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LOCAL ADAPTATION WHEN COMPETITION DEPENDS ON PHENOTYPIC SIMILARITY

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Recent work incorporating demographic–genetic interactions indicates the importance of population size, gene flow, and selection in influencing local adaptation. This work typically assumes that density-dependent survival affects individuals equally, but individuals in natural population rarely compete equally. Among-individual differences in resource use generate stronger competition between more similar phenotypes (frequency-dependent competition) but it remains unclear how this additional form of selection changes the interactions between population size, gene flow, and local stabilizing selection. Here, we integrate migration–selection dynamics with frequency-dependent competition. We developed a coupled demographic-quantitative genetic model consisting of two patches connected by dispersal and subject to local stabilizing selection and competition. Our model shows that frequency-dependent competition slightly increases local adaptation, greatly increases genetic variance within patches, and reduces the amount that migration depresses population size, despite the increased genetic variance load. The effects of frequency-dependence depend on the strength of divergent selection, trait heritability, and when mortality occurs in the life cycle in relation to migration and reproduction. Essentially, frequency-dependent competition reduces the density-dependent interactions between migrants and residents, the extent to which depends on how different and common immigrants are compared to residents. Our results add new dynamics that illustrate how competition can alter the effects of gene flow and divergent selection on local adaptation and population carrying capacities.

KEY WORDS: **Dispersal, disruptive selection, ecological character displacement, frequency-dependent selection, migration–selection balance, niche partitioning, quantitative genetics.**

Persistent, spatially varying selection promotes the evolution of different morphologies, behaviors, or life-history characteristics in different populations (Kawecki and Ebert 2004). Gene flow between populations, however, tends to oppose the diversifying effects of local selection by "pulling" the population's mean phenotype away from the optimal for the environment that population experiences. Such gene flow can hinder local adaptation in heterogeneous environments and at the edges of species ranges (Kirkpatrick and Barton 1997; Lenormand 2002). There is a rich history of empirical (e.g., King and Lawson 1995; Hendry and Taylor 2004; Nosil and Crespi 2004; Fitzpatrick et al. 2008) and theoretical (e.g., Tufto 2000; Ronce and Kirkpatrick 2001; Lenormand 2002; Barton 2010) explorations into how traits evolve under the balance between gene flow and local selection. Recent work incorporating demographic–genetic interactions indicates the importance of population size in mediating the effects gene flow and selection on local adaptation (Holt 1987; Holt 1996; Garcia-Ramos and Kirkpatrick 1997;

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Holt and Gomulkiewicz 1997; Kirkpatrick and Barton 1997; Gomulkiewicz et al. 1999; Ronce and Kirkpatrick 2001; Tufto 2001; Kawecki and Holt 2002; Garant et al. 2007). Although crucial in bringing attention to the importance of demography, this work typically assumes that density-dependent survival affects individuals equally. However, a large literature, both theoretical (e.g., Taper and Case 1985; Doebeli 1996; Ackermann and Doebeli 2004) and empirical (e.g., Bolnick 2004; Bolnick et al. 2004), on individual specialization indicates that the strength of intraspecific competition can depend on phenotypic similarity. How such phenotype-dependent competition influences local adaptation and population carrying capacities when there is migration and selection remains largely unexplored (although see Wilson and Turelli 1986; Case and Taper 2000).

Population size affects the degree of competition for resources, and individuals in natural population rarely compete equally. Many species in nature exhibit substantial within-population variation in resource use (Bolnick et al. 2004). That is, individuals typically use only a subset of the resources available to the whole population. Such individual specialization generates greater intraspecific competition for limiting resources between individuals with similar phenotypes compared to individuals with different phenotypes, which is often called frequency-dependent competition (a form of diversifying selection: MacArthur and Levins 1967; Slatkin 1980; Taper and Case 1985; Doebeli 1996; Bolnick et al. 2003; Ackermann and Doebeli 2004). Individuals with rare resource-use phenotypes gain an advantage when the preferred resources of the most common phenotypes are depleted. Frequency-dependent competition could, therefore, influence the effect of migration and selection on local adaptation because it creates asymmetries in the strength of density-dependent interactions between migrants and residents adapted to different resource types.

