

Partial Clonality Expands the Opportunity for Spatial Adaptation

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ABSTRACT: Reproductive mode may strongly impact adaptation in spatially varying populations linked by dispersal, especially when sexual and clonal offspring differ in dispersal. We determined how spatial structure affects adaptation in populations with mixed clonal and sexual reproduction. In a source-sink quantitative genetic deterministic model (with stabilizing selection around different optima), greater clonal reproduction or parent-offspring association (a measure of the part of the parent's phenotype other than the additive genetic component inherited by clonal offspring) increased the selective difference (difference between phenotypic optima) allowing sink populations to adapt. Given dispersal differences between clonally and sexually produced juveniles, adaptation increased with an increasing fraction of clonal dispersers. When considering migrational meltdown, partially clonal reproduction reduced cases where dispersal caused habitat loss. Stochastic individual-based simulations support these results, although the effect of differential dispersal was reversed, with decreased clonal dispersal allowing greater adaptation. These results parallel earlier findings that for an instantaneous shift in phenotypic optimum, increasing clonality allowed population persistence for a greater shift; here, selective change is spatial rather than temporal. These results may help explain the success of many partially clonal organisms in invading new habitats, complementing traditional explanations based on avoiding Allee effects.

Keywords: clonal reproduction, dispersal, spatial structure, migrational meltdown.

Introduction

Most species occupy geographic ranges that span considerable variation in the conditions of life that affect components of fitness, leading to the opportunity for local adaptation. Understanding how selection and gene flow jointly influence the pattern of spatially varying adapta-

tion—and the potential for local maladaptation (Brady et al. 2019)—is a classic problem in evolutionary genetics (Gould and Johnston 1972; Kawecki and Ebert 2004). There has been particular focus in recent decades on understanding adaptation in marginal habitats, which may occur at the edges of species' ranges, as well as at more central locations (Kawecki 2008; Sexton et al. 2009). Such habitats may have low population abundance, and thereby asymmetric gene flow from more abundant populations in nonmarginal habitats may constrain local adaptation (genetic swamping; Lenormand 2002), or genetic drift may overwhelm local selection (Polechová and Barton 2015) and allow the buildup of deleterious mutations (e.g., expansion load; Peischl et al. 2013). Together, these maladaptive processes could even lead to local extinction (as in the scenario of migrational meltdown; Ronce and Kirkpatrick 2001).

Empirical studies have found some support for the prediction that marginal populations should show reduced fitness or abundance; for example, in a summary of studies of range limits, Sexton et al. (2009) examined the extent of declines in fitness or abundance at range limits and found support or partial support in 57% and 67% of the studies, respectively. Gaston (2009) found stronger support for a center-to-edge decrease in occupancy than in either local population size or density, while Pironon et al. (2017) showed a strong signal across studies for a decrease in species occurrence (81% of studies); lowered occupancy could reflect elevated extinction risks. On the other hand, Dallas et al. (2017) concluded that species are not necessarily most abundant near range or niche centers; a definitive answer has not been reached on this important empirical question (Soberón et al. 2018; Kottler et al. 2021). There are a number of mechanistic reasons why one might not observe a relationship between niche or range centrality and abundance (Osorio-Olvera et al. 2019; Holt 2020). Moreover,

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spatial variation in maladaptation decoupled from variation in abundance can occur for other reasons, such as asymmetric dispersal (Kawecki and Holt 2002) or density dependence at life stages other than those experiencing selection (e.g., soft selection; Reznick 2016).

Reproductive mode may interact in important ways with such patterns. For example, the phenomenon of geographical parthenogenesis (Gaggiotti 1994; Tilquin and Kokko 2016) predicts that asexuality is more likely to occur in habitats that are in some sense marginal (with “marginal” taking on a multitude of meanings with regard to both biotic and abiotic factors; Tilquin and Kokko 2016), implying that spatial structure may favor asexuality or at least allow the coexistence of sexual and asexual forms across a complex distribution of habitats. In a simulation model allowing asexual individuals to arise within a purely sexual population, Peck et al. (1998) showed that structured habitats with regions where migrants were much more common than non-migrants favored the establishment of asexual lineages over sexual lineages. Both field studies (Dorken et al. 2001, aquatic plant; Karako et al. 2002, sea star; Tatarenkov et al. 2005, brown seaweed) and laboratory experiments (Lagator et al. 2014, *Chlamydomonas*) point to empirical evidence for this relationship between the ability to persist in marginal, low-quality, or novel habitats and asexual forms of reproduction.

In addition to purely asexual forms of reproduction, many taxa have a mixture of sexual and clonal reproduction (partial clonality; Orive and Krueger-Hadfield 2021), and this could influence their spatial patterns of adaptation and maladaptation. Given spatial variation in selection, differences in dispersal for clonal versus sexual offspring may result in important differences in the average selective regime experienced by the two types of offspring, altering the final realized pattern of adaptation. Clonal and sexual offspring often exhibit different adaptations for dispersal (e.g., short-distance clonal or vegetative spread of a plant via runners versus longer-distance propagation via seed), such that organisms with partial clonality will necessarily have heterogeneities among their offspring in their patterns of dispersal. These differing forms of dispersal may involve vastly different average and extreme dispersal distances. Examples can be found across the tree of life: aquatic animals such as the invasive hydrozoan *Cordylophora caspia* can disperse sexually via planula larvae and asexually either via local budding or via potentially widely dispersing somatic fragments (Roos 1979; Chang 2018), while pathogenic fungi such as *Zymoseptoria tritici* (a wheat pathogen) have asexual spores that disperse very locally (~40 cm) via rain splash and sexual ascospores that can disperse by air for hundreds of kilometers (Steinberg 2015; Karisto et al. 2019). Plants may reproduce clonally via vegetative growth (e.g., underground stolons or rhizomes in ramets) over short distances, but sexually produced seeds often can

move over much longer distances. For such taxa, we expect a higher correlation between the parent and offspring environments for clonal than for sexual offspring.

Differences between clonal and sexual offspring dispersal may interact in interesting ways with local population size and density dependence in producing local adaptation. Clonal offspring that are closer to their parents (produced by fragmentation, budding, and other types of clonal spread) may experience more competition with parents where density is high (perhaps in the middle of the range or in favorable habitats) than do more distantly dispersed sexual offspring. This general effect may be lessened in marginal habitats, where population size may be lower and competition less intense; this difference between sexual and clonal offspring may thus differ among habitats.

We will address the fundamental question of whether partial clonality hinders or helps adaptation to a spatially heterogeneous environment, considering a simple landscape of two discrete habitat patches with different selective optima. To accomplish this, we develop models to examine how populations with a mixture of clonal and sexual reproduction adapt to spatially varying phenotypic optima. Previous theoretical work that has considered the interaction between reproductive mode and spatial adaptation has for the most part focused on selfing in sexually reproducing populations (Sachdeva 2019) or has focused on purely sexual or asexual populations (reviewed in Kawecki 2008). A previous treatment considering partial clonality (mixed sexual and asexual reproduction) utilized solely an individual-based simulation model (Bazin et al. 2014). Here, we consider partial clonality using both deterministic analytical modeling and stochastic simulations, investigating how both genetic and demographic stochasticity interact with the processes of mixed reproduction and spatial adaptation.

We first develop a deterministic model for adaptation in two discrete habitats coupled by movement, in which we assume that genetic and phenotypic distributions are Gaussian and the variance of each is fixed. This builds on previously developed models combining the demography of stage-structured populations and phenotypic evolution of quantitative traits (Barfield et al. 2011; Orive et al. 2017). That earlier work developed models for multivariate quantitative traits; here, we consider evolution of just a single trait, z . We then carry out individual-based simulations relaxing assumptions of the deterministic model, in that genetic and phenotypic distributions are allowed to evolve due to mutation, selection, and drift and local populations can become extinct because of demographic stochasticity. The advantage of the deterministic model is that it can shed analytic insight into constraints on local adaptation in heterogeneous landscapes, but it does depend on idealized assumptions. In many cases, we find that the two modeling

approaches agree well, but there are some interesting exceptions, which we will discuss.

Methods

The interplay of the use of deterministic and stochastic simulation approaches to analyze evolution in complex populations continues a strategy we have developed over time. Barfield et al. (2011) developed a general, deterministic discrete-time model of population and evolutionary dynamics in a stage-structured sexual population composed of discrete stages (e.g., life history stages). Assuming that breeding values and phenotypes have Gaussian distributions with constant variances, they showed that one could generalize Lande's (1982) classic theorem for life history evolution to stage-structured populations. Using individual-based simulations in which genotypes and phenotypes could evolve and have non-Gaussian distributions, they showed that the Gaussian assumption (which permits analytical results) provided a surprisingly accurate portrayal of evolution. Orive et al. (2017) extended this framework to encompass a mixture of sexual and clonal reproduction. They also compared deterministic models with the Gaussian assumption to individual-based simulations permitting evolution of genetic variance. Again, the deterministic model provided reasonably good agreement with the fully stochastic individual-based model (Orive et al. 2017, app. B, pt. B, and supplemental fig. B1) when there was low to moderate clonality. Deviations between these two modeling approaches can help identify situations in which there is an important evolutionary consequence of deviations from phenotypic and genotypic normality or constant variances.

Here, we utilize a comparable approach to analyzing how the mixture of sexual and clonal reproduction influences evolutionary dynamics in a spatially structured landscape consisting of two habitat patches between which individuals disperse. We suggest that discrete habitat patches can be viewed as "stages" in a stage-structured population, and with this interpretation the machinery that Barfield et al. (2011) and Orive et al. (2017) developed for analyzing evolution in stage-structured populations can be applied to this problem. As in those articles, we start with a deterministic model in which we make Gaussian, fixed-variance assumptions, and we then develop comparable individual-based simulations that include stochasticity and permit non-Gaussian genotype and phenotype distributions.

Deterministic Model

Basic Model for a Single, Closed Habitat Patch

The organism we consider has a simple life cycle, comparable to an annual plant with discrete nonoverlapping

generations, and both sexual and clonal reproduction. In a single habitat patch without dispersal, the population size recursion is $N' = s(c + f)N$, where N and N' are the number of juveniles in the current and next generation, respectively; s is the average survival probability from juvenile to adult; and c and f are the average number of clonal and sexual offspring per adult. We extend this to two demes below, so that N_i is the juvenile population size in deme i ($i = 1, 2$).

