immigration from the source, we begin with

almost all individuals in the sink having the maladapted genotype A_2 and then determine the conditions that allow the initially rare A_1 genotype to increase. Let $p = N_1/(N_1 + N_2)$ be the frequency of allele A_1 in the sink in the current generation. The mean fitness of the sink population is then $\overline{W}(N) = pW_1(N) + (1 - p)W_2(N)$.

Recursions for the abundance of A_1 and A_2 individuals in the sink from the current to the following generation (the latter denoted by a prime) are, respectively,

$$N_1' = N_1 W_1(N), \tag{1a}$$

$$N_2' = N_2 W_2(N) + m, \tag{1b}$$

where m is the number of immigrants entering the sink that generation. Immigration is periodic, with a period consisting of one generation of immigration followed by a number of generations (in some cases zero) without immigration; in generations in which an immigration event occurs $m = I_T$ and in generations without an immigration event $m = 0$. This allows us to model different frequencies of immigration events.

To incorporate negative density dependence into this model, we chose a convenient form, $W_i(N) = w_i/(1 + cN)$, where w_i is the density-independent absolute fitness of genotype A_i and c determines the strength of density dependence. There is no density dependence if $c = 0$. With this choice of density dependence, if populations persist and the environment is constant (including immigration), population dynamics are stable (i.e., no cycles or chaos). In other models below, we also examined alternative forms of density dependence.

Deterministic Single-Locus Diploid Model

This model (modified from Gomulkiewicz et al. 1999) makes the same ecological assumptions as the haploid model, but fitness is now determined by a single-locus diploid trait for a species with sexual reproduction. As in the haploid model, this locus has two alleles, A_1 and A_2, and the former is assumed to be the favored allele in the sink environment. Let $W_{ij}(N)$ denote the (density-dependent) fitness of genotype A_iA_j in the sink, where N is again the adult population size after immigration and p denotes the frequency of allele A_1 in the sink among these adults (surviving offspring from the previous generation plus immigrants in the current generation). We assume that genotype and density dependence affect offspring survival, and fecundity is constant. The mean fitness of the sink population after random mating and reproduction is $\overline{W}(N) = p^2W_{11}(N) + 2p(1 - p)W_{12}(N) + (1 - p)^2W_{22}(N)$. As with the haploid model, density dependence is implemented by dividing the density-independent absolute fitness of each genotype, w_{ij}, by $1 + cN$, so that $W_{ij}(N) = w_{ij}/(1 + cN)$.

The sink population size N and gene frequency p change across generations as follows:

$$N' = N\overline{W}(N) + m, \tag{2a}$$

$$p' = \left(\frac{N}{N'}\right)\overline{W_1}(N)p, \tag{2b}$$

where $\overline{W_1}(N) = pW_{11}(N) + (1 - p)W_{12}(N)$ is the offspring marginal fitness of A_1. We assume that all individuals in the source are A_2A_2 and begin with nearly all individuals in the sink having genotype A_2A_2. We make the same assumptions about immigration as in the haploid model.

Deterministic Multilocus Quantitative Genetic Model

This model (modified from Holt et al. 2004*a*) assumes that survival in the sink is determined by a polygenic quantitative trait (a trait determined by many genes that contribute additively to the phenotype, such as body size in many organisms; Falconer and Mackay 1996). An individual's phenotype (trait) z is the sum of an additive genetic component (genotype) g and a normally distributed environmental component with mean zero and variance E. The probability that an individual with phenotype z survives to adulthood in the sink is $V(z) = \exp[-(z - \theta)^2/(2\omega^2)]$, where θ is the optimal trait value and ω^2 is inversely proportional to the strength of stabilizing selection in the sink. We assume the source has a fixed mean genotype of zero, so the magnitude of θ measures the harshness of the sink.

As is standard practice for analyzing polygenic quantitative traits, we assume that genotypes in offspring are normally distributed (Falconer and Mackay 1996; Lynch and Walsh 1998) with means of zero and $\bar{g}$ in the source and the sink, respectively, and a constant variance G in both populations. In the sink, we also assume heritability is fixed and that the trait is normally distributed among offspring with a mean equal to the mean genotype of the parents (recall that we assume that immigrants are part of the parent population) and a constant phenotypic variance P, which is the sum of G and E. These assumptions imply that selection and immigration are relatively weak forces compared with recombination (Tufto 2000); we relax these assumptions in the individual-based simulations presented below. Averaging viability over the trait distribution gives the mean viability of the sink population, which is

$$\overline{V} = \left(\sqrt{\frac{\omega^2}{\omega^2 + P}}\right)\exp\left[\frac{-(\bar{g} - \theta)^2}{2(\omega^2 + P)}\right]. \tag{3}$$

Each generation, the recursions for the mean genotype (Bulmer 1985, p. 181) and offspring population size N_j (Holt et al. 2004*a*) in the sink are, respectively,

$$\bar{g}' = (1 - f)\left[\bar{g} + \frac{G}{\omega^2 + P}(\theta - \bar{g})\right], \tag{4a}$$

$$N_j' = B(\overline{V}N_j + m), \tag{4b}$$

where $f = m/(\overline{V}N_j + m)$ is gene flow into the sink and B is the per capita birth rate. The term in brackets in equation (4a) gives the response to selection, with the change in genotype being $Gd\ln\overline{V}/d\bar{g}$, consistent with equation (7) in Lande (1976); the $(1 - f)$ term reflects the reduction of the average genotype because of the immigration from the source, which has a mean genotype of zero. A derivation of the term in brackets in equation (4a) is provided in section A1 of the appendix (available online).

For this model, we consider two forms of density dependence, both of which act by affecting mean viability of the sink population (eq. [3]). In the first form, there is negative density dependence in viability, which we incorporate by dividing the right-hand side of equation (3) by $1 + cN_j$. In the second form, there is positive density dependence at low population sizes (an Allee effect) and no negative density dependence. The Allee effect was implemented by multiplying the right-hand side of equation (3) by $1 - e^{-\varepsilon N_j}$, where ε is a constant governing the strength of the Allee effect (Dennis 1989; Wang et al. 2011). Note that in the quantitative genetic case we track offspring population size (N_j) rather than that of adults.

Individual-Based Simulations

The deterministic models described above make some unrealistic assumptions, most prominently that the sink population (or an allele type within it) cannot become extinct (it can only reach infinitesimally small sizes). Of course, many discrete immigration events to a harsh sink environment surely end in local extinction. Our deterministic models also assume that the source population is fixed, as is heritability, and ignore stochastic processes. We relax these assumptions using individual-based simulations, which extend a protocol originally designed by Bürger and Lynch (1995) and further developed by Holt et al. (2003), among others. The full C++ source codes and accompanying documentation for the individual-based simulations are provided in a zip file, available online.[1]

We constructed versions of our simulations to match the genetic assumptions of all three of our deterministic models (haploid, diploid, and quantitative genetic). In the latter two versions individuals were hermaphroditic and reproduction was sexual, while in the haploid model mating pairs still formed in order to maintain ecological consistency among models but each offspring was a clone of one of the parents. We tracked individuals and their genotypes and phenotypes in two discrete habitats—one a source, the other a sink—with unidirectional movement from source to sink. Each habitat had a finite population and discrete generations, synchronized between the habitats. For most results (exceptions are noted below), events occurred in the following order every generation: immigration from the source to the sink, then reproduction, followed by viability selection on offspring. During viability selection, the probability of an offspring surviving to adulthood was determined by its genotype (haploid and diploid models) or phenotype (quantitative genetics model). If an immigration event occurred, I_T individuals were randomly selected from the source population (without replacement) and moved to the sink during that bout of immigration (with no cost of immigration); otherwise, no individuals moved. Each population was regulated just before reproduction by limiting the number of mating sites to $K = 200$. If there were more than K adults in the population, K individuals were randomly chosen (without replacement) to mate as females at these sites (this "ceiling" form of density dependence differs from the deterministic models described above; we consider alternative forms below). For each mating female, a mate was randomly selected (with replacement) from all surviving adults (so selfing was possible), and $B = 4$ offspring were produced by each mating pair (this mating system makes offspring per adult constant, unlike the fixed mating pairs in Bürger and Lynch [1995], for which one individual does not mate if the population size is odd and below K). In all cases, we allowed mutation in both the source habitat and the sink habitat; this permitted immigration to be a conduit for novel genetic variation arising in the source.