Intuition suggests that if immigrants express different phenotypes compared to residents, then frequency-dependent competition should increase the survival of rare immigrants compared to common residents. Frequency-dependent competition also reduces competitive interactions between resident and immigrant genotypes. By reducing the influence that immigrants have on the amount of density-dependent mortality experienced by residents, frequency-dependent competition allows residents to experience less density-dependent mortality than if they compete equally with the whole population. By reducing the influence that residents have on the amount of density-dependent mortality by immigrants, frequency-dependent competition reduces the likelihood of maladapted immigrants being excluded from the new population via competition. Because frequency-dependent competition causes proportionately higher mortality in common phenotypes compared to rare phenotypes (Ackermann and Doebeli 2004; Bolnick 2004; Rueffler et al. 2006; File et al. 2011), local adaptation and patch carrying capacity will depend on how exactly stabilizing selection and migration alter the frequency and abundance of phenotypes in the population. Furthermore, stabilizing selection reduces phenotypic variation, whereas frequency-dependent competition increases variation and can maintain genetic polymorphisms (Bulmer 1974; Slatkin 1979; Bürger and Gimelfarb 2004).

Among-individual niche variation has been a central feature of empirical studies on niche evolution (reviewed by Bolnick et al. 2003) and has motivated a large literature on the role of resource competition in diversification and sympatric speciation (e.g., Taper and Case 1985; Dieckmann and Doebeli 1999; Schluter 2000; Ackermann and Doebeli 2004). Importantly, the species that provide the empirical basis to which the well-established theory of migration–selection balance is based on (e.g., Schluter and McPhail 1992; Bolnick et al. 2004) overlap with species that provide the empirical basis for the equally well-established theories of niche evolution and character displacement (Brown and Wilson 1956; Slatkin 1980; Abrams 1998; Taper and Case 2000). There is, therefore, substantial empirical support for a scenario in which migration between patches with different resource types can interact with divergent selection for different resource-use phenotypes among patches and frequency-dependent competition for resources within patches. An illustrative example is threespine sticklebacks (*Gasterosteus aculeatus*) in which individuals with similar gill raker lengths and gape widths compete more strongly for the same type of prey items (Bolnick 2004) and the composition of prey varies between benthic and limnetic habitats that are connected by dispersal (Schluter and McPhail 1992). Other empirical examples of traits conceivably connected to frequency-dependent competition and local adaptation are those related to habitat use, utilization of food types, and the timing of activities (Benkman 1996; Bolnick et al. 2004).

To better understand how frequency-dependent competition influences the effects of migration and selection on trait evolution and population size, we develop a model that couples demographic and genetic dynamics in two populations subject to gene flow and local stabilizing selection for different phenotypic optima. We explore when and how much frequency-dependent competition influences local adaptation as it depends on the relative strengths of migration and mortality from stabilizing selection and competition. We find that frequency-dependent competition changes both the demographic and genetic outcome of the antagonism generated by migration and divergent selection.

Methods

MODEL OVERVIEW

Our model integrates models that explore how population size influences trait evolution via the effects of selection and migration

(e.g., Holt and Gomulkiewicz 1997; Kirkpatrick and Barton 1997; Gomulkiewicz et al. 1999; Ronce and Kirkpatrick 2001; Tufto 2001; Kawecki and Holt 2002) or by frequency-dependent competition (e.g., Bulmer 1974; Taper and Case 1985; Wilson and Turelli 1986; Abrams 1998; Dieckmann and Doebeli 1999; Day 2000; Ackermann and Doebeli 2004; Bürger and Gimelfarb 2004). We model the joint dynamics of a quantitative trait and population size in two habitats connected by gene flow and subject to local stabilizing selection and competition. We assume resource type varies in space so that optima for traits related to resource use vary spatially (e.g., stickleback prey in lakes vs. streams). With frequency-dependent competition, the optimal resource in each patch gets depleted, increasing the advantage for nonoptimal phenotypes. All parameters are equivalent between habitats, except the optimal phenotype. To separate effects of frequency-dependent competition from other known ways population size influences the effects of migration and selection (e.g., Kawecki 1995; Holt and Gomulkiewicz 1997; Ronce and Kirkpatrick 2001; Tufto 2001), we compare situations with and without frequency-dependent competition whereas migration rate, the strength of selection, and per capita density-dependent mortality are equivalent between populations.