We assume that selection acts on survivorship from the juvenile to the adult stage, such that a juvenile with phenotype z (the trait under selection) has survival $\exp[-(z - \theta)^2 / (2v^2)]$, where θ is the phenotypic optimum and v is the width of the individual survival function. If the distribution of z over the population is Gaussian with mean $\bar{z}$ and variance P , then it can be shown that the average survival over the population is given by $s = s_{\max} \exp[-(\bar{z} - \theta)^2 / (2\omega^2)]$, where $\omega = (v^2 + P)^{1/2}$ is the width of the population survival function and $s_{\max} = (v^2 / (v^2 + P))^{1/2}$ is the maximum population survival (at $\bar{z} = \theta$). Survivorship thus depends on the mean, $\bar{z}$, of a univariate phenotype, z . We assume that it does not depend on the mode of reproduction that generated an offspring.

We assume that $z = g + e$, where g is the additive genetic (breeding) value of the phenotypic trait and e is the non-additive genetic value plus a random environmental deviation, a common assumption in quantitative genetic models (Falconer 1989); z , g , and e are all assumed to be Gaussian with constant variances. (We discuss these assumptions, which are also very common, below in the section "Assumption of Gaussian Distributions with Constant Variances.") As in Orive et al. (2017), we also allow for a relationship between e of a clonal offspring and that of its parent through an association parameter ρ (which is similar to a correlation coefficient; a value of 0 indicates no relationship, and a value of 1 indicates that a clonal offspring's e is the same as its parent's). For sexual reproduction with random mating, there is an uncoupling of the nonadditive genetic and random environmental components of parents and offspring, so $\rho = 0$. Clonal offspring, however, are assumed to inherit the full parental genotype, including dominance and epistatic (nonadditive) genetic interactions, and may also inherit aspects of the environmental (random) component of phenotype (depending on the type of somatic tissue involved in producing clonal offspring; note that this can include cytoplasmic inheritance—for a recent review, see Camus et al. 2022). Types of asexual reproduction involving vegetative propagation and fragmentation (which lack some of the epigenetic resetting mechanisms that take place during gametogenesis and in early zygotes) may, for example, increase the probability of stable transmission of epigenetic markers compared with sexual reproduction (Verhoeven and Preite 2013). Thus, we

allow nonzero values of the association parameter (ρ) for clonal reproduction (see discussion in Orive et al. 2017).

*Extension to Two Habitats Coupled by Dispersal:
A Two-Deme Model*

We initially assume symmetric juvenile movement between the two demes and that the two demes differ in survivorship (and not in allocation to clonal or sexual reproduction). We assume the maximum survival ($s_{\max}$) is the same in both demes, and we focus on systems in which the habitats differ in their phenotypic optima (denoted θ_i). We assume that juveniles disperse, after which there is selection on survival to adulthood; then the survivors reproduce.

Individuals undergoing selection in either of the two demes could have arisen in the same or opposite deme, so we define survival functions for each origin/destination pair. Average survivorship for individuals born in deme j that undergo selection in deme i is given by

$$s_{ij} = s_{\max} \exp[-(\bar{z}_j - \theta_i)^2 / (2\omega_i^2)].$$

Note that the parameters of the selection function (θ_i , ω_i) are those of the habitat in which selection occurs, but the mean phenotype ($\bar{z}_j$) is that of the habitat in which individuals are born.

We allow the probability of movement to differ between offspring produced clonally and those produced sexually. Recall that the order of events each time step is (i) juvenile movement, (ii) survival to adulthood, and (iii) production of new individuals. Of the N_i juveniles in habitat i before dispersal, a fraction $r_c = c/(c+f)$ are clonal, which each move with probability m_c , and a fraction $r_f = f/(c+f)$ are sexual, which move with probability m_f . Therefore, the number that move to the other habitat is $(r_c m_c + r_f m_f)N_i$, and the number that do not is $(1 - r_c m_c - r_f m_f)N_i$. The average number of juveniles born in habitat i that do not move and survive to adulthood is then $(1 - r_c m_c - r_f m_f)s_{ii}N_i$, while the average number of survivors in habitat j among those that moved from habitat i is $(r_c m_c + r_f m_f)s_{ji}N_i$. Fi-

nally, those that survive reproduce, with $c+f$ offspring per adult (assumed equal in the two habitats). Therefore, the average number of juveniles in habitat i in the next generation due to individuals that did not move is $(1 - r_c m_c - r_f m_f)s_{ii}N_i(c+f) = (c+f - cm_c - fm_f)s_{ii}N_i = [c(1 - m_c) + f(1 - m_f)]s_{ii}N_i$, and the average number of offspring in habitat j due to those that moved from habitat i is $(r_c m_c + r_f m_f)s_{ji}N_i(c+f) = (cm_c + fm_f)s_{ji}N_i$.

Therefore, the recursion for population size in habitat i is

$$N'_i = [c(1 - m_c) + f(1 - m_f)]s_{ii}N_i + (cm_c + fm_f)s_{ij}N_j, \quad (1)$$

where either $i = 1$ and $j = 2$ or the reverse. These two equations can be written in matrix form as

$$N' = \mathbf{A}N = \begin{pmatrix} N_1 \\ N_2 \end{pmatrix}' = \begin{pmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{pmatrix} \begin{pmatrix} N_1 \\ N_2 \end{pmatrix}, \quad (2)$$

where, for the simple two-deme model given in figure 1, the transition matrix is

$$\mathbf{A} = \begin{pmatrix} [c(1 - m_c) + f(1 - m_f)]s_{11} & (cm_c + fm_f)s_{12} \\ (cm_c + fm_f)s_{21} & [c(1 - m_c) + f(1 - m_f)]s_{22} \end{pmatrix}.$$

Note that selection in deme i will act on both non-dispersing juveniles (with deme i parents) and dispersing juveniles (with deme j parents). If $m_c < m_f$, increased clonal reproduction could decrease the overall amount of maladaptation in that it causes more juveniles to experience selection in the deme of their parents.

Two-Deme Model with Clonality—Juvenile Genotype

To determine the effect of clonality on phenotypic evolution given spatial structure, we utilized the two-deme model developed above and derived recursions for changes in

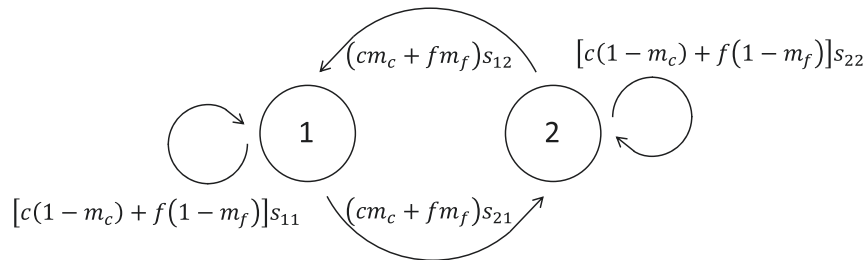


Figure 1: Diagram showing transitions given in equation (2) between two demes in simple model.

mean genotype $\bar{g}_i$ (given here) and mean phenotype $\bar{z}_i$ (next section). The recursion for mean genotype is

$$\bar{g}'_i = \bar{a}_{ii} \frac{N_i}{N'_i} \left(\bar{g}_i + G_i \frac{d \ln \bar{a}_{ii}}{d \bar{z}_i} \right) + \bar{a}_{ij} \frac{N_j}{N'_i} \left(\bar{g}_j + G_j \frac{d \ln \bar{a}_{ij}}{d \bar{z}_j} \right), \quad (3)$$

where overbars indicate population means and G_i is the genetic variance in population i . This equation can be interpreted as follows. A fraction $\bar{a}_{ii}N_i/N'_i$ of the population of habitat i in the next generation are offspring of individuals that were already in habitat i in the current generation (current residents). This is multiplied by the first expression in parentheses, which is just the breeder's equation (the basis of evolutionary quantitative genetics; see Lande 1982) for individuals that did not move from habitat i . Similarly, a fraction $\bar{a}_{ij}N_j/N'_i$ of the population of habitat i in the next generation are offspring of individuals that moved from habitat j in the current generation. This is multiplied by the second expression in parentheses, which is the breeder's equation for individuals that moved from habitat j to habitat i before undergoing selection. Mean genotype is not changed by reproduction, so this weighted average of parental genotypes also applies to their offspring. A full derivation is given in Orive et al. (2017).

Two-Deme Model with Clonality—Juvenile Phenotype

We next derived recursions for the change in mean phenotype, allowing an explicit decomposition of the effects of clonal reproduction on phenotype into genotype-dependent and phenotype-dependent components, mediated by the association parameters ρ_{ij} . The general recursion for phenotype includes the mean contribution to stage i by a stage- j individual via direct transition, sexual reproduction, and clonal reproduction. For the two-deme model considered here, there are no direct transitions from one deme to the other, since our time step is a complete generation and so every transition includes reproduction. The effective habitat-specific sexual and clonal fecundities in each habitat, measured as individuals surviving to reproduction, are $\bar{f}_{ii} = f(1 - m_f)s_{ii}$, $\bar{f}_{ij} = fm_f s_{ij}$, $\bar{c}_{ii} = c(1 - m_c)s_{ii}$, and $\bar{c}_{ij} = cm_c s_{ij}$. The phenotype recursion is

$$\bar{z}'_i = \frac{N_i}{N'_i} \left[\bar{a}_{ii} \left(\bar{g}_i + G_i \frac{d \ln \bar{a}_{ii}}{d \bar{z}_i} \right) + \rho_{ii} \bar{c}_{ii} \left\{ (\bar{z}_i - \bar{g}_i) + \frac{d \ln \bar{c}_{ii}}{d \bar{z}_i} (P_i - G_i) \right\} \right] + \frac{N_j}{N'_i} \left[\bar{a}_{ij} \left(\bar{g}_j + G_j \frac{d \ln \bar{a}_{ij}}{d \bar{z}_j} \right) + \rho_{ij} \bar{c}_{ij} \left\{ (\bar{z}_j - \bar{g}_j) + \frac{d \ln \bar{c}_{ij}}{d \bar{z}_j} (P_j - G_j) \right\} \right], \quad (4)$$

where G_i and P_i are genotypic and phenotypic variances for deme i and ρ_{ij} is the association between the random component of trait z of a parent and its clonal offspring

for a juvenile in deme i that originated in deme j . These equations in their general form allow the association parameters ρ_{ij} to each be different, such that ρ can depend on the habitat before and after dispersal, but for simplicity we will always assume them to be equal (so we drop the subscripts).