For the haploid case, an individual's genotype was a single locus with either allele A_1 or A_2. This genotype determined the density-independent probability of survival to adulthood in each habitat; A_i individuals survived to adulthood with probability V_{ih} in habitat h. For all presented results, the probabilities of survival to adulthood in the source were $V_{1,\text{source}} = 0.05$ and $V_{2,\text{source}} = 0.5$. The mean fitness of each genotype (in the absence of density dependence) was $w_{ih} = BV_{ih}$ in habitat h. The genotype of each offspring was determined by randomly choosing the genotype of one of its parents. During the birth of each offspring, a mutation occurred with probability $\mu = 0.001$, changing the genotype from one allele to the other.

In the diploid case, a single locus (each allele A_1 or A_2) determined the density-independent probability of survival to adulthood; A_iA_j individuals survived to adulthood with probability V_{ijh} in habitat h. For all presented results, $V_{11,\text{source}} = 0.05$, $V_{12,\text{source}} = 0.125$, and $V_{22,\text{source}} = 0.5$. The mean fitness of each genotype was $w_{ijh} = BV_{ijh}$ in habitat h. The genotype of an offspring was determined by randomly

1. Code that appears in *The American Naturalist* is provided as a convenience to readers. It has not necessarily been tested as part of peer review.

selecting one allele from each parent. During reproduction, each offspring allele mutated to the opposite allele with probability $\mu = 0.001$.

In the quantitative genetic case, the genotypic value of an individual was determined by 10 additive, freely recombining diploid loci. An individual's phenotype z was calculated by adding the allelic values plus a random environmental component, drawn from a zero-mean, unit-variance normal distribution. The density-independent probability of an individual with phenotype z surviving to adulthood was $V(z) = \exp[-(z - \theta_h)^2/(2\omega^2)]$, where ω^2 determines the strength of stabilizing selection and θ_h is the optimal trait value in habitat h (θ_{source} was always set to zero). The genotype of each offspring was determined by first randomly selecting (with equal probability) one of the two alleles of the mother's genotype at each locus for the maternal haplotype and one of the two alleles of the father's genotype for the paternal haplotype. For each haplotype, a mutation occurred at each locus with probability $\mu = 0.001$. When a mutation occurred, a value drawn from a zero-mean normal distribution with variance $\alpha^2 = 0.05$ was added to the previous value of the allele at that locus. (Altering chosen parameter values moderately does not alter the qualitative results we present.)

Relaxing Ecological Assumptions

For all models (deterministic and individual based) we relaxed our assumption that the total number of immigrants over the course of the simulation was fixed ("fixed total") and instead allowed a constant number of immigrants to arrive each immigration event ("fixed pulse"). In this scenario, the total number of immigrants was higher the more frequent immigration events were. We also ran additional individual-based simulations in which we changed the order of events so that immigration was prior to selection (i.e., juvenile immigration). Additionally, we ran individual-based simulations with continuous density dependence in the sink so that each offspring's probability of surviving to adulthood, given by the equations above, was divided by $1 + cN_j$ (N_j is the offspring population size). In this scenario, we maintained ceiling density dependence in the source in order to isolate the effect of density dependence in the sink. Finally, we ran individual-based simulations in which the timing of immigration events was stochastic rather than periodic; immigration events were random and independent, with a fixed probability each generation. Simulations were performed with a range of probabilities (average immigration frequencies).

Model Analysis

We analyzed the three deterministic models by numerically iterating the equations for 1,000 generations with a range of frequencies of immigration events (the results are expressed in terms of generations between immigration events, equal to the period of an immigration cycle T, with $T = 1$ for immigration every generation). For both the haploid model and the diploid model, the initial sink population size was set at the equilibrium value for a constant immigration rate and with all individuals having the source genotype (derivations are provided in the appendix, sec. A2). We began iterations of both these models with the beneficial allele at a low frequency ($p = 0.001$). This simulates the spread of a rare mutant in the sink. For the quantitative genetic model, simulations began with an empty sink; unlike the previous two models, this model assumes that there is genetic variance in the source, so it was not necessary to include initial genetic variance in the sink. After 1,000 generations, dynamics of all deterministic models were either constant or periodic. We considered a population "adapted" to the sink environment if its average fitness was greater than 1 at the end of the simulation (equations for adapted states without density dependence are provided in the appendix, sec. A3).

We analyzed the individual-based simulations by running 100 simulations for each immigration event frequency ($1/T$). We initiated each simulation run with K adults in the source. In the haploid and diploid simulations, all individuals were initiated with genotypes A_2 and A_2A_2, respectively. In the quantitative genetic simulations, initial genotypes were assigned by randomly selecting allelic values from a zero-mean normal distribution with a standard deviation of 0.258, which was chosen to approximate the expected population genetic variance using equation (14) (a stochastic house-of-cards approximation) in Bürger and Lynch (1995; note that the standard deviation of the phenotype's random component was 1, so the heritability is fairly low, ~0.06). After genotype initialization, we simulated source dynamics for 1,000 generations in isolation to allow the source population to reach selection-drift-mutation balance. At this point, immigration to the initially empty sink began (with the first pulse in the first generation), and the simulation continued for a further 1,000 generations. At that time, we discontinued immigration and simulated the sink for another 200 generations in isolation. If the sink population did not become extinct, we considered it "adapted" (the results were similar using other measures of adaptation, which are not shown). The probability of adaptation for each frequency was the number of adapted populations divided by the number of replicates (100).

Results

Single-Locus Haploid Models

For the deterministic density-independent version of the haploid model, the frequency of immigration events did not affect adaptation to a sink environment. This is consistent

with the results of Holt and Gomulkiewicz (1997), who showed that for the density-independent haploid case, immigration rate (with immigrants arriving every generation) was irrelevant to the spread of A_1 when rare; when $p \approx 0$, its spread is described by $p' \approx w_1 p$. Thus, if $w_1 > 1$ the allele will spread, and if $w_1 < 1$ it will not, regardless of fluctuations in the rate of immigration. This makes biological sense, as in the absence of density dependence or sex, immigrants are causally irrelevant to the dynamics of a resident allele. Our individual-based simulations of the haploid case (with ceiling density dependence, at a ceiling greater than the largest number of immigrants in any immigration episode) confirmed this result.

When negative density dependence is incorporated into our deterministic haploid genetic model, the frequency of immigration events can affect local adaptation (fig. 1*A*). With the assumption that the cumulative number of immigrants over the course of the simulation is fixed (so that as immigration becomes less frequent, the size of the immigrant propagule per episode grows), sufficiently spacing out immigration events results in cycles between maladapted and adapted states (fig. 1*B*). This occurs because during immigration events, the flood of immigrants causes the maladaptive immigrant allele to become more abundant and depresses the fitness of both alleles due to increased density dependence, often leading to local maladaptation. However, during the gaps between immigration events, the adapted allele increases in frequency due to its higher relative fitness. Also, because a maladapted population decreases in size, the density-dependent fitness of both alleles rises, and average fitness rises above