When frequency-dependent competition for limiting resources occurs, density regulation depends on the frequency of phenotypes in the population as well as overall population size. Frequency-dependent competition thus creates an additional form of (disruptive) selection, in addition to local stabilizing selection around a phenotypic optimum that differs between habitats. When frequency-independent competition occurs, density regulation is independent of phenotype and competition affects every individual similarly, so that density regulation simply follows the traditional per-capita treatment of density-dependence. We use the term "migration" to mean the proportion of individuals exchanged between populations. It is well known that species disperse and compete at different life-history stages and the order in which migration, mortality, and recombination occur in discrete time changes the effect that selection within patches has on both mean trait value and population size (e.g., de Jong 2005; Hendry and Day 2005; Saccheri and Hanski 2006; Thibert-Plante and Hendry 2011). To determine the effect of the order of life-history events, we investigated two alternative life cycles. Life cycle stage are denoted as M (migration), S (selection and competition), and R (reproduction), and we explore two orders: MSR and SMR. Census time is arbitrary, but occurs in our model after reproduction. The important differences between the MSR and SMR order are whether patch-specific mortality from selection and competition occurs following migration but before reproduction (MSR) or following reproduction but before migration (SMR).

MODEL DETAILS

In our model, g represents the genotypic, or breeding, value of an individual, which is the sum of the additive effects of its genes (Lynch and Walsh 1998). To couple the genetic and demographic dynamics, we follow the population density of genotypes g in patch i, denoted as $n_{i,t}(g)$. The total population size is then $N_{i,t} = \int n_{i,t}(g)\, dg$ (similar to Coulson et al. 2010 and Baskett and Waples 2013). The genotype probability density is given by $\psi_{i,t}(g) = n_{i,t}(g)/N_{i,t}$.

We present the model below according to the MSR order (Fig. 1). Migration is random with respect to both phenotype and genotype and symmetrical between patches. The population density after migration (Fig. 1), given the proportion (m) of each population that migrates to the other patch, is:

$$n'_{1,t}(g) = (1-m)n_{1,t}(g) + mn_{2,t}(g), \tag{1a}$$

$$n'_{2,t}(g) = (1-m)n_{2,t}(g) + mn_{1,t}(g). \tag{1b}$$

Individual fitness is determined by two components, based on a genotype's phenotype, which operate at the same time to influence survival: (1) stabilizing selection around at phenotypic optima, and (2) competition among individuals. The phenotype f that a given genotype expresses depends on V_E, which is the environmental variance indicating random environmental effects on the expressed phenotype independent of the genotype, according to:

$$P(f\,|g) = \frac{1}{\sqrt{2\pi V_E}} \exp\left(\frac{-(f-g)^2}{2V_E}\right). \tag{2}$$

Stabilizing selection acts on phenotypes and depends on how far an individual is from the phenotypic optimum θ_i in patch i as well as the variance of the selection surface V_s. The survival probability for a given phenotype is then:

$$S_i(f) = \exp\left(\frac{-(f-\theta_i)^2}{2V_s}\right). \tag{3}$$

The strength of stabilizing selection is therefore given by $s = 1/V_s$ (Fig. 1).

Competition is based on the Beverton–Holt function with parameter α describing the strength of the per capita reduction in survival with increasing density ($\alpha = R - 1/(RK)$ where R is the population growth rate at low density and K is the patch carrying capacity without migration and stabilizing selection). The type of competition for limiting resources leading to frequency-dependence is more likely to result from contest competition and less likely to result from over-compensatory dynamics (e.g., cannibalism). As a result, we did not explore other types of density regulation like that in the Ricker model. When there is