The terms beginning with $\bar{a}_{ii}$ and $\bar{a}_{ij}$ in the expressions in brackets above (along with the coefficients in front of each bracketed expression) give the mean genotype and are analogous to equation (3). Unlike mean genotypes, mean phenotypes of offspring are not the same as those of their parents if there is an association in the random component of phenotype. The terms in braces are the breeder's equation for the random component of the phenotype for individuals that stayed in habitat i (top row) and those that moved from habitat j (bottom row), with the difference in mean phenotype and genotype being the mean random component ($(\bar{z}_i - \bar{g}_i) = \bar{e}_i$, etc.). Selection also acts on the random component, which has variance $P_i - G_i$ for habitat i . The effect of this selection is not included with sexual reproduction because it is assumed to be lost during reproduction (with sexual reproduction under random mating, only the additive genetic component of phenotype [breeding value] is usually assumed to be passed to offspring; Falconer 1989). But here, a fraction ρ of both the mean random component and the effect of selection on the random component are retained for clonally produced offspring. Again, the full derivation is given in Orive et al. (2017).

Assumption of Gaussian Distributions with Constant Variances

In deriving the recursions above for both juvenile genotype and phenotype, the joint distribution of genotype and phenotype in each habitat was assumed bivariate Gaussian with constant variances G and P (and correlation equal to the heritability, G/P). These assumptions allowed recursions for the joint probability density function of phenotype and genotype to be simplified into much more straightforward recursions for mean genotype and phenotype. The assumption of Gaussian distributions is often a reasonable approximation for polygenic traits and sexual reproduction for projections of mean population states, even with recombination and strong selection leading to substantial deviations from normality (Turelli and Barton 1994). However, with increasing clonal reproduction, this assumption is likely to become less accurate. In section A of the supplemental PDF, we discuss this issue and present results comparing the use of analytical equations derived here using the Gaussian assumption and the assumption of fixed variances with an individual-based simulation model (discussed below), which does not make either of these assumptions; there is

generally good agreement for moderate levels of clonality and less agreement for quite high clonality and parent-offspring association (both r_c and $\rho \approx 0.89$). This suggests that the assumptions of the deterministic model are often reasonable, even with moderate clonality (fig. S1), although the agreement for mean genotype and phenotype worsens for larger differences in optimum phenotype between the demes (strong selection); this holds even in fully sexual populations. In considering the assumption of constant genotypic and phenotypic variances, with no clonal reproduction, variances are very close to the values of the assumed constant values, with some inflation of the variances due to mixing of different populations, more so when the two optima differ more (figs. S2, S3, top rows). As the amount of clonality increases, we see decreasing phenotypic variance, as we would expect for higher values of ρ . The noisiness of the variance is greater for the phenotypic variance and shows the greatest deviation from approximately constant values with very high clonality and when mixing very different populations (fig. S2L). Developing the deterministic model and then comparing it to a fully stochastic individual-based model (see below) can help identify potential situations where deviations from Gaussian assumptions matter in determining eco-evolutionary expectations.

Individual-Based Simulation Model with Evolving Genetic Variance and Demographic Stochasticity

The model described above is deterministic and assumes that genetic variance is fixed, and moreover that the genotypes and phenotypes have Gaussian distributions. Yet non-Gaussian distributions (with nonconstant variances) might be expected to arise when there are admixtures of migrants from habitats with different selective optima, and the mixture of clonal and sexual reproduction might contribute to non-Gaussian distributions as well. Previous work (Holt and Barfield 2011) has shown that such effects can at times lead to unexpected effects during adaptation to heterogeneous landscapes. To examine the robustness of our conclusions, we extended the individual-based model (IBM) used in Orive et al. (2017) and Orive et al. (2019) to two discrete habitats coupled by movement (see sec. B of the supplemental PDF for details). The IBM incorporates important aspects of stochasticity in both evolution (e.g., mutation, drift) and demography (e.g., demographic stochasticity, which can result in local extinction events, although they are temporary since there is recurrent immigration). The IBM simulated a source-sink system in a population with both clonal and sexual reproduction, with two-way movement between the source population (deme 1) and a sink population (deme 2), initially assuming equal clonal and sexual juvenile migration rates ($m_c =$

$m_f = 0.05$). These simulations parallel the deterministic model presented above in their assumptions about life history, movement, and selection. Because of the stochasticity in the IBM, there is not a sharp transition between adapted and maladapted sinks. Instead, we summarized the simulations by using a metric for the probability of adaptation (which gradually decreased with increasing θ_2). The probability of adaptation is estimated as the fraction of populations able to persist after immigration from the source ceases (after 1,000 generations of immigration for the simulations shown) for each set of parameters. Additional simulation details match appendix A, part B, of Orive et al. (2017), except that we have two populations with movement between them rather than a single population. Here, we used the same parameters as in Orive et al. (2017) except for ceiling density (on the number of mating sites), which was set to $K = 64$ (except as noted below); see section B of the supplemental PDF for details. Note that we do not include a measure of variance in the IBM plots. The number of adapting populations should have a binomial distribution with parameters p (the actual probability of adaptation) and N (the number of runs, which is 400). The variance of each estimate is therefore $p(1 - p)/400$, where p is approximately the estimated value.

Results

Numerical Exploration of the Deterministic Two-Deme Model with Equal Movement Probabilities ($m_c = m_f$)

To determine the effect of increasing clonality on adaptation in a source-sink landscape, we carried out numerical iterations of the deterministic equations for population size, genotype, and phenotype (eqq. [1], [3], [4]). We first assumed equal movement probabilities, $m_c = m_f$ (and the other symmetry assumptions noted above), in which case the effects of clonal reproduction occur via the composite parameter $r_c\rho$ (the product of the fraction of offspring that are clonal and the association between the random component of the phenotype of the parent and clonal offspring). To keep the populations bounded, ceiling density dependence was imposed by limiting the juvenile population to $K = 256$ in each habitat (by imposing mortality independent of phenotype or type of offspring when the number of juveniles exceeded K). Many ecologists find that a ceiling form of density dependence is reasonable at least for some species (e.g., Hanski et al. 1996), and it is an approximation for the many species that show weak density dependence at low to intermediate densities, which strengthens sharply near carrying capacity. The effect of this form of density dependence on equations (1)–(4) is to replace N_i (but not N'_i) by $\min(N_i, K)$. We assumed that deme 1 is a source (i.e., the habitat where the population initially has a positive growth rate) with phenotypic optimum θ_1 set to 0 and usually that

deme 2 is an initially empty habitat (the sink) with $\theta_2 > 0$ (so increasing values of θ_2 indicate increasing differences in the phenotypic optimum between the two demes, making adaptation in the sink more difficult). Our results indicate that greater clonality or parent-offspring association (greater $r_c\rho$) increases the difference in the phenotypic optimum that allows the population to adapt in deme 2 (fig. 2A). We note that for the parameter values used in figure 2, a phenotypic optimum of $\theta_2 > 2.05$ for habitat 2 indicates a sink (where immigrants adapted to θ_1 have an absolute fitness < 1 in habitat 2). Even in the absence of clonality, moderate sinks can adapt despite gene flow (in the current instance, θ_2 from 2.05 to about 2.8), but more severe sinks remain sinks. Moreover, when adaptation occurs, we see an increasing difference between the final mean phenotype and the final mean genotype in deme 2 under increasing clonality, with the phenotype getting closer to the optimum, while the genotype falls further away (fig. 2B).

In figure 2B, both genotype and phenotype initially move closer to the deme 2 optimum (closer to 1.0) as we increase the difference in optima between the two demes starting from zero. For these small differences in optima, immigrants are not too maladapted and so are able to create a population in deme 2 that can persist and become perfectly adapted, were dispersal discontinued. However, if dispersal continues the decreased adaptation seen for values of θ_2 close to θ_1 (values of θ_2 near zero) arises because more dispersers from deme 1 survive in deme 2 when the two optima are close in value. Gene flow thus moves the mean phenotype in deme 2 closer to θ_1 and thus causes more maladaptation in deme 2. As the difference in the optimum between the two demes increases (greater θ_2), there is stronger selection on migrants, which due to the order of life history events we have assumed (movement, then selection, followed by reproduction) leads in effect to a decrease in gene flow, and hence less maladaptation, until a critical value of θ_2 is reached, above which dispersers are unable to create a persistent population in deme 2. (This effect would likely be different for a different ordering of life history events.) The final population size of deme 1 is identical to that of deme 2 when the latter becomes adapted because back-migration similarly causes maladaptation in deme 1. When deme 2 does not become adapted, there is little back-migration, so population 1 stays near its optimum phenotype and is near its carrying capacity.