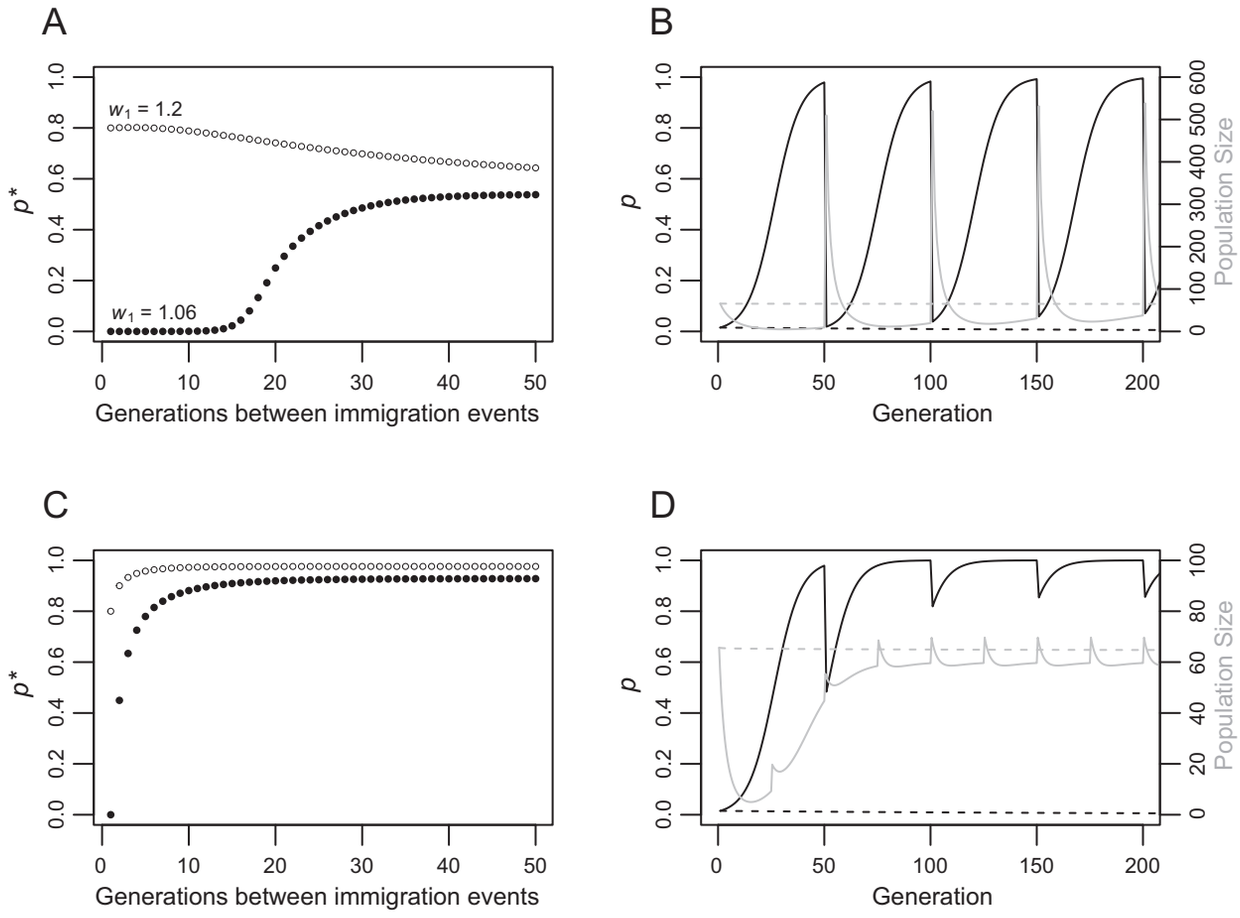

Figure 1: Adaptation to a sink environment when fitness is determined by a haploid trait with density dependence ($c = 0.001$). *A* and *B* show results of the deterministic model with a fixed cumulative number of immigrants (~10,000 immigrants), while *C* and *D* show results with a fixed number of immigrants per immigration event (10 immigrants). *A* and *C* show the effect of spacing out immigration events on the frequency of the beneficial allele in the sink averaged over the final immigration cycle (p^*) for both mild (white circles, $w_1 = 1.2$, $w_2 = 0.9$) and harsh (black circles, $w_1 = 1.06$, $w_2 = 0.9$) sink environments. *B* and *D* are time series showing changes in the frequencies of the beneficial allele (black lines) and population sizes (gray lines) when immigration occurs every generation (dashed lines) or when immigration occurs every 50 generations (solid lines) in a harsh sink ($w_1 = 1.06$, $w_2 = 0.9$).

unity if the time to the next immigration event is long enough. In situations in which adaptation would occur with constant immigration ($W_1(N) > 1$ at the A_2 equilibrium population size; eq. [A4]), the average A_1 allele frequency is high with constant immigration and decreases with greater spacing between immigration events (fig. 1*A*, white circles). Conversely, in situations in which populations remain maladapted with constant immigration ($W_1(N) < 1$ at the equilibrium population size; eq. [A4]), the average A_1 allele frequency is very low with constant immigration and increases with greater spacing (fig. 1*A*, black circles).

Single-Locus Diploid Models

In contrast to the haploid case, when fitness is determined by a single-locus diploid trait, the frequency of immigration events can dramatically affect adaptation to the sink, even without density dependence. From equation (2b), in the density-independent case the frequency of the beneficial allele increases if and only if

$$\left(\frac{N}{N'}\right)[pw_{11} + (1 - p)w_{12}] > 1. \tag{5}$$

When immigration is constant and A_1 is rare, the population size remains constant at the A_2A_2 equilibrium value (given in the appendix, sec. A2); thus, the term $N/N' = 1$, and assuming the initial p is very small, $pw_{11} + (1 - p)w_{12} \approx w_{12}$. Therefore, in this deterministic model with constant immigration, the spread of the beneficial allele is determined by the fitness of the heterozygote; if $w_{12} < 1$, A_1 does not spread when rare, and if $w_{12} > 1$, it does. By contrast, when immigration events are spaced and p is very small, the population size decreases during years without immigration because the mean fitness in the sink is approximately $w_{22} < 1$; therefore, in these years $N/N' > 1$. This means that p can sometimes increase even if $w_{12} < 1$; in years without immigration, the number of A_1 alleles decreases but the number of A_2 alleles decreases more (if $w_{12} > w_{22}$). In addition, as p increases the average fitness of A_1 (which can be written as $w_{12} + p(w_{11} - w_{12})$, an increasing function of p since $w_{11} > w_{12}$) increases, which furthers the spread of the beneficial allele. The average fitness of A_1 can exceed 1 if $w_{11} > 1$, which allows for an increase in the number of A_1 alleles. In other words, in years without immigration the heterozygotes increase in relative abundance, which increases the production of locally adapted homozygotes (A_1A_1) and can lead to an increase in the absolute abundance of A_1.

These conclusions are relevant to the spread of a rare beneficial allele, but they do not show whether adaptation will eventually occur (a rare beneficial allele may initially increase in relative abundance—or even absolute abundance—but then be swamped by gene flow). To do so, we have to evaluate the long-term dynamics of the model. Unfortunately, although the equilibria for this model with constant immigration are relatively simple (see the appendix, sec. A4), when immigration is periodic, the periodic solutions (which separate initial conditions for eventual adaptation from those for maladaptation) become complicated equations from which little analytical insight can be gained. Even for an immigration period of 2, there is no simple closed-form solution for $p > 0$ (see the appendix, sec. A4). Therefore, we focus on numerical iterations of equations (2a) and (2b) for analyses of the long-term dynamics of this model. In agreement with our conclusions above about the spread of an initially rare beneficial allele, numerical iterations showed that if the heterozygote fitness $w_{12} < 1$ and homozygote fitness $w_{11} > 1$, the frequency of the A_1 allele in the sink after 1,000 generations, p^*, is close to zero when immigration is frequent, but if immigration events are sufficiently spaced out, p^* approaches 1 (fig. 2*A*). The immigration period at which p^* switches from the maladapted to the adapted state is determined by the fitnesses of the three genotypes and the initial frequency of the beneficial allele but is independent of I (because initial population size was proportional to I, masking any effects). This threshold period is lowest when w_{12} is high and w_{22} is low (fig. 2*B*), because in this scenario heterozygotes become relatively abundant fastest and can produce sufficient locally adapted homozygotes in a shorter time before maladapted homozygotes arrive with the next immigration event. Note that because there is no density dependence, if adaptation occurs the population becomes very large and gene flow eventually becomes insignificant, so p^* approaches unity.