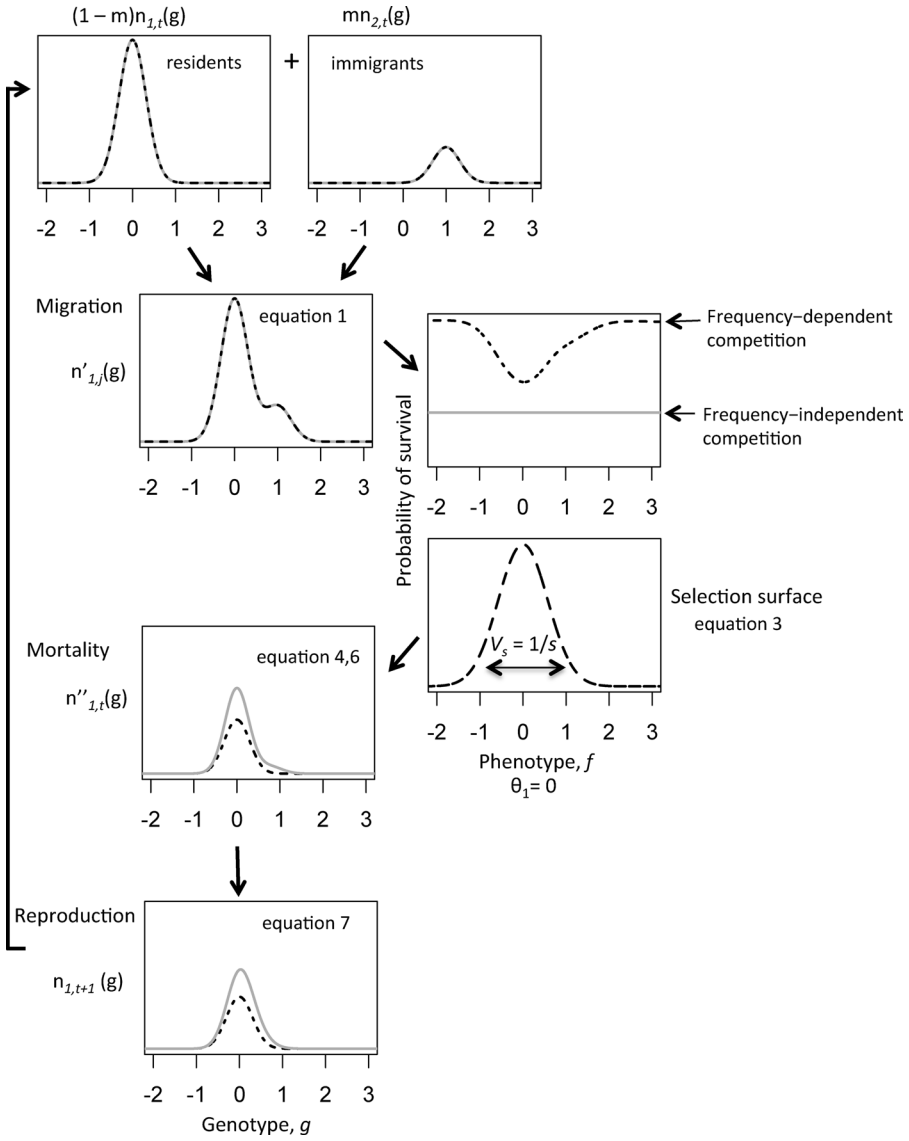

Figure 1. Schematic of the model presented for the migration, selection/competition, reproduction (MSR) order of life-history events for one generation in patch $i = 1$. Solid gray lines represent frequency-independent competition and the dashed black lines represent frequency-dependent competition. The model iterates through each stage until an equilibrium distribution is reached.

frequency-independent competition, the number of individuals after an episode of stabilizing selection and competition is then:

$$n''_{i,t}(g) = \frac{\int S_i(f)P(f|g)n'_{i,t}(g)\,df}{1+\alpha N'_{i,t}} = \frac{\sqrt{\frac{V_S}{V_S+V_E}}\exp\left(-\frac{(g-\theta_i)^2}{2(V_E+V_S)}\right)n'_{i,t}(g)}{1+\alpha N'_{i,t}}. \quad (4)$$