We also see greater maladaptation in the genotypic state with increasing $r_c\rho$ (the solid lines in fig. 2B are increasingly further from 1 as $r_c\rho$ increases); here, increased clonality allows maladapted genotypes to persist due to the random environmental component of phenotypes, which can allow an individual to have a well-adapted phenotype despite having a genotype that is far from the optimum. We note that under complete clonality ($r_c\rho = 1$; fig. 2, pink

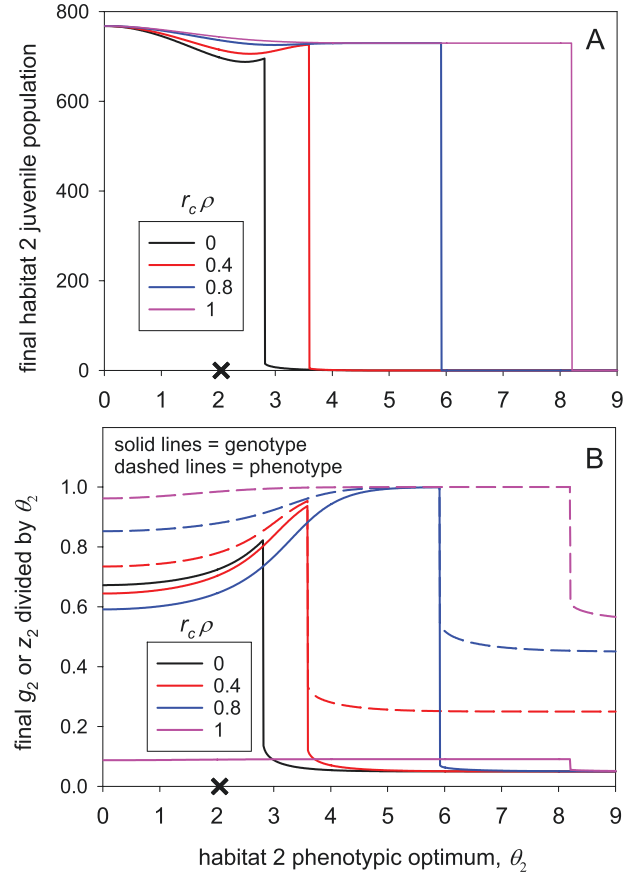


Figure 2: Effect of increasing clonality ($r_c\rho$) on final juvenile population size before density dependence (A) and normalized mean genotype and phenotype (B) in deme 2 under equal movement rates. $\theta_1 = 0$; increasing values of θ_2 give increasing differences in phenotypic optimum between the two demes. Genotype and phenotype in B are normalized to θ_2 ; a value of 1 indicates a mean genotype or phenotype exactly at the optimum. Parameters used are $G = 0.1$, $P = 1.1$, $\omega^2 = 2$, $s_{\max} = 0.75$, $b = c + f = 4$, $r_c = \rho$, and $m_c = m_f = 0.05$ in both directions. For these parameter values, $\theta_2 > 2.05$ is the critical value where habitat 2 becomes a sink (indicated by an X mark on the abscissa; see text for details).

lines), the final normalized mean genotype remains quite far from the optimum in deme 2, while the phenotype there is well adapted. We can show that under this symmetric case at equilibrium with both demes phenotypically adapted to their optima, the normalized mean genotype in deme 2 is approximately the heritability (G/P ; see sec. C of the supplemental PDF). We further note that the critical value of θ_2 for the case of complete clonality ($r_c\rho = 1$) is an artifact of the assumptions of the deterministic model; with a Gaussian distribution of phenotypes there will always be movement of some individuals that are adapted to the deme 2 optimum, which will produce clonal offspring with that same phenotype, and so there will never be a difference in optimum that prevents adaptation, with no genetic

mixing of immigrants and residents. Note that complete clonality is obviously a special case and often very different from even very high clonality with a small amount of sexual reproduction.

To characterize what determines the maximum difference in phenotypic optimum that allows for adaptation (viz., the threshold value of θ_2 , at which the sharp change in final population size and final genotype/phenotype shown in fig. 2 occurs), we numerically solved equations (1), (3), and (4), given symmetry between the two demes and equal clonal and sexual migration rates ($m_c = m_f$), and then found the threshold θ_2 as a function of $r_c\rho$ (fig. 3). In both panels, the solid line is for the parameter values used in figure 2, showing that the threshold θ_2 increases in an accelerating manner with increasing $r_c\rho$. The effects of increasing either the underlying genotypic (G) or phenotypic (P)

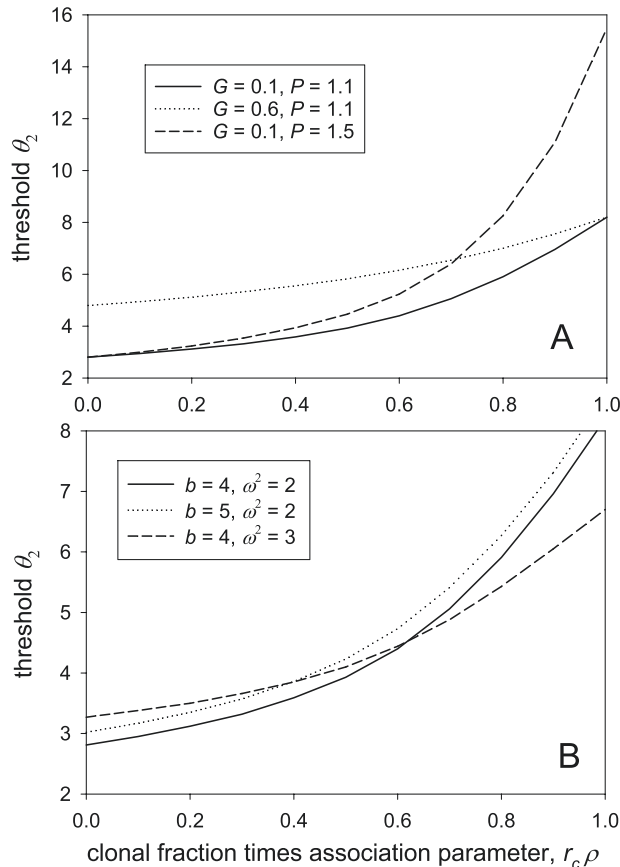


Figure 3: Effect of increasing clonality ($r_c\rho$) on the maximum difference in phenotypic optimum that allows adaptation in the sink deme (threshold θ_2), for increasing underlying genotypic (G) or phenotypic (P) variance for the trait, z (A), and increasing total number of offspring (both clonal and sexual, b) or increasing width of the survival function (ω^2 ; B). For both A and B, the solid line is for the same parameter values used in figure 2. The threshold was calculated to the nearest 0.01 for $r_c\rho$ a multiple of 0.1.

variance for the phenotypic trait are shown in figure 3A. Increasing G (dotted line) boosts the threshold θ_2 compared with the baseline for low values of clonality but converges to the baseline as we move to completely clonal reproduction ($r_c\rho = 1$). Conversely, increasing P (dashed line) progressively increases the threshold θ_2 as clonality increases because more clonality allows the population to tap into additional components of phenotypic variance (nonadditive genetic and random environmental) permitting adaptation in the sink population.

Examples of the effects of either increasing the total number of offspring (both clonal and sexual, b) or increasing the width of the survival function (ω^2) are shown in figure 3B. (Note that $c = br_c$ gives the number of clonal offspring and $f = b(1 - r_c)$ gives the number of sexual offspring per adult.) Increasing the overall reproductive rate increases the threshold θ_2 across all values of clonality (dotted line), as we might expect. A wider survival function ($\omega^2 = 3$, dashed line; weaker selection) increases the threshold θ_2 for lower values of clonality while decreasing the threshold as clonality increases. A similar pattern results if there is no clonality and G is increased while keeping P constant, which therefore increases heritability G/P (fig. S4). Of course, the threshold increases with increasing G , but with weaker selection the threshold is higher at low G and lower at high G than with stronger selection. A likely reason for this pattern in both cases is that gene flow is limiting adaptation, and gene flow has a larger effect for high clonality (high $r_c\rho$) or high heritability (high G). This is because with high clonality or high heritability, dispersers to deme 2 more closely resemble their parents, which survived selection in deme 1, and because weaker selection allows more dispersers to survive in deme 2. The larger values of threshold θ_2 for high $r_c\rho$ or G also makes survivors more disruptive to adaptation. (Although increasing G without clonality increased the adaptation threshold, it did not of course lead to differences between mean juvenile genotype and phenotype, and it caused a more linear increase in threshold than did increasing r_c or ρ , so there are important differences between increasing heritability and clonality.)

IBM Simulations—Equal Movement Probabilities ($m_c = m_f$)

Figure 4 shows characteristic results of the IBM simulations assuming equal movement probabilities for clonal and sexual offspring. The results shown in figure 4 demonstrate that increasing either the relative proportion of clonal reproduction (r_c ; fig. 4A) or the association parameter (ρ ; fig. 4B) usually increased the probability of adaptation for a given difference between source and sink phenotypic optima. This broadly agrees with the results from our analytical

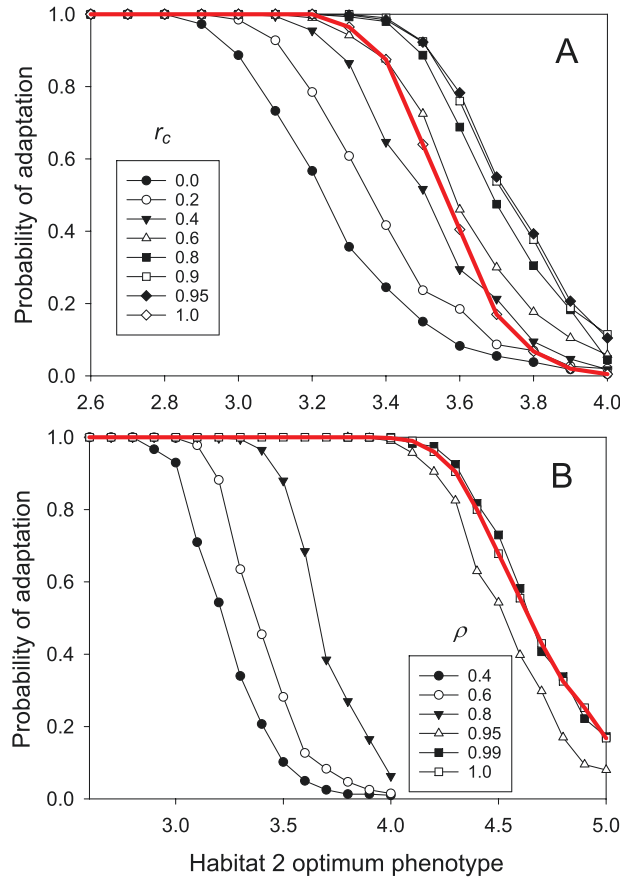


Figure 4: Probability of adaptation in the sink deme as a function of the difference in phenotypic optima, using an individual-based model. As in figure 2, $\theta_1 = 0$, so increasing values of θ_2 indicate increasing differences in the phenotypic optima between the two demes. A, Effect of varying the relative amount of clonal reproduction (r_c), with $\rho = 0.5$. B, Effect of varying the association parameter (ρ), with $r_c = 0.5$. Lines traced in red indicate $r_c = 1$ (A) and $\rho = 1$ (B). Parameters are $m_c = m_f = 0.05$, $K_1 = K_2 = 64$, $b = 4$, $v_i^2 = 1$, $n = 10$, $\alpha^2 = 0.05$, and $\mu_g = \mu_s = 0.001$ (A) or $\mu_g = 0.001$ and $\mu_s = 0.00001$ (B).

treatment (fig. 2) in that increasing clonality permits a greater degree of selective difference between the two demes before deme 2 fails to adapt. However, for very high values of either r_c or ρ , there was little change with a further increase except for a significant decrease when r_c increased from 0.95 to 1 (complete clonality), when all of the additional genetic variation introduced by sexual reproduction is lost (Bengtsson 2003). Therefore, increasing clonality increases adaptation to a marginal habitat as long as there is at least a small amount of sexual reproduction.