In the density-dependent version of this model, with constant immigration, the population could not adapt to the sink environment if $W_{12}(N) < 1$ at the equilibrium population size (given in the appendix, sec. A2), but as in the density-independent version, adaptation could sometimes occur if immigration events were spaced out (fig. 2*A*). However, if density dependence was strong enough or the immigration events were sufficiently infrequent and large, the population would cycle between adapted and maladapted states, as seen in the haploid model above. Therefore, with density dependence an intermediate spacing of immigration events led to the highest average A_1 frequency when $W_{12}(N) < 1$. Conversely, if $W_{12}(N) > 1$ at the maladaptive equilibrium population size, adaptation could occur with constant immigration, and spacing out immigration events could hamper adaptation (results not shown).

In our individual-based simulations with diploid genetics and ceiling density dependence, we observed a benefit of spacing out immigration events only for sufficiently harsh sink environments (defined in terms of heterozygote fitness). In mild sink environments, the probability of adaptation was high for all immigration frequencies despite the mean

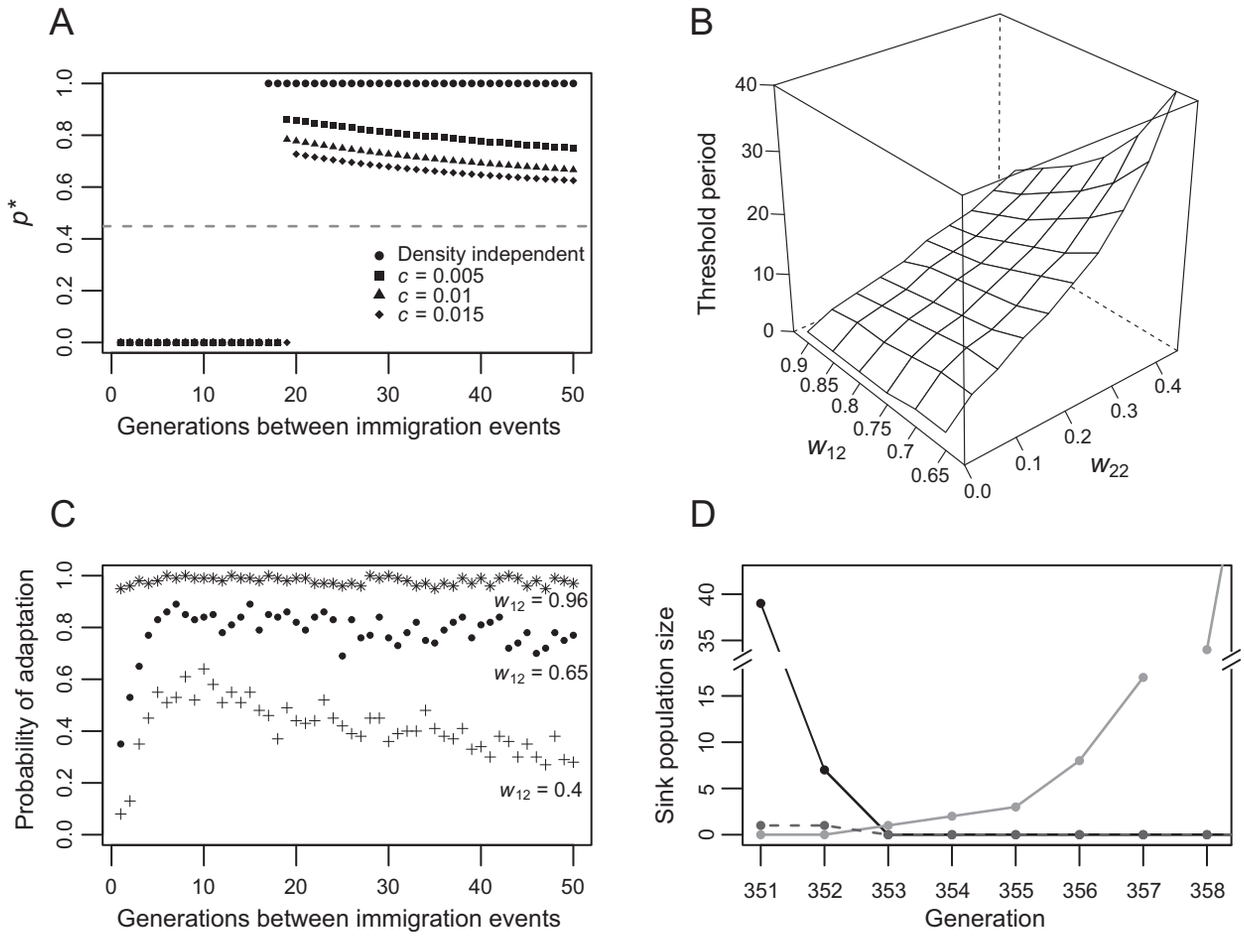

Figure 2: Adaptation to a sink environment when fitness is determined by a diploid trait. *A*, Results of the deterministic model for different magnitudes (including none) of negative density dependence ($w_{11} = 2.0$, $w_{12} = 0.9$, $w_{22} = 0.5$). p^* represents the frequency of the beneficial allele in the sink averaged over the last immigration cycle. The dotted gray line denotes the threshold value for the adapted state when there is no density dependence; values of p above this line will have a positive population growth rate. Threshold values are not shown for the density-dependent cases because the threshold values change with fluctuations in population size. *B*, Immigration period (T) at which p^* switches from the maladapted to the adapted state in the density-independent deterministic model (threshold period; e.g., $T = 18$ in *A*). *C*, Results of the diploid individual-based simulations (with ceiling density dependence) for different degrees of maladaptation. Each point in the plots represents the proportion of 100 runs of the simulation in which adaptation occurred. *D*, Part of one run of the individual-based simulation in a harsh sink environment ($w_{12} = 0.65$) with immigration occurring every 10 generations, providing an example of how adaptation occurs. The sink population sizes (measured in adults after immigration but before viability selection) are shown for homozygotes A_2A_2 (black circles, solid black line), heterozygotes A_1A_2 (dark gray dots, dashed gray line), and homozygotes A_1A_1 (light gray dots, solid gray line). For all models, $I_1 = 4$; for individual-based simulations (*C*, *D*), $\mu = 0.001$, $K = 200$, $B = 4$.

heterozygote fitness being less than 1 (fig. 2*C*, asterisks). This occurred because stochasticity (in both survival and mating) allowed mildly maladapted heterozygotes to occasionally increase in frequency enough in the sink to sufficiently produce locally adapted homozygotes (and p only had to reach less than 0.04 for the mean fitness of A_1 to be greater than 1). Alternatively, in harsh sink environments with frequent immigration, heterozygotes were relatively rare because of their low fitness, making the production of A_1A_1 individuals infrequent (when $w_{12} = 0.65$, p had to reach over 0.26 for the mean fitness of A_1 to be greater than 1). These individual-based simulations with harsh sink conditions closely matched the predictions of our deterministic models such that spacing out immigration events facilitated adaptation (fig. 2*C*, circles and crosses). In accordance with the deterministic model, this benefit occurred because the higher relative fitness of heterozygotes allowed them to become relatively more abundant in generations without immigration, increasing their probability of mating with another heterozygote and increasing the production rate of locally adapted A_1A_1 individuals (fig. 2*D*).

Interestingly, in these simulations there was a decline in the probability of adaptation when immigration events became very infrequent, especially when $w_{12} = 0.4$ (fig. 2*C*), that was not predicted by our deterministic model. Because the relative abundance of A_1 is initially low in the sink and both w_{22} and w_{12} are low, most immigrant pulses rapidly decline, and there is insufficient time for the relative abundance of A_1 to increase enough for $\overline{w}_1$ to exceed 1 deterministically. Therefore, adaptation usually requires that at least one A_1 allele persist until the population size is quite low, at which time demographic stochasticity may sometimes cause $\overline{w}_1 > 1$, via the pairing of heterozygotes and the production of locally adapted homozygotes. A larger spacing between pulses means a larger pulse size, in which case the population will take longer to reach this low level. Therefore, fewer A_1 alleles are expected to survive until this point, as each generation A_1 alleles are lost (because almost all are in heterozygotes and $w_{12} < 1$). Comparing immigration every 50 generations ($T = 50$) with immigration every 10 generations ($T = 10$), in the former case there are five times as many A_1 alleles per immigration pulse, but most are lost in the initial generation (during which the expected population size drops to be very close to the initial population in the latter case). If adaptation of immigration pulses were independent, this loss of A_1 alleles would make the probability of adaptation per event only about twice as high with $T = 50$ as with $T = 10$, while the number of immigration events is only one-fifth as high. This would lead to a lower adaptation probability for $T = 50$ than with $T = 10$, as seen in figure 2*C* (a more detailed version of this explanation is provided in the appendix, sec. A5). However, this explanation does not fully explain the patterns in figure 2*C* because it assumes that adaptation in different pulses are independent, which is likely incorrect.