Competition between phenotypes f and f^* depends on their similarity $(f - f^*)$ and the width of interaction neighborhood V_U. Individuals with more similar phenotypes interact more strongly according to the "competition kernel"

$$U_i(f, f^*) = \exp\left(\frac{-(f-f^*)^2}{2V_U}\right). \quad (5)$$

Under this formulation, V_U can be interpreted as the variance (in the same units as V_s) of an individual's resource utilization function (Taper and Case 1985; Abrams 1998; Dieckmann and Doebeli 1999; Ackermann and Doebeli 2004; Bürger and Gimelfarb 2004). Small V_U means that individuals are specialized on a small fraction of the total available resource axis, which translates into stronger frequency-dependent competition. When there is frequency-dependent competition, the number of individuals after an episode of stabilizing selection and competition is

then:

$$n''_{i,t}(g) = \int \frac{S_i(f)P(f|g)n'_{i,t}(g)}{1+\alpha(V_U)\iint U_i\,(f,\,f^*)\,P\,(f^*|g^*)\,n'_{i,t}\,(g^*)\,dg^*df^*}\,df. \quad (6)$$

In the limit as $V_U \to \infty$, frequency-dependent competition vanishes because all individuals can use the full spectrum of the resource axis so all individuals compete equally and the competition kernel $U_i(f, f^*)$ tends to one, that is, equation (6) approaches equation (4). To properly compare frequency-dependent versus frequency-independent competition in terms of the effects on population size, we scaled α with V_U so that the frequency-independent and frequency-dependent competition resulted in the same equilibrium population sizes in the absence of migration and stabilizing selection. We then present the results for population size as being relative to the size the population would attain without migration. Relative population size therefore measures the extent to which migration depresses absolute population size. We assess the effects of frequency-dependent competition by comparing models that use either equation (4) or (6) (with the strength of the per-capita reduction in survival, α, scaled according to V_U). For the SMR order, equation (1) is swapped with equations (4) and (6) for the frequency-independent and frequency-dependent cases, respectively.

For reproduction, we base inheritance on the infinitesimal model, which is appropriate for modeling evolutionary changes in continuous traits, determined by many loci, over relatively short times scales (those relevant to changes in population size; Lande and Arnold 1983; Turelli and Barton 1994; Huisman and Tufto 2012). Therefore, the distribution of genotypic values g among offspring within a family follows a normal distribution with a mean of the mid-parent value and fixed variance of $V_{LE}/2$, where V_{LE} is the genetic variance at linkage equilibrium. Given random mating, such that the joint probability density of encounters with genotypes g_1 and g_2 is $\psi''_{i,t}(g_1)\psi''_{i,t}(g_2)$, the offspring probability density is the product of the joint probability and the offspring distribution of each mating pair integrated over all mating pairs (Slatkin 1970; Turelli and Barton 1994). Then, given the per-capita reproductive output (offspring per adult) R, the population density of genotypes after reproduction is:

$$n_{i,t+1}(g) = \frac{RN''_{i,t}}{\sqrt{\pi V_{LE}}} \iint \psi''_{i,t}(g_1)\,\psi''_{i,t}(g_2) \times \exp\left(-\frac{(g-(g_1+g_2)/2)^2}{V_{LE}}\right) dg_1 dg_2 \quad (7)$$

MODEL ANALYSIS

Our model is not analytically tractable, so we numerically iterate through the recursion equations (1), (4) or (6), and (7) (Fig. 1) until we reach equilibrium (the change in subsequent values of $n_i(g)$ at each grid point are sufficiently small, 10^{-6}; equilibrium values designated by $\overline{n}_i(g)$, $\overline{\psi}_i(g)$, and $\overline{N}_i$). Simulations follow a discretized distribution that numerically represents $n_{i,t}(g)$. We calculated integrals (except those in equation 7) using the Simpson's 3/8 rule. The convolution in equation (7) was evaluated using the fast Fourier transform method (Turelli and Barton 1994; Tufto 2001). Census at equilibrium is taken after equation (7). We follow the full breeding value distributions of the population rather than assume a Gaussian approximation with fixed genetic variance because frequency-dependent competition, strong selection, and migration between populations experiencing differential selection can all create strong departures from normality and constant variance (Fig. 1; Turelli and Barton 1994; Tufto 2000; Huisman and Tufto 2012).