Note that for the simulations shown in figure 4A, we increased the somatic (asexual) mutation rate to equal the gametic (sexual) mutation rate ($\mu_s = \mu_g = 0.001$). A low rate of somatic mutation decreases the source genetic var-

iation; the effect of changing the amount of clonality combines the direct effect of clonality on adaptation in the sink and this lower source variance, which work in opposite directions. The result is a very small effect of different amounts of clonality when the asexual mutation rate is much lower than the sexual mutation rate, except for the case of completely clonal reproduction ($r_c = 1$), where the probability of adaptation is lower (see fig. S5).

Effect of Differential Clonal and Sexual Dispersal ($m_c \neq m_f$) in the Deterministic Two-Deme Model

An important extension of this initial symmetric model is to consider the impact of different patterns of asymmetry, especially differences in dispersal of clonally and sexually produced juveniles, so $m_c \neq m_f$. The effects of clonal reproduction now occur separately via r_c and ρ , but we still assumed the simplest case of no deme effect on the association parameter, so that all $\rho_{ij} = \rho$. We again carried out numerical iteration of the deterministic equations for population size, genotype, and phenotype (eqq. [1], [3], [4]), holding the total proportion of offspring that move fixed ($r_c m_c + r_f m_f = 0.05$) and varying the fraction of migration that is due to clonal offspring ($M_c = r_c m_c / (r_c m_c + r_f m_f) = 0.1, 0.5, 0.9$). Figure 5 shows the final juvenile population size before density dependence (fig. 5A, 5C, 5E) and normalized mean genotype and phenotype (fig. 5B, 5D, 5F) in deme 2. Decreased clonal migration ($M_c = 0.1$; red line in fig. 5A, 5C, 5E) decreases the difference in phenotypic optimum allowing adaptation, while increased clonal migration ($M_c = 0.9$; pink lines in fig. 5A, 5C, 5E) increases the maximum difference in θ permitting adaptation in the sink.

Increasing the association parameter ρ from 0.5 (fig. 5A, 5B) to 0.8 (fig. 5C, 5D) increases the maximal difference in phenotypic optimum that allows the population to adapt in deme 2 and also increases the relative effect of asymmetric clonal and sexual offspring migration (in the figure, this increases the spacing between lines). Increasing the fraction of reproduction that is clonal (r_c) from 0.5 (fig. 5A, 5B) to 0.8 (fig. 5E, 5F) again increases the difference in phenotypic optimum that allows the population to adapt in deme 2. More clonal migration (given that $\rho > 0$) allows adaptation at higher θ_2 (a greater difference in optimum between the two demes). Clonal migrants are less likely to survive in the sink because they have a closer phenotypic match to their source-adapted parents than do sexual offspring; their random component of phenotype is closer to the source optimum (partially inherited from their source-adapted parents), and they therefore generate a lower effective amount of gene flow. This results in less of an effect of gene flow in preventing adaptation to the sink (less genetic swamping). Higher ρ and r_c increase this effect.

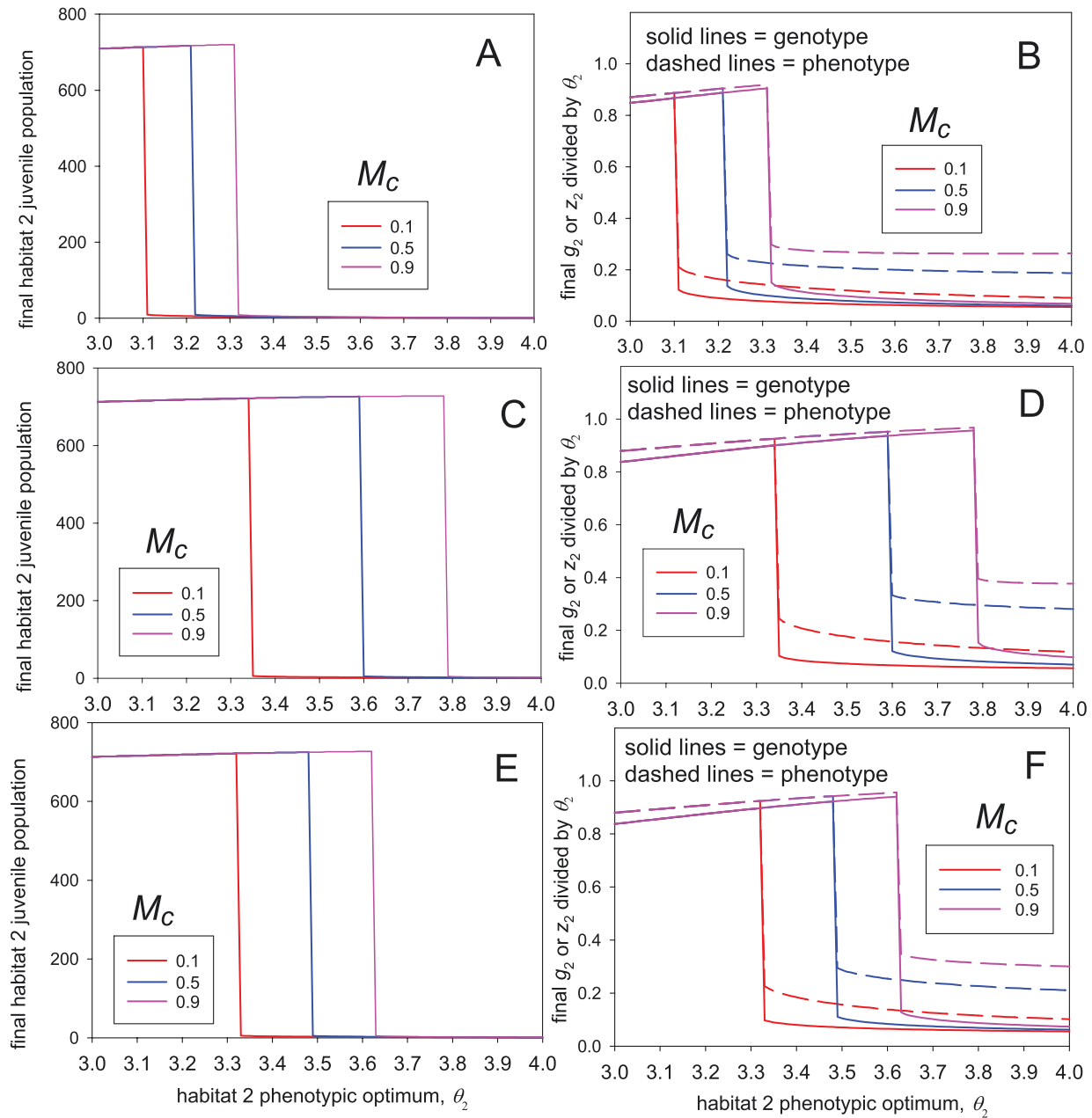


Figure 5: Effect of different clonal movement fractions ($M_c = r_c m_c / (r_c m_c + r_f m_f)$) on final juvenile population size (A, C, E) and normalized mean genotype and phenotype (B, D, F) in deme 2. The total proportion of offspring that move is fixed ($r_c m_c + r_f m_f = 0.05$), while the fraction of dispersal due to clonal offspring varies ($M_c = 0.1, 0.5$, and 0.9 ; red, blue, and pink lines, respectively). $\theta_1 = 0$; increasing values of θ_2 give increasing differences in phenotypic optimum between the two demes. Genotype and phenotype in B, D, and F are normalized to θ_2 ; a value of 1 indicates a mean genotype or phenotype exactly at the optimum. A, B, $r_c = 0.5$, $\rho = 0.5$; C, D, $r_c = 0.5$, $\rho = 0.8$; E, F, $r_c = 0.8$, $\rho = 0.5$.

Differences in clonal and sexual offspring dispersal have almost no effect on the genotype and phenotype for those populations that are able to adapt (fig. 5B, 5D, 5F; for θ_2 lower than the steep drop off, only the phenotype and genotype curves for $M_c = 0.9$ are visible because all three M_c values give indistinguishable results). However, for pop-

ulations that are not able to adapt (fig. 5B, 5D, 5F; for θ_2 greater than the steep drop-off), increasing the relative amount of clonal migration acts to increasingly separate the genotype and phenotype for the migrant individuals entering and surviving in deme 2. The majority of successful dispersers moving into the maladapted deme are successful

because of the value of the random environmental component of their phenotype (e). This can lead an individual with a phenotype close enough to the optimum to survive, despite having a genotype that may be far from the local optimum (but with an on-average positive genotype, since that also increases the phenotype). These successful individuals then pass their genotype to their offspring. With clonal offspring, part of the parent's high random component of phenotype is also passed, so with an increased clonal dispersal rate the average offspring phenotype increases while the genotype may not, leading to a larger gap. Although we see this gap only for populations that do not adapt (to the right of the steep drop-off), there is likely a similar gap before successful populations adapt (to the left of the steep drop-off). In both cases, clonal offspring of sink immigrants that survived selection are more likely than new immigrants or sexual offspring to survive selection, and offspring in the successive generations should be likewise better adapted (and this is one of the reasons we see more adaptation with increased clonal dispersal).