Multilocus Quantitative Genetic Models

For the quantitative genetic scenario, the frequency of immigration events also significantly affected adaptation to harsh sinks (note that here we define harshness in terms of θ, while in the diploid case it was in terms of heterozygote fitness). In the deterministic model with constant immigration, there is a critical value of sink harshness, θ_c, below which adaptation to the sink occurs and above which it does not (Holt et al. 2004*a*). We show that below this critical value, adaptation always occurs independent of the frequency of immigration events (fig. A1*A*; figs. A1–A4 are available online); however, temporally spacing out immigration events can allow adaptation when $\theta > \theta_c$ (figs. 3*A*, A1*B*–A1*F*). The greater θ is above θ_c, the more spaced out immigration events must be in order for adaptation to occur (fig. A1).

The deterministic quantitative genetic model with negative density dependence is qualitatively similar to the density-independent version. However, with stronger density dependence, immigration events must be more spaced out for adaptation to occur, and the final average genotype of an adapted population gets farther away from the optimum phenotype due to smaller population sizes caused by density dependence and thus increased gene flow (fig. 3*A*). In addition, as in the previous models, if density dependence is strong enough, when immigration events become sufficiently infrequent and large the population can cycle between adapted and maladapted states. Therefore, if $\theta > \theta_c$, with strong density dependence, an intermediate frequency of immigration events is optimal for adaptation to the sink, and if $\theta < \theta_c$, more frequent small immigration events are optimal.

When we incorporated an Allee effect into our model, there was also a benefit of spacing out immigration events. The stronger the Allee effect (lower ε), the less frequent immigration events had to be for adaptation to occur (fig. A2*A*–A2*C*). With an Allee effect, less frequent immigration events were beneficial not only because of the temporary releases from gene flow but also because we assumed that less frequent immigration events were larger (see "Relaxing Ecological Assumptions" for alternative assumptions). Because of the positive density dependence in this model, large enough immigration events allow the density-dependent fitness of the beneficial allele to become greater than unity and lead to adaptation, when it would not occur with smaller immigration events.

Comparable to our results for diploid genetics, in the quantitative genetic individual-based simulations (with ceiling density dependence) we observed an adaptive benefit of spacing out immigration events only when sink environments were sufficiently harsh (high θ). For relatively mild sink environments, the probability of adaptation was high for all immigration frequencies (fig. 3*B*, asterisks), but for harsher sink environments the probability of adaptation was low for constant immigration but then increased as immigration events became less frequent (fig. 3*B*, circles and crosses). In these harsh environments, strong gene flow inhibits adaptation when immigration is frequent, and spacing out immigration events allows for temporary escapes from gene flow during which adaptation can occur (fig. 3*C*). Note that although parameter values were chosen to be comparable, the values of θ that constitute a harsh sink differ between our deterministic and individual-based models, such that some values of $\theta > \theta_c$ are still relatively mild sinks in the individual-based simulations. This is due to the additional sources of stochasticity included in our individual-based simulations, such as the explicit modeling of the source population. In the deterministic models, we assume that genetic variation in the source is fixed with a mean of zero and a variance of G, but in the individual-based simulations the mean and variance of the source population can vary over time. In addition, the immigrant pool is a random subset of this population.

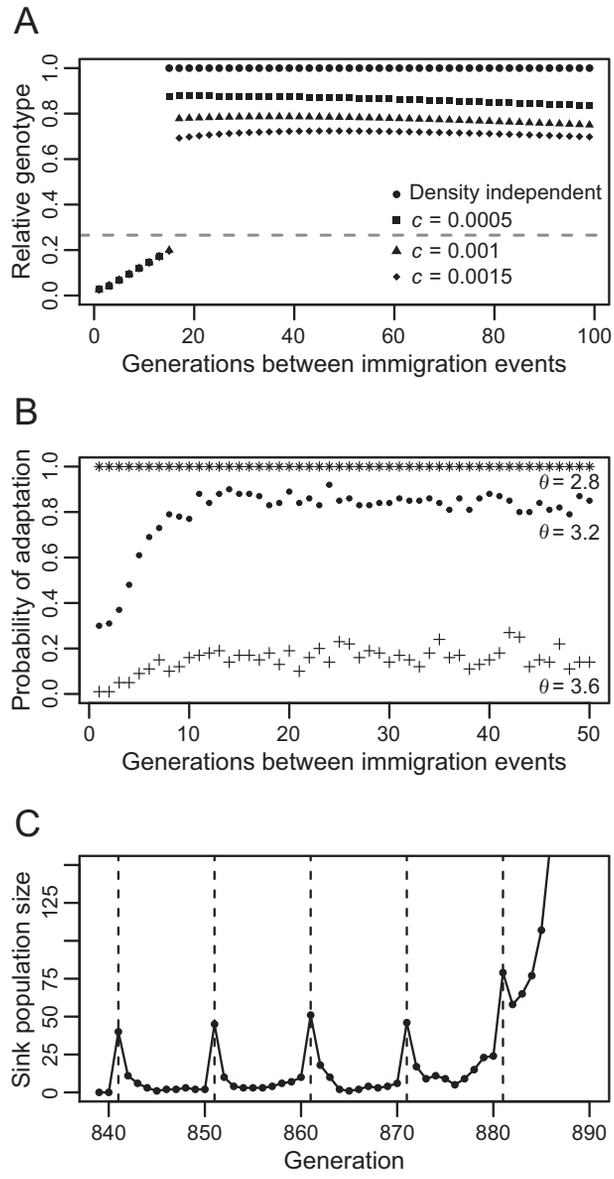

Figure 3: Adaptation to a sink environment when fitness is determined by a multilocus quantitative trait. *A*, Results of the deterministic model with a harsh sink ($\theta = 2.8$) for different magnitudes (including none) of negative density dependence. Relative genotype is the mean genotype of the sink population averaged over the final immigration cycle divided by the optimum phenotype in the sink (a perfectly adapted population has a relative genotype of 1). The dashed gray line denotes the threshold value for the adapted state when there is no density dependence, above which the population will have a positive growth rate. Threshold values are not shown for cases with density dependence because the threshold values change with fluctuations in population size. *B*, Results of the quantitative genetic individual-based simulations. Each point represents the proportion of 100 runs of the simulation in which adaptation occurred. *C*, Part of one run of the individual-based simulation in the harsh sink environment ($\theta = 3.2$) with immigration occurring every 10 generations, providing an example of how adaptation occurs. The dashed lines denote immigration events. For all models, $I_1 = 4$, $B = 4$, $\omega = 1$; for deterministic models (*A*), $G = 0.067$, $P = 1.067$; for individual-based simulations (*B*, *C*), $\mu = 0.001$, $K = 200$.

Because of these factors, sometimes the immigrants arriving into the sink are less maladapted than is assumed in the deterministic models. These and additional sources of stochasticity can facilitate adaptation to sink environments.