To analyze both the genetic and demographic effects of competition and gene flow on local adaptation, we present: (1) the equilibrium mean genotype $\overline{g}_i = \int g\overline{\psi}_i(g)dg$; (2) the equilibrium population genetic variance $\overline{V}_A = \int (g-\overline{g}_i)^2\overline{\psi}_i(g)dg$, relative to V_{LE}, $\overline{V}^*_A = \overline{V}_A/V_{LE}$; and (3) the equilibrium population size $\overline{N}_i$ relative to the equilibrium population size in the habitat in the absence of migration $N_{i,m=0}$, $\overline{N}^*_i = \overline{N}_i/\overline{N}_{i,m=0}$. Populations are locally adapted on average when $\overline{g}_i = \theta_i$, but increasing genetic variance $\overline{V}_A$ around this mean introduces a "genetic load" that reduces population fitness (Lande and Shannon 1996). Our equilibrium relative population size $\overline{N}^*_i$ indicates the demographic consequences of maladaptation (cf. genetic load, e.g., Lande and Shannon 1996), but it should be noted that our demographic results are in terms of our standardization. Relative population size $\overline{N}^*_i$ can therefore be interpreted as the proportional change in the local realized carrying capacity due to gene flow at a given strength of stabilizing selection. In the main text, we explore the effect of frequency-dependent competition on $\overline{g}_i$, $\overline{V}^*_A$, and $\overline{N}^*_i$ as it depends on the strength of stabilizing selection s, frequency-dependent competition V_U, and the sequence of life cycles. Following Tufto (2000), we keep $\theta_1 = 0$ and $\theta_2 = 1$ so that all variances, V_E, V_s, V_U, and V_{LE} are expressed in standardized units of the squared differences between optimal phenotypes (Supporting Information). For example, a V_{LE} of 0.1 corresponds to a situation where phenotypic optima differ by $1/\sqrt{0.1} = 3.16$ genetic standard deviations. The relative importance of local stabilizing selection V_s and frequency-dependent competition V_U can be assessed with the quantity $\tau = V_s/V_U$, where τ greater than 1 indicates stronger frequency dependence. Heritability is calculated as $h^2 = V_A/(V_A + V_E)$, where V_A was initialized as V_{LE}, but changes over time. In the online supplement, we present the sensitivity of $\overline{g}_i$, $\overline{V}^*_A$, and $\overline{N}^*_i$ to m, V_{LE}, and V_E, and to initial values. Asymmetrical initial values of genotypic mean $g_{i,0}$ and population size $N_{i,0}$ between patches did not alter the equilibrium values (Supporting Information; in the default case we initialized the genetic distribution with mean θ_i and variance V_{LE}). All code was written in R 2.15.1 (R Core Development

Team 2012) and is available from the Dryad doi:10.5061/dryad.7j564.

Results

With frequency-independent competition, the mean genotypic value of each population becomes closer to the patch-specific optima, indicating local adaptation, with increasing strength of stabilizing selection (gray lines in Fig. 2A, B). Population genetic variance increases or decreases depending on whether mortality from selection and competition occurs following migration but before reproduction (MSR) or following reproduction but before migration (SMR; gray lines in Fig. 2C, D). Frequency-dependent competition has a small effect of increasing local adaptation (black lines in Fig. 2A, B), but a large effect of increasing the genetic variance, especially for weaker stabilizing selection (black lines in Fig. 2C, D). In general, migration increases the amount that a population mean genotype and optimum differ, which decreases local adaptation and population fitness, and genetic variance decreases population fitness via stabilizing selection, creating a genetic load (e.g., Lande and Shannon 1996). In our model, despite the increased genetic load, frequency-dependent competition reduces the amount that migration depresses population size (Fig. 2E, F; although not for all parameter values, see below and Supporting Information). Essentially, more immigrants make it through selection and competition (which increases population genetic variance), but more residents do too, which increases relative population size $\overline{N}_i^*$.

Frequency-dependent competition causes the effects described earlier even when the strength of stabilizing selection is stronger than the strength of competitive interactions between similar phenotypes (i.e., $\tau < 1$, Fig. 2; when the fitness surface describing selection, eq. 3, is narrower than the competition kernel, eq. 5). Frequency-dependent competition causes the greatest departure from the frequency-independent case when frequency-dependence is stronger than selection ($\tau > 1$; Fig. 2). Increasing stabilizing selection decreases the sensitivity of population genetic variance to changes in the strength of frequency-dependent competition (regardless of τ).