In figure 6, we plot the threshold value of θ_2 as a function of the fraction of movement that is due to clonal offspring, for various values of r_c and ρ . While increasing the clonal reproduction fraction r_c nearly uniformly increases the threshold value (compare the solid and dashed curves), increasing the association parameter ρ has a strongly positive interaction with the increasing clonal fraction of dispersers (compare the dashed and dotted curves). Overall, the effect of the clonal dispersal fraction on the thresh-

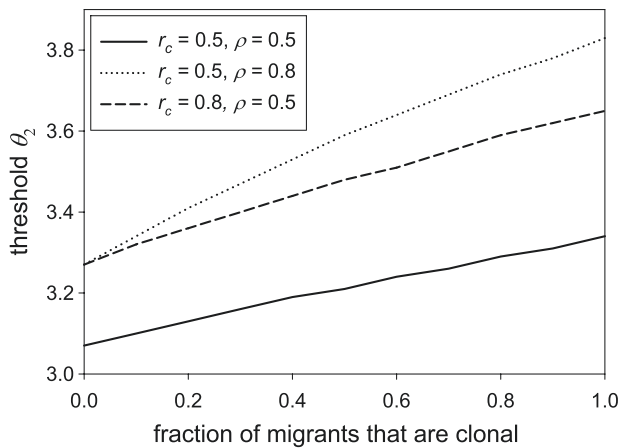


Figure 6: Effect on the threshold value of θ_2 of increasing the fraction of movement due to clonal offspring $M_c = [r_c m_c / (r_c m_c + r_f m_f)]$, for $r_c = 0.5$, $\rho = 0.5$ (solid line); $r_c = 0.5$, $\rho = 0.8$ (dotted line); and $r_c = 0.8$, $\rho = 0.5$ (dashed line). Note that the threshold was calculated to the nearest 0.01 for M_c a multiple of 0.1, which causes the slight waviness in the curves.

old value of θ_2 shown in figure 6 is much weaker, and more linear, than is the effect of the combined clonality parameter $r_c \rho$ shown in figure 3.

Effect of Differential Clonal and Sexual Offspring Migration ($m_c \neq m_f$) in the IBM Simulations

We next consider stochastic IBM simulations of the asymmetric dispersal case explored analytically in figure 5, with unequal clonal and sexual juvenile migration (fig. 7). In these simulations, we utilized the same fixed total migration rate, the same three values of M_c , and the same three pairs of r_c and ρ values considered in figure 5 for the deterministic model. For all of these examples, as we increase the fraction of dispersal that is due to clonal offspring (M_c), the probability of adaptation for a given difference in phenotypic optimum is intriguingly decreased, in marked contrast to the analytical results where increased clonal movement increased the threshold difference in phenotypic optima permitting adaptation. We see the strongest effect of increasing M_c when the association parameter ρ is high; under high phenotypic association between parents and their clonal offspring, dispersing clonal offspring will tend to have a value of e that corresponds well to the phenotypic optimum of deme 1 and are less likely to survive in deme 2. Sexual offspring that disperse, on the other hand, have random e and thus greater phenotypic variability. Clonal migrants will be closer to the source optimum than sexual migrants, making the former more likely to be eliminated by selection. Also, if any do survive, their clonal offspring will inherit part of their e , making it more likely that they will not survive selection. With greater clonal dispersal, the sink population will thus be more susceptible to extinction under demographic stochasticity, forcing adaptation to start over with the next pulse of immigrants. Such recurrent extinction due to demographic stochasticity is not incorporated into the analytical model, in which population size is a continuous variable that never reaches zero (we never rounded a low value down to zero).

This stochastic effect is due to the small size of the sink deme when that population is very maladapted; figure S6 shows the results for additional simulations run for the same simulation parameters as figure 7A but with the adult ceiling population density at $K = 1,024$ for both demes (vs. $K = 64$ in fig. 7; juvenile population size was 4,096 in fig. S6). While the curves are shifted to the right, showing that the deme 2 phenotypic optimum that allows for adaptation is raised due to the larger population size, the overall pattern remains the same, with larger fractions of migrants being clonal (larger M_c) leading to lower probabilities of adaptation. The increase in the source population size and therefore the number of sink immigrants is

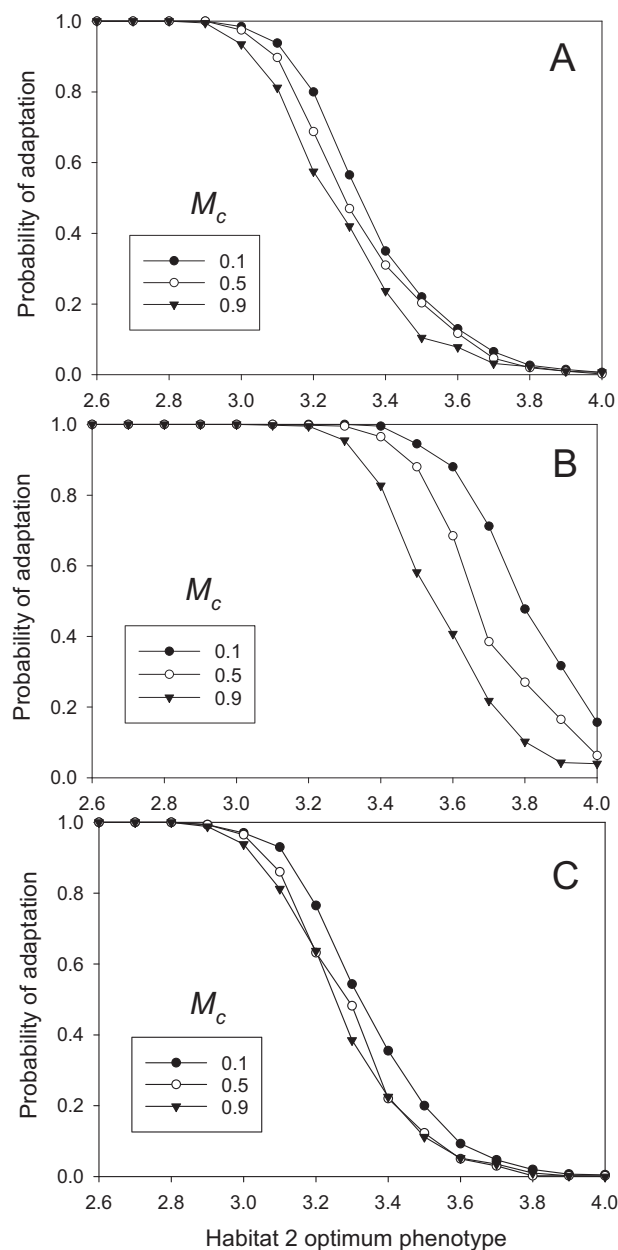


Figure 7: Effect of different clonal migration fractions ($M_c = r_c m_c / (r_c m_c + r_f m_f)$) on the probability of adaptation in the sink deme as a function of the difference in phenotypic optimum, using an individual-based model. The total proportion of offspring that migrate is fixed ($r_c m_c + r_f m_f = 0.05$), while the fraction of migration due to clonal offspring varies ($M_c = 0.1, 0.5, 0.9$). $\theta_1 = 0$; increasing values of θ_2 give increasing differences in phenotypic optima between the two demes. A, $r_c = \rho = 0.5$ (corresponding to fig. 5A, 5B); B, $r_c = 0.5$, $\rho = 0.8$ (fig. 5C, 5D); C, $r_c = 0.8$, $\rho = 0.5$ (fig. 5E, 5F). Other parameters are as in figure 4.

counterbalanced by the increase in sink optimum phenotype, so the number of surviving immigrants is low in both cases where adaptation probability drops.

To see the interaction between the association parameter (ρ) and the clonal movement fraction (M_c) more clearly, figure 8 depicts IBM simulation results for varying values of ρ and three values of M_c ($M_c = 0.1$, dotted lines; $M_c = 0.5$, dashed lines; $M_c = 0.9$, solid lines). For all values of $\rho > 0$, there is a decreased probability of adaptation in deme 2 as the clonal movement fraction increases, with an increasing effect as ρ increases (spacing between the lines is greatest for larger values of ρ).

Environmental Asymmetry and Migrational Meltdown in the Deterministic Model

Another important form of asymmetry to consider is environmental asymmetry, which could include asymmetric dispersal between the two demes and differences in habitat size or carrying capacity between the demes. Prior theoretical work has demonstrated the importance of dispersal asymmetry in biasing natural selection toward source habitats, where the reproductive value of individuals is often (although not always) higher (Holt 1996; Rousset 1999; Kawecki and Holt 2002; Loreau et al. 2013). We consider an example with different carrying capacity here; future

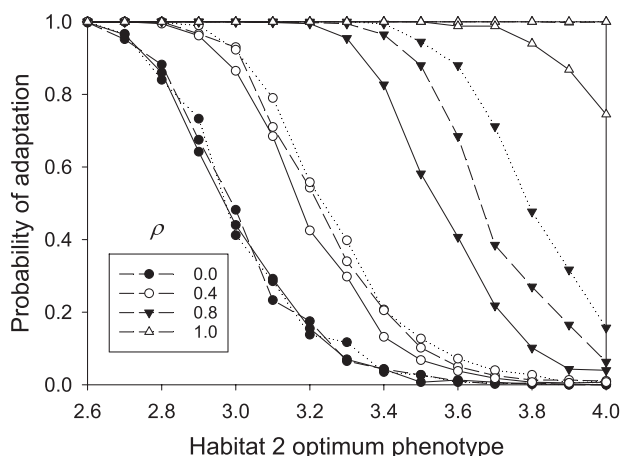


Figure 8: Interaction of different clonal dispersal fractions ($M_c = r_c m_c / (r_c m_c + r_f m_f)$) and association parameters (ρ) on the probability of adaptation in the sink deme as a function of the difference in phenotypic optima, using an individual-based model. The total proportion of offspring that move is fixed ($r_c m_c + r_f m_f = 0.05$), while the fraction of dispersal due to clonal offspring varies ($M_c = 0.1$, dotted lines; $M_c = 0.5$, dashed lines; $M_c = 0.9$, solid lines). $\theta_1 = 0$; increasing values of θ_2 give increasing differences in phenotypic optima between the two demes. Other parameters are as in figure 4.

extensions of the models presented in this article will consider asymmetric dispersal.