Relaxing Ecological Assumptions

In the absence of density dependence, the deterministic models for all three genetic architectures behaved similarly, regardless of whether we held the immigration pulse size or the total number of immigrants constant. However, in the individual-based simulations, when there was a fixed number of immigrants per pulse, the probability of adaptation always decreased as immigration events became very infrequent (fig. 4, gray circles). This decline occurs because with fewer immigration events there are fewer cumulative immigrants into the sink over the course of the simulation, and hence less beneficial genetic variation is made available for selection in the sink. For example, in the haploid simulations the average frequency of A_1 in the source was 0.00275. Therefore, with $I_1 = 4$, when immigration occurs every generation, an expected number of 11.0 individuals with the A_1 allele immigrate into the sink over the course of an entire simulation, but only 0.22 do when immigration occurs every 50 generations. In general, if each immigrant individual has the same probability q of generating a persistent lineage in the sink habitat and all lineages are independent, then given n cumulative immigrants, the probability that at least one of them generates a lineage that persists is $Q = 1 - (1 - q)^n$, an expression that does not depend on the timing of immigration. A plot of $\log_{10}(1 + Q)$ against n has a linear relationship, matching what was seen in our simulation results for haploid organisms and no density dependence, for which the assumptions of this equation apply (fig. 5). We did not see these declines in the probability of adaptation with less frequent immigration in our deterministic models because in those models we did not explicitly model the source populations and local populations could not become extinct.

Despite the decreased total number of immigrants (and thus beneficial mutations) entering the sink, there was still a benefit of spacing out immigration events in our diploid and quantitative genetic individual-based simulations when there was a fixed number of immigrants per pulse (with both ceiling and continuous density dependence). In these simulations, assuming harsh sink environments, the probability of adaptation peaked at an intermediate frequency of immigration events and then declined as immigration events became very infrequent (fig. 4*C*–4*F*, gray circles).

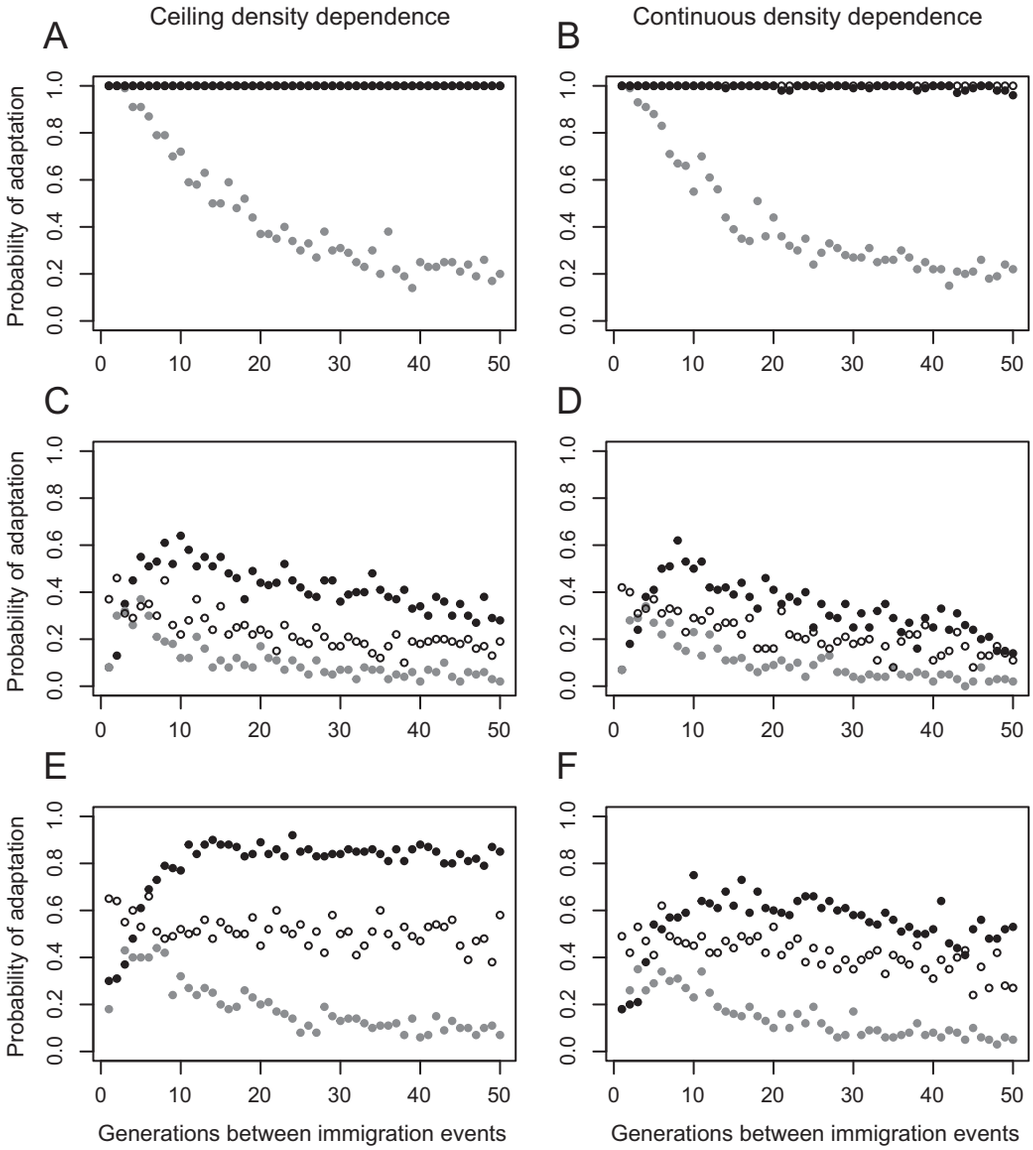

Figure 4: Relaxing ecological assumptions of individual-based simulations for all three genetic assumptions: haploid (*A*, *B*), diploid (*C*, *D*), and quantitative genetic (*E*, *F*). The first column (*A*, *C*, *E*) shows results for simulations with ceiling density dependence, and the second column (*B*, *D*, *F*) shows results for simulations in which density dependence acts continuously across population sizes. Each point represents the proportion of 100 runs of the simulation in which adaptation occurred. Black circles show the simulation results with adult migration and a fixed cumulative number of immigrants over 1,000 generations (about 4,000 immigrants). Gray circles show results for simulations in which immigration is restructured so there were a constant number of immigrants each immigration event ($I_1 = 4$). White circles show results for simulations with juvenile migration and a fixed cumulative number of immigrants (about 4,000 immigrants). Additional parameters: for *A*, $w_{1,\text{sink}} = 1.6$, $w_{2,\text{sink}} = 0.2$, $B = 4$, $\mu = 0.001$, $K = 200$; for *B*, $w_{1,\text{sink}} = 1.6$, $w_{2,\text{sink}} = 0.2$, $B = 4$, $\mu = 0.001$, $c = 0.0015$; for *C*, $w_{11,\text{sink}} = 2.0$, $w_{12,\text{sink}} = 0.4$, $w_{22,\text{sink}} = 0.2$, $B = 4$, $\mu = 0.001$, $K = 200$; for *D*, $w_{11,\text{sink}} = 2.0$, $w_{12,\text{sink}} = 0.4$, $w_{22,\text{sink}} = 0.2$, $B = 4$, $\mu = 0.001$, $c = 0.0015$; for *E*, $\theta = 3.2$, $\omega = 1$, $B = 4$, $\mu = 0.001$, $K = 200$; for *F*, $\theta = 3.2$, $\omega = 1$, $B = 4$, $\mu = 0.001$, $c = 0.0015$.