Changing the migration rate m had more effect on the overall equilibrium outcomes than it did on the relative differences between the frequency-dependent versus frequency-independent case (Fig. B1, B2). Frequency-dependent competition can in fact slightly decrease local adaptation when the within family genetic variance V_{LE} is high, which is equivalent to a large difference between patch phenotypic optima (Supporting Information), and when selection is strong and mortality occurs after migration and before reproduction (MSR; Fig. S3). However, population size does not decline as much because the genetic load is reduced by the concomitant decrease in genetic variance. Frequency-dependent competition has little effect when trait heritability is low (i.e., when environmental effects V_{E} are large; Fig. S5, S6). Note that populations can go extinct when the population mean genotype and optimum differ a lot and the population genetic variance is large (i.e., which occurs with high V_{E}, or V_{LE}, Supporting Information; essentially the genetic load depresses populations below replacement). Frequency-dependent competition however, does not increase or decrease the likelihood of population extinction in our model (Supporting Information).

The transient dynamics of population mean genotype, population genetic variance, and population size were altered by the strength of frequency-dependent competition and asymmetrical initial conditions (Fig. 3, Supporting Information). Example transient dynamics for relatively weak stabilizing selection when mortality occurs after migration are shown in Figure 3. In particular, where population size in one patch was initially 10% of the other patch (asymmetric $N_{i,0}$), the population genetic variance fluctuates more when frequency-dependent competition is strong ($V_{\mathrm{U}} = 0.01$) compared to when it is weak ($V_{\mathrm{U}} = 1$; Fig. 3). The SMR order and the MSR order with strong stabilizing selection are shown in the online supplement for completeness.

Discussion

Our results indicate that frequency-dependent competition, which arises when individuals with similar phenotypes compete more strongly for limiting resources compared to individuals with different phenotypes, can alter the genetic and demographic effects of local adaptation in species occupying variable environments. Because of reduced density-dependent interactions between migrants and residents, frequency-dependent competition increases divergence in mean genotypic values between patches, increases genetic variance within patches, and decreases the negative effects of gene flow on population size. The quantitative effects on the equilibrium population mean genotype were, however, quite small, whereas the quantitative effects on both equilibrium and transient genetic variance as well as equilibrium population size were more significant. We find effects of competition based on phenotypic similarity given equivalency in all parameters between habitats (e.g., per capita density dependence, migration rate, reproductive output) except optimal traits values. Previous studies on how population size influences local adaptation in spatially variable environments find that size is typically important in cases of asymmetric immigration (Kawecki 1995; Holt 1996; Garcia-Ramos and Kirkpatrick 1997; Gomulkiewicz et al. 1999; Ronce and Kirkpatrick 2001; Tufto 2001; Kawecki and Holt 2002). Our model with symmetrical migration incorporates variation in individual-level resource use that can lead to an additional form of selection, the relative importance of which increases with increasing population size. Therefore, the frequency-dependent