The ability of a strictly sexual species to support specialization in different habitats was shown to depend on the amount of connectivity by Ronce and Kirkpatrick (2001), with specialists with narrow niches arising under intermediate dispersal rates, while both very low and very high rates of dispersal led to the evolution of generalists. An open question is how the magnitude of connectivity interacts with reproductive mode in allowing habitat specialization versus habitat loss due to movement and maladaptive evolutionary processes (migrational meltdown; Ronce and Kirkpatrick 2001). As an initial investigation of how partial clonality influences such meltdown, we considered the case of two demes with different optimal phenotypes and different carrying capacities (equivalent to different deme areas or differences in resource availability, for example) and examined the effect of partial clonality on dispersal-induced population loss in the smaller deme (i.e., migrational meltdown).

We again carried out numerical iteration of the deterministic equations for population size, genotype, and phenotype (eqq. [1], [3], [4]), assuming equal movement rates ($m_c = m_f$) as well as the other symmetry assumptions we have made previously, so that once again the effects of clonal reproduction occur via the composite parameter $r_c \rho$. We now assume that the two demes differ not only in their optimal phenotypes but also in their carrying capacities (maximum number of juveniles), with $K_1 = 256$ and $K_2 = 128$. Figure 9 shows the results of these numerical iterations for final deme 2 population size (fig. 9A) and final normalized deme 2 genotype and phenotype (fig. 9B) for two values of the composite clonality parameter ($r_c \rho = 0, 0.09$) and for two different initial populations of deme 2. In one case deme 2 is initially empty ($N_{2,0} = 0$), and as above these results characterize how dispersal influences adaptation to the conditions of deme 2. The other case is for deme 2 to be initially at its carrying capacity ($N_{2,0} = K_2 = 128$) and adapted there (mean phenotype equals the deme 2 optimum); with this initial condition, dispersal can lead to loss of adaptation in the habitat with lower abundance (which makes that local population more vulnerable to migrational meltdown). Note that in these examples, we have used a higher movement rate than above ($m_c = m_f = 0.2$ rather than 0.05).

Matching what we showed above, when considering dispersal into an empty sink deme, increasing clonality (increasing the product of the relative proportion of clonal reproduction and the phenotypic association for clonal offspring) increases the difference between the two deme optima that allows adaptation in the sink deme (black vs. blue lines in fig. 9). When deme 2 was initially populated and well adapted, without clonality (red lines in fig. 9) there is an

intermediate range of θ_2 , approximately between 3.5 and 4.2 for these parameter values, where the population size plummets and the mean genotype and mean phenotype drop very far from the optimum. This is an example of migrational meltdown, where dispersal from deme 1 is causing maladaptation in deme 2. For smaller values of θ_2 , dispersers from deme 1 are not maladapted enough in deme 2 to disrupt adaptation there, and for higher values of θ_2 , immigrants from deme 1 into deme 2 are so far from the deme 2 optimum that they are removed by selection prior to reproduction and so have little effect. The addition of moderate clonal reproduction (pink lines in fig. 6) eliminates this region of migrational meltdown by reducing the effective amount of gene flow into deme 2 from deme 1.

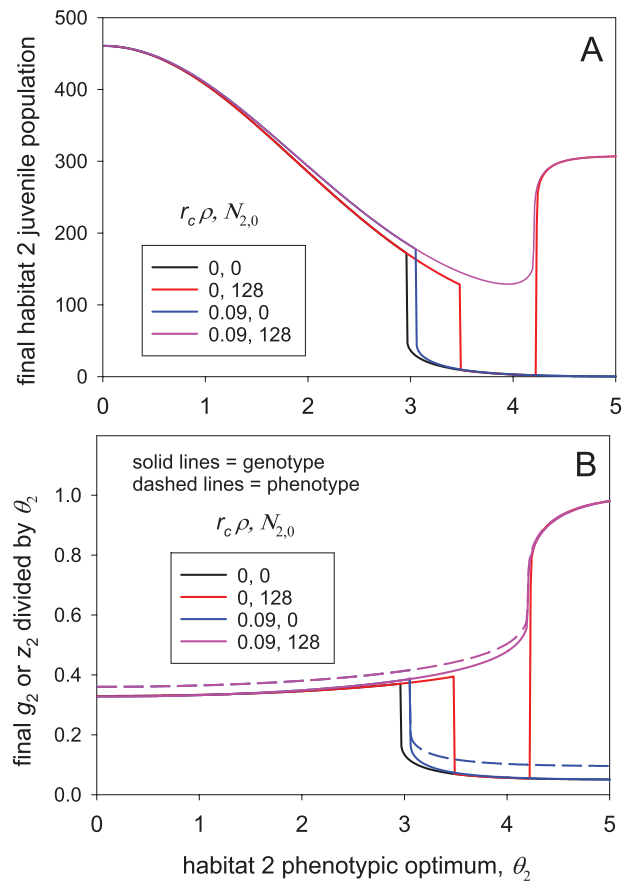


Figure 9: Effect of increasing clonality ($r_c \rho$) and initial deme 2 population size ($N_{2,0}$) on final juvenile population size before density dependence (A) and normalized mean genotype and phenotype (B) in deme 2 under equal migration rates. $\theta_1 = 0$; increasing values of θ_2 give increasing differences in phenotypic optimum between the two demes. Genotype and phenotype in B are normalized to θ_2 ; a value of 1 indicates a mean genotype or phenotype exactly at the optimum. Parameters used are as in figure 2, except that $K_1 = 256$, $K_2 = 128$, and $m_c = m_f = 0.2$ in both directions.

*Environmental Asymmetry and Migrational
Meltdown in the IBM*

Finally, we carried out stochastic IBM simulations of the symmetric dispersal case with different deme carrying capacities, matching the examples of the deterministic model presented in figure 9, which illustrated an effect of partial clonal reproduction on migrational meltdown (fig. 10). We again examined two contrasting situations: when deme 2 is initially empty ($N_{2,0} = 0$; fig. 10A) and when deme 2 starts at carrying capacity ($N_{2,0} = K_2 = 64$) and is initially adapted (mean phenotype equals the deme 2 optimum; fig. 10B). In both cases, $K_1 = 128$.

As was seen with the deterministic model, increasing clonality (r_c, ρ) increases the difference between the two deme optima that allows adaptation in the sink deme; a larger

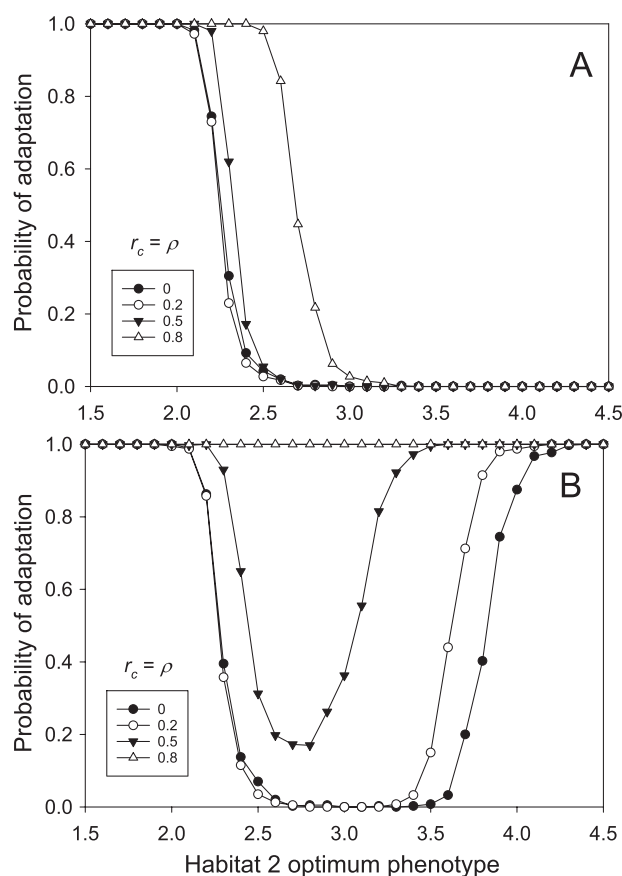


Figure 10: Effect of increasing clonality (r_c, ρ) on the probability of adaptation (at the end of the simulation) in the smaller deme as a function of the difference in phenotypic optimum, using an individual-based model. $\theta_1 = 0$; increasing values of θ_2 give increasing differences in phenotypic optimum between the two demes. A, $N_{2,0} = 0$; B, $N_{2,0} = K_2 = 64$ and initial mean phenotype is near the deme 2 optimum. Parameters used are as in figure 4, except that $K_1 = 128$, $K_2 = 64$, $b = 2$, and $m_c = m_f = 0.2$ in both directions.

deme 2 optimum is needed before the probability of adaptation goes to near zero with increasing clonality (fig. 10A). When dispersal is into a well-adapted but smaller deme at carrying capacity (fig. 10B), we again see the migrational meltdown behavior shown by the deterministic model in figure 9. As we increase the amount of clonality, we see a much weaker effect on the probability of adaptation, with no reduction for high levels of clonality ($r_c = \rho = 0.8$, $r_c \rho = 0.64$). Thus, the effect of increasing clonality is qualitatively comparable for both the deterministic model and the stochastic simulations—it reduces the parameter space for migrational meltdown in terms of the maximal tolerable difference in optimal phenotype between the two demes, and it decreases the probability of meltdown for some parameter values.

Discussion

The results from both our analytical and our numerical exploration of a simple two-deme deterministic model, as well as from stochastic IBM simulations with a similar spatial structure, show that partial clonality (the existence of both sexual and clonal reproduction in the same life history) can expand the opportunity for spatial adaptation, allowing adaptation in a sink deme with a different phenotypic optimum than the source. These results parallel our earlier finding that after a single time-step shift in optimum for a population with no spatial structure, increasing clonality allows a population to more effectively utilize standing genotypic and phenotypic variation and thereby persist (Orive et al. 2017); here, a change in selection over space substitutes for an abrupt change in selection over time. (A difference to note is that with the single population, all individuals undergo the change at the same time, whereas in the source-sink system, a fraction of the source is exposed to the change each generation.) This general conclusion holds true for both a completely symmetric dispersal scenario, where both clonal and sexual juveniles move at equal rates, and for cases where the fraction of migration due to clonal offspring is either higher or lower than that for sexual offspring. We note that the analytical model developed here focused on the mean ($\bar{z}$) of a univariate Gaussian phenotype, z . It is straightforward to expand this to the multivariate case, which in future work could allow consideration of interesting cases involving the interaction of phenotypic covariance and spatial structure.