There are a few interesting results that emerge from our density-dependent deterministic models when we restructure immigration such that there is a constant number of immigrants arriving each pulse. With this assumption, in our haploid model with negative density dependence, spacing out immigration events can lead to adaptation when it would not occur with constant immigration (fig. 1*C*, black circles). This is because if immigration occurs every generation, the population size remains constant at the equilibrium value, $W_1(N)$ thus remains less than 1, and so this allele cannot

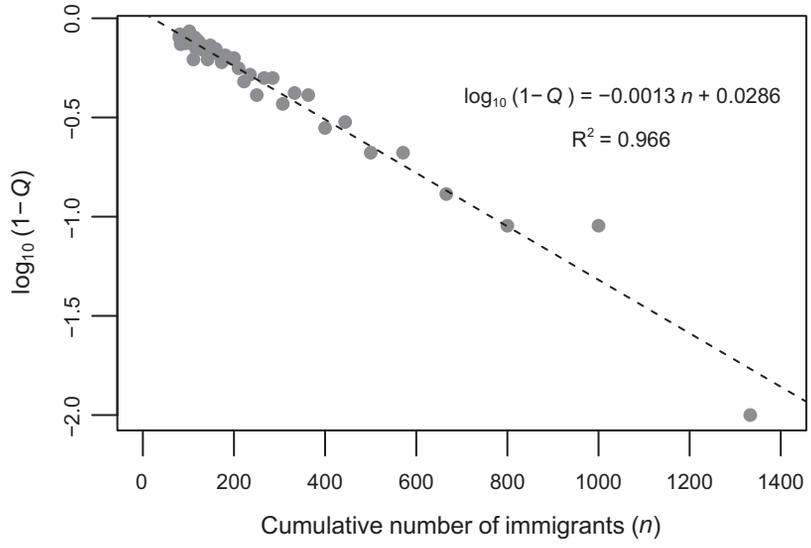

Figure 5: Relationship between the cumulative number of immigrants that enter the sink during each simulation and the probability of adaptation (Q) in the haploid individual-based simulations. Each point represents the proportion of 100 runs of the simulation in which adaptation occurred. The dashed line is a linear regression line. Parameters: $w_{1,\text{sink}} = 1.6$, $w_{2,\text{sink}} = 0.2$, $B = 4$, $\mu = 0.001$, $K = 200$.

increase when rare (fig. 1*D*, dashed lines). However, when immigration events are spaced out, the frequency of A_1 then increases. Also, population size decreases in years without immigration, as the majority of individuals are A_2 and by assumption $W_2(N) < 1$. If there are enough consecutive years without immigration, the population size will drop low enough that $W_1(N) > 1$ and the absolute number of A_1 alleles increases, which is necessary for adaptation with recurrent A_2 immigration (fig. 1*D*, solid lines). Unlike with a fixed cumulative number of immigrants, in this scenario the population remains well adapted after the next immigration event because immigration events are not larger at low frequency (compare fig. 1*B* with 1*D*).

When we made immigration pulse size constant in the deterministic diploid and quantitative genetic models (without density dependence), there was no longer any negative effect of spacing out immigration events because rarer immigration events were not correspondingly larger. Therefore, in these models spacing out immigration events had a beneficial effect only on adaptation to the sink (results not shown).

When there was an Allee effect in the quantitative genetic model, restructuring immigration dramatically changed how the frequency of immigration events affected local adaptation. In this scenario, having less frequent immigration events can prevent adaptation to a sink to which adaptation can occur with frequent immigration (fig. A2*D*–A2*F*). With an increased strength of the Allee effect (lower ε), immigration events must be more frequent in order for adaptation to occur (fig. A2*D*–A2*F*). This pattern emerges because more frequent immigration sustains larger sink populations in which individuals will have higher fitness. With the Allee effect and infrequent immigration, the population size reaches low-enough levels near the end of a period that the fitness of even adapted individuals is pushed below unity.

In general, incorporating continuous negative density dependence into the individual-based simulations decreased the probability of adaptation (fig. 4). This was most prominent in the simulations with a fixed cumulative number of immigrants over the entire simulation (black circles). In these simulations, the probability of adaptation decreased as immigration events became very infrequent because the large population sizes following immigration events significantly decreased mean population fitness. Given a fixed cumulative number of immigrants and continuous negative density dependence, there tended to be a higher probability of adaptation with many small immigration events than with a few large ones in mild sink environments (fig. A3). But in harsh sink environments (in the diploid and quantitative genetic models), immigration events that were intermediate in frequency and size were optimal for adaptation (fig. 4*D*, 4*F*).

If viability selection occurred before immigrants had a chance to mate (juvenile immigration), we no longer observed a benefit of spacing out immigration events (fig. 4*C*–4*F*, white circles) for the diploid or quantitative genetic individual-based model with either ceiling or continuous negative density dependence. The reason is that with juvenile immigration, gene flow is less likely to inhibit adaptation because most maladapted immigrants are culled by selection before having the opportunity to mate with residents, so breaks in the flow

of immigrants are less consequential to local evolutionary dynamics.

Finally, when we treated immigration as a stochastic rather than a periodic process, such that in each generation there was a fixed probability of an immigration event occurring (and the total number of immigrants was fixed), we saw a pattern similar to that observed for periodic immigration. That is, in the haploid model the per-generation probability of an immigration event did not affect the probability of adaptation, and in the diploid and quantitative genetic models the probability of adaptation increased as the probability of an immigration events decreased (fig. A4). As with periodic immigration, in the diploid model there was a decrease in the probability of adaptation when the probability of immigration became very infrequent (fig. A4, gray circles).

Discussion

One way to think about adaptation in systems with pulsed immigration is as repeated opportunities for evolutionary rescue interrupted by pulses of gene flow (e.g., fig. 3*C*). In periods between pulses of immigration, local population dynamics resemble a closed population experiencing a sudden unfavorable environmental change, in which case, given a harsh sink with genetic variation, the local population will decline toward extinction and begin to adapt (the evolutionary rescue scenario of Gomulkiewicz and Holt 1995). When immigration events are infrequent, effects of gene flow are negligible because either extinction or adaptation is likely to occur before the next immigration event. However, as immigration events become more frequent, it is increasingly likely that an adapting sink population will have only partially adapted before the next immigration event. Then, when the next immigration event occurs, the influx of maladapted individuals will cause the population to lose some (or all) of the local adaptation that was gained during isolation. Therefore, with too high an immigration frequency, gene flow can inhibit local adaptation.

We have shown that for a variety of ecological and genetic scenarios spacing out immigration events can facilitate adaptation to harsh sink environments because it allows temporary escapes from gene flow during which local selection can be unleashed (results are summarized in table 1). Even when we did not fix the total number of immigrants (such that the total number of immigrants decreased as immigration became less frequent), breaking up continuous immigration was still beneficial in harsh environments, although when immigration became very infrequent the probability of immigration began to decrease. These results suggest that niche evolution may at times be facilitated by temporal variation in immigration, whereas niche conservatism might be expected with invariant immigration.

However, we did find that in some circumstances temporal variation in immigration may hamper adaptation to sink environments and hence be an agent of niche conservatism. This occurred most often when we incorporated strong density dependence into our models. Generally, with strong negative density dependence, rare large immigration events

Table 1: Overview of our conclusions for different model assumptions

Genetics	Immigration structure	Density dependence	Effect of spacing out immigration events
Haploid	Fixed total	None	No effect on adaptation
Diploid	Fixed total	None	Facilitates adaptation (fig. 2)
Quantitative genetic	Fixed total	None	Facilitates adaptation (fig. 3)
Haploid	Fixed pulse	None	Hampers adaptation (fig. 4*A*)
Diploid	Fixed pulse	None	Intermediate spacing optimal for adaptation to harsh sinks (fig. 4*C*)
Quantitative genetic	Fixed pulse	None	Intermediate spacing optimal for adaptation to harsh sinks (fig. 4*E*)
Haploid	Fixed total	Negative	Results in cycles between adapted and maladapted states (fig. 1*A*, 1*B*)
Diploid	Fixed total	Negative	Intermediate spacing optimal for adaptation to harsh sinks (fig. 2*A*); hampers adaptation to mild sinks
Quantitative genetic	Fixed total	Negative	Intermediate spacing optimal for adaptation to harsh sinks (fig. 3*A*); hampers adaptation to mild sinks
Haploid	Fixed pulse	Negative	Facilitates adaptation to harsh sinks (fig. 1*C*, 1*D*)
Diploid	Fixed pulse	Negative	Intermediate spacing optimal for adaptation to harsh sinks (fig. 4*D*)
Quantitative genetic	Fixed pulse	Negative	Intermediate spacing optimal for adaptation to harsh sinks (fig. 4*F*)
Quantitative genetic	Fixed total	Allee effect	Facilitates adaptation (fig. A2*A*–A2*C*)
Quantitative genetic	Fixed pulse	Allee effect	Hampers adaptation (fig. A2*D*–A2*F*)