We focus mostly on adaptation in the sink of a source-sink system, in which the sink population tends to be small prior to adaptation, and so we expect little density dependence. Once adaptation occurs in the sink, density dependence is needed to limit the population size there (and also in the source population). We thus utilize ceiling density dependence here, as a reasonable approach,

assuming that adaptation mostly occurs before the population grows large enough for significant density dependence. Examining the consequences of alternative functional forms for density dependence could be a worthwhile topic (e.g., by adding partial clonality to the models in Filin et al. 2008), but is beyond the scope of this article.

The results from our stochastic IBM simulations match results reported by Bazin et al. (2014), who showed a greater probability of adaptation in a sink habitat with an increasing rate of asexuality (except for very high rates or complete asexuality). While their simulation-based study considered only one-way migration and differed in some other important details from our models, including by excluding selfing and not considering the additional inheritance of phenotype during clonal reproduction considered by our model, the overall result that partial asexual reproduction (partial clonality) is beneficial for invasion of a new environment is a general conclusion of both studies. Furthermore, both studies showed that the rate of asexual or clonal reproduction that corresponded to the highest probability of invasion into a new sink habitat was quite high; Bazin et al. (2014) found that the maximum invasiveness for their results occurred at an asexuality rate (r) of 0.95, while the maximum probability of adaptation in the sink in our results occurs for $r_c = 0.95$ (with $\rho = 0.5$) and $\rho = 0.99$ (with $r_c = 0.5$; fig. 4). There is some empirical support for these general theoretical results from studies of invasion biology, such as the finding that rare sexual reproduction combined with clonal propagation proved to be the best strategy for invasiveness in a freshwater gastropod (Facon et al. 2008).

Barfield et al. (2011) presented a general deterministic schema for analyzing evolution in stage-structured populations, assuming sexual reproduction. This approach was generalized to encompass organisms with partially clonal reproduction by Orive et al. (2017), who explored how a mixture of reproductive strategies influenced the likelihood of evolutionary rescue in abruptly changed environments. Here we have provided a further extension to the Barfield et al. (2011) approach, where “stage” now equals “habitat,” and we have used this extension to examine the influence of partial clonality on local adaptation in heterogeneous landscapes. Our comparison of the deterministic model results with individual-based simulations shows that assuming Gaussian phenotype and genotype distributions with constant variances, although not completely realistic, nonetheless provides quite accurate insights into evolution over a broad range of circumstances. More broadly, we suggest that the general approach of Barfield et al. (2011) provides a useful tool for the analysis of evolution in spatially structured populations.

One advantage of clonality given spatially varying selection, reminiscent of the results seen after a sudden

change of environment in time (one of the scenarios explored in Orive et al. 2017), may be that this reproductive mode allows standing genetic variation to be rapidly utilized by selection for adaptation without dissolution by sexual reproduction and recombination. Indeed, this is one hypothesized explanation for experimental results showing that habitat heterogeneity favors asexual grass thrip lineages over sexual lineages (Lavanchy et al. 2016). We also note a recent study of ecological differentiation in *Boechera* showing that asexual reproduction was associated with greater fine-scale environmental heterogeneity (Ruthworth et al. 2018). Suppressed recombination at the wave front of a range expansion under an additive model of allelic effects has been shown to have beneficial effects, allowing the spread of lineages with high fitness (Peischl et al. 2015); reduced recombination due to asexual reproduction may work in concert with high heterozygosity to allow successful invasions (Peischl and Excoffier 2017). Examples of invasive spread with clonal dispersal can be found across a wide range of organisms, from aquatic plants such as water hyacinth (Zhang et al. 2010) to the clam genus *Corbicula* (Pigneur et al. 2014), and in the adaptation of fungal pathogens to their novel plant hosts (McDonald and Linde 2002), lending support to these ideas. Another advantage of clonality is the ability of parents to pass on part of the nonadditive genetic component of phenotype, in addition to the additive genetic component.

An important extension of this initial work will be to consider continuous selective changes across space, such as across smooth gradients. It is an open question whether adaptation across continuous spatial gradients reveals a different effect of partial clonality on the probability of adaptation than in the discrete coupled habitats examined here. Understanding adaptive differentiation across shorter spatial scales may be particularly important in considering local adaptation for marine foundation species that shape ecological function, such as salt marsh grasses, mangroves, seagrasses, and corals (Hays et al. 2021), where environmental conditions can change rapidly across short distances.

The effect of varying the fraction of clonal dispersers depends strongly on stochastic effects; here, the deterministic and stochastic models differ markedly. While both evolutionary stochasticity (e.g., mutation, drift, distributions of genotypes and phenotypes) and demographic stochasticity are at play in the IBM, demographic stochasticity is likely a particularly important factor differentiating the results of this model from the deterministic model (in combination with reduction in phenotypic variance of sink immigrants under increased clonal dispersal). For the deterministic model, we showed numerically that increased clonal movement (M_c) increases the maximum difference between source and sink optima permitting adaptation in deme 2. In this deterministic model, gene flow from the

source is the only factor preventing adaptation in the sink; if gene flow were completely cut off, both demes would adapt to their respective optima. Since we are assuming a Gaussian distribution of phenotypes (and use a continuous variable for population size), in a deterministic world there is always at least some part of the population with phenotypes in the tail of the distribution that would survive selection and allow adaptation.

In contrast, stochastic IBM simulations under asymmetric clonal and sexual dispersal showed that an increased clonal dispersal fraction decreased the probability of adaptation for a given difference in optimum phenotype, with a strong interaction between the clonal migration fraction and the association parameter ρ . In these stochastic simulations, adaptation in the sink deme initially requires the survival to reproduction of juveniles born in the source that immigrate to the sink. The parents of these juveniles have survived selection in the source and so are likely to have genotypes and phenotypes close to the source optimum. Both types of offspring inherit genotypes from their parents. Clonal offspring that immigrate from the source to the sink, for $\rho > 0$, also inherit part of the nonadditive genetic and random component of the phenotype (e) of their parents and therefore tend to have phenotypes closer to the source optimum than do sexual offspring, which have random values of e and thus greater phenotypic variability. Since survival after migration to the sink requires a phenotype close to the sink optimum, which can be very different from the source optimum, adaptation is increased with a greater fraction of sexual offspring. With a large difference between source and sink phenotypic optima and a high fraction of clonal offspring, in a given generation all clonal sink immigrants might fail to survive, and the clonal offspring of any of those immigrants that do manage to survive might in turn fail to survive, rendering the sink temporarily empty and forcing the process of adaptation to start over. This does not occur in the deterministic model because it does not include demographic stochasticity. The deterministic model predicts many qualitative features of the fully stochastic individual-based simulation, but this is one circumstance where its predictions break down. This is not because of genetic assumptions in the deterministic model, such as constant variances, but rather ecological assumptions, in particular the absence of demographic stochasticity and hence extinction risk.

In considering dispersal between two demes that each are initially populated and at their respective phenotypic optima but with different carrying capacities or sizes, an asymmetry in deme population size and equal per capita movement rates leads to more individuals leaving the high-carrying-capacity deme than returning. This results in relatively more matings in the low-carrying-capacity deme between residents and poorly adapted immigrants, which can

lead to population collapse in the smaller deme for some differences in deme phenotypic optimum (migrational meltdown; Ronce and Kirkpatrick 2001; see also Filin et al. 2008; Holt and Barfield 2010; Holt et al. 2011). Increasing clonality reduces this effect by decreasing the disruptive effect of gene flow into the smaller deme, decreasing the amount of mating between residents and immigrants. We have shown that indeed for both the deterministic model and the IBM stochastic simulations, increased clonality reduces the parameter space for migrational meltdown in terms of the range of differences in optimal phenotype for which the smaller population can be forced to become maladapted.

The results presented here, for both deterministic and individual-based stochastic models, demonstrate that an ecologically important reproductive mode, partial clonality, has important impacts on the expected spatial pattern of adaptation or maladaptation for phenotypic traits. We have shown that partial clonality expands the opportunity for spatial adaptation. Identifying key life history components, such as reproductive mode, that make populations especially vulnerable to changing environmental conditions, leading to maladaptation and even extinction, or that conversely allow populations to more easily adapt to new environments, is especially important in applied contexts, such as the study of invasive species, emerging infectious diseases, biological control, and the impacts of rapid environmental change. Our results may help explain the many examples where clonal or partially clonal populations or species have been found to be highly successful invaders of new habitats, complementing traditional explanations based on the avoidance of Allee effects. For example, recent theoretical work has considered how selfing within a large source population influences the ability of dispersers to establish in a new habitat (Sachdeva 2019). A high rate of (but not complete) selfing was found to be beneficial in a scenario where the establishing population experiences both inbreeding depression and maladaptation at a polygenic trait, even in the absence of mate limitation (the traditional explanation for selfing, i.e., reproductive assurance; Baker 1955). (Note that our IBM allows selfing.) Our results point to a similar benefit of partial clonality and underscore the importance of considering how this widespread reproductive mode impacts the scope for adaptation in heterogeneous environments.

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Statement of Authorship

All authors saw and approved for submission the final version of the manuscript, and all persons entitled to authorship have been named. M.E.O., M.B., and R.D.H. conceptualized the work and developed the model; M.E.O. and M.B. carried out model analysis with input from R.D.H.; M.B. wrote the code and carried out numerical analyses and simulations with input from M.E.O. and R.D.H.; M.E.O. wrote the original draft; all authors contributed to writing, review, and editing of the manuscript.

Data and Code Availability

The code for performing the numerical analyses and simulations is available on Zenodo (<https://doi.org/10.5281/zenodo.7698895>; Orive et al. 2023).

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