Note: Conclusions are based on interpreting the results of both the deterministic model and the individual-based model. The first column denotes different genetic assumptions, the second column denotes the immigration structure ("fixed total" indicates models with a fixed cumulative number of immigrants, and "fixed pulse" indicates models with a fixed number of immigrants per immigration pulse), the third column denotes the type of density dependence, the final column summarizes the effect of spacing out immigration events on adaptation to the sink environment.

could inhibit adaptation because the flood of immigrants sharply decreases mean population fitness. Furthermore, when there was an Allee effect (which was analyzed only for the quantitative genetics model), spacing out immigration events facilitated adaptation when the cumulative number of immigrants over the simulation was fixed but hindered adaptation when the number of immigrants per event was fixed. These results highlight the importance of understanding local density dependence when analyzing adaptation in sink environments. There are empirical examples of both negative and positive density dependence in populations colonizing new habitats (for the former, see Keddy 1981; for the latter, see Davis et al. 2004). Because these different assumptions lead to different evolutionary outcomes, it is important to understand the underlying ecology of a population when attempting to apply our results to empirical systems.

Our results also emphasize the importance of considering different genetic architectures. Advances in genomic sequencing capabilities now permit researchers to better understand the genetics underlying adaptation (Stapley et al. 2010). Theoretical studies must keep up with these advances in order to generate predictions about how genetic architecture influences the adaptive process. In this study, we have considered three different genetic architectures and showed how the frequency of immigration events had different effects on adaptation in each case. However, we did not consider many aspects of genetic architecture, such as linkage or epistasis, not to mention complex gene networks relating the genotype to the phenotype (e.g., Kimbrell and Holt 2007). Additionally, genetic architecture itself can be shaped by natural selection, which could further alter how temporal variation in immigration modulates evolution in sinks (Orr 1998; Griswold 2006; Holt and Barfield 2011; Yeaman and Whitlock 2011).

We have framed our results in the context of a species immigrating to a new environment, but our findings are also relevant to the study of invasive species. A prevailing principle in invasion ecology is that invasion success is well predicted by propagule pressure—a composite measure of both the size and the frequency of introduction events—because increased propagule pressure lessens the negative effects of both demographic and environmental stochasticity on establishment and can also help populations overcome the negative effects of small population size, such as Allee effects or inbreeding depression (Lockwood et al. 2005; Simberloff 2009). However, there is growing evidence that it is important to consider both the size and the frequency of introduction events independently rather than as a composite metric, particularly when there is significant environmental stochasticity or density dependence (Haccou and Iwasa 1996; Haccou and Vatutin 2003; Wittmann et al. 2014; Koontz et al. 2018). Most studies of propagule pressure ignore genetics and evolutionary dynamics (but for a discussion of inbreeding depression, see Cassey et al. 2014), but rapid adaptation is often a major component of successful invasions (Colautti and Lau 2015). In these situations, propagule pressure may not accurately predict invasion success because of the effects of gene flow. We show that propagule pressure is an accurate predictor of invasion success only in mild environments, where establishment does not require much evolutionary change. When species are introduced to sufficiently harsh environments, an aggregate measure of propagule pressure does not accurately predict invasion success because less frequent introductions can facilitate adaptation even if propagule pressure decreases (e.g., our fixed pulse scenario) by allowing transient escapes from gene flow. With negative density dependence, many small introductions tend to be optimal for establishment in mild environments (fig. A3), while in harsh environments establishment is most probable with introductions of intermediate size and frequency (fig. 4*D*, 4*F*, black circles). In agreement with the ecological studies mentioned above, we have shown that in order to accurately predict establishment success, it is important to consider both the size and the frequency of introduction events.

Throughout this study, we used a mixture of deterministic and stochastic models. Our deterministic models require some limiting assumptions but provide direct insight into the forces affecting the dynamics of the system. These are complemented by our stochastic individual-based simulations, which provide more realism by relaxing many of these limiting assumptions but at the cost of the loss of some causal inference. By combining these approaches, we gain robust insights into how variation in immigration rates affects local adaptation. However, as with most theoretical studies, there are still numerous assumptions in our models that may limit the ready applicability of our results to empirical systems. To adequately investigate eco-evolutionary dynamics at the range boundary of any particular species, model assumptions and parameters must be adjusted to fit that system, but in the absence of detailed field measurements, our results can be used to make predictions based on knowledge of a few key ecological and genetic characteristics. The following paragraphs briefly discuss some of our major assumptions.

Many of our main conclusions rely on the assumption that immigrants are adults that have the chance to mate, and their offspring are subject to viability selection. This increases the effect of gene flow because given random mating, many locally maladapted individuals (the immigrants) will mate with better-adapted residents, producing offspring that are less adapted than the offspring of two resident and presumably better-adapted parents. Many organisms disperse as adults, but in other species dispersal is limited to the juvenile life stages (e.g., seed- and pollen-dispersing plants). Our individual-based simulations suggest that with juvenile immigration there is no longer a benefit of spacing out immigration events (fig. 4, white circles), probably because in this

scenario most maladapted immigrants are culled by selection before mating in the sink, which dramatically reduces the effect of gene flow; indeed, surviving juveniles are likely to be well adapted, so this enhances the facilitative effect of immigration on local adaptation. We also assumed a simple life history with only two stages and discrete generations; more work is needed in order to fully understand how temporal variation in immigration affects local adaptation in species with more complex life histories.

Our models also make many assumptions about dispersal. First, we assume that the immigration pool is a random sample of individuals from the source; however, dispersal may be influenced by many individual, social, or ecological conditions (Ims and Hjermann 2001; Clobert et al. 2009). Such condition-dependent dispersal can alter evolutionary dynamics (Ronce et al. 2001; Edelaar and Bolnick 2012). Furthermore, we have assumed that there is one-way dispersal from a single source. Some systems are likely to have strongly asymmetric flows (e.g., aquatic organisms living in streams on steep mountainsides), but in others there could be substantial backflow to sources. In such systems, the assumption of one-way dispersal is not problematic if immigrants are arriving to the sink from a large source population because back migration from sink to source will have little effect on the genetics or demography of the source (at least until the sink has adapted). But if the source population is small, considering back migration can be very important (e.g., Ronce and Kirkpatrick 2001). Few studies have considered evolution to a sink environment when immigrants are arriving from multiple sources, but this could potentially alter our conclusions (e.g., Kolbe et al. 2004; Gillis et al. 2009). Future studies should investigate the effects variation in immigration rates on local adaptation in more complex landscapes.

In this study, we focused on the case in which immigration occurs via pulsed events, which is relevant to the dispersal of many taxa as well as the introduction of nonnative species. That being said, our results may also be relevant to systems in which immigration occurs continuously but varies in magnitude over time. We predict that adaptation is most probable when periods with high immigration are followed by periods with low immigration, because what is important for adaptation in our models is the arrival of many immigrants followed by a relaxation in gene flow. Future studies should investigate more complicated patterns of temporal variation in migration rates going beyond our idealized "on-off" scenarios.

By treating immigration as a temporally constant process, many pertinent and interesting evolutionary dynamics may be obscured. Our findings show that constant immigration can be an agent of niche conservatism and that spacing out immigration events may facilitate niche evolution. Theoretical studies have demonstrated that temporal variation in migration rates can influence a variety of evolutionary processes (Nagylaki 1979; Rice and Papadopoulos 2009; Rice et al. 2011; Baskett et al. 2013; Burgess et al. 2018), but empiricists have yet to test these theoretical predictions. Laboratory-based experiments and studies in natural systems are necessary if we want to understand how variation in migration rates affects local adaptation and ultimately population persistence in the real world. Globalization, climate change, and anthropogenically driven land-use change are likely to alter both the average connectivity of habitats and the temporal variance of such connectivity, so this seemingly abstract issue is likely to have important implications for projections of eco-evolutionary responses to the large-scale impacts humans are at present inflicting on our planet.

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