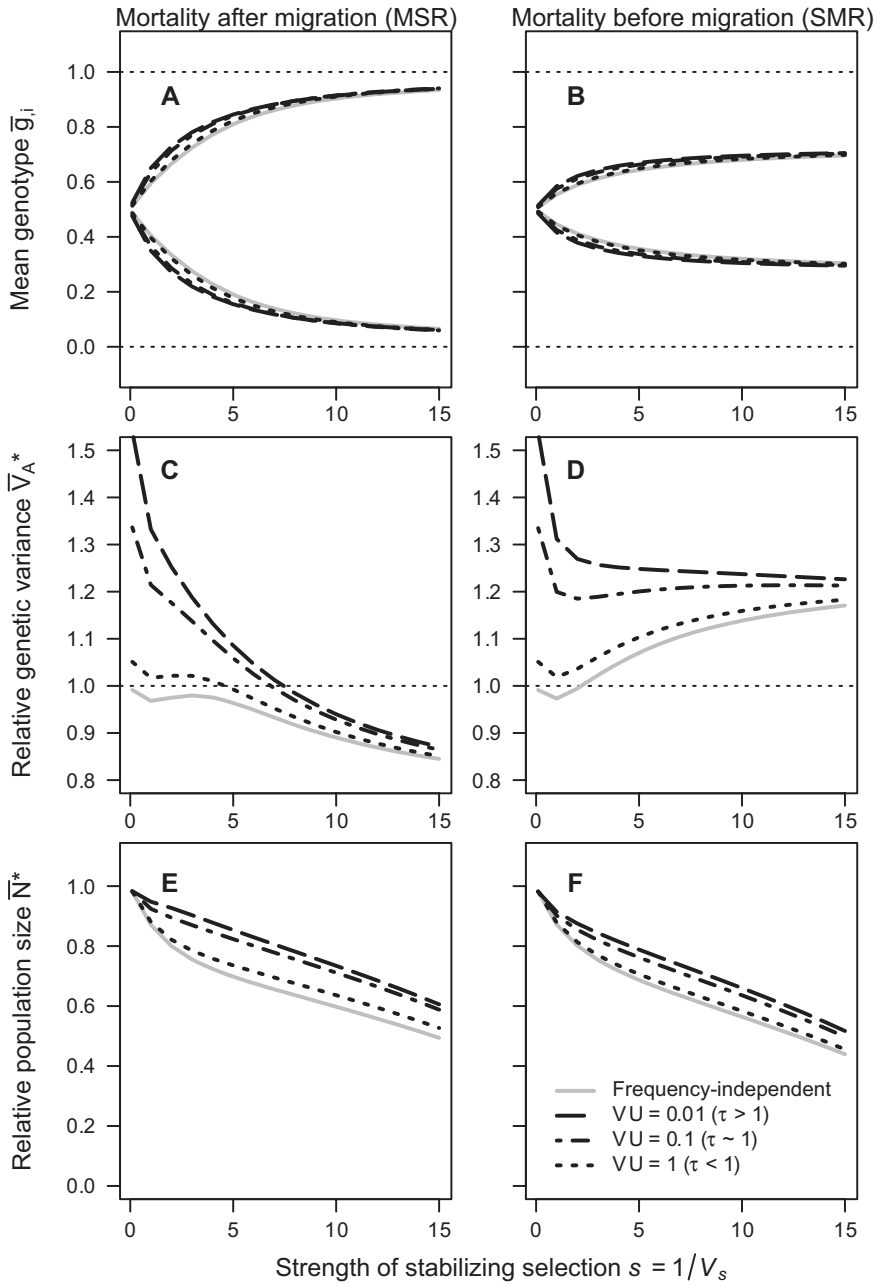

Figure 2. The relationship between the strength of stabilizing selection (in standardized units of s, see Supporting Information) and the equilibrium values of mean genotype $\bar{g}_i$ in both patches (A and B; dotted horizontal lines indicated patch-specific phenotypic optima), population genetic variance within patches relative to V_{LE} (C and D; dotted horizontal line indicates $\bar{V}_A^* = V_{LE}$), and relative population size within each patch (E and F). The latter, $\bar{N}_i^*$, is the proportion reduction in equilibrium population size due to gene flow. Solid gray lines represent frequency independent competition (eq. 4). The dashed black lines represent frequency-dependent competition with varying degrees of strength (eq. 6). Note that τ goes from >1 to <1 around $s = 10$ when $V_U = 0.1$. The order of life-history events (left or right column) is given by the order of M = migration; S = selection and competition; or R = reproduction. Parameter values used in this plot are: $\theta_1 = 0$; $\theta_2 = 1$, $m = 0.2$, $V_{LE} = 0.1$, $V_E = 0.1$. When $V_U = 0.01$, $\alpha = 0.00092$ ($R = 3$, $K = 723$); $V_U = 0.1$, $\alpha = 0.00030$ ($R = 3$, $K = 2233$); $V_U = 1$, $\alpha = 0.00015$ ($R = 3$, $K = 4362$). In the frequency-independent case ($V_U \to \infty$), $\alpha = 0.00013$ ($R = 3$, $K = 5000$).

competition we investigate here also influences local adaptation and the demographic consequences of maladaptation (Hairston et al. 2005; Kokko and Lopez-Sepulcre 2007; Coulson et al. 2010).

Our model is based on a quantitative trait that influences both frequency-dependent competitive ability and local fitness within a patch. Selection can act directly on such a trait when the optimal resource utilization differs among patches that have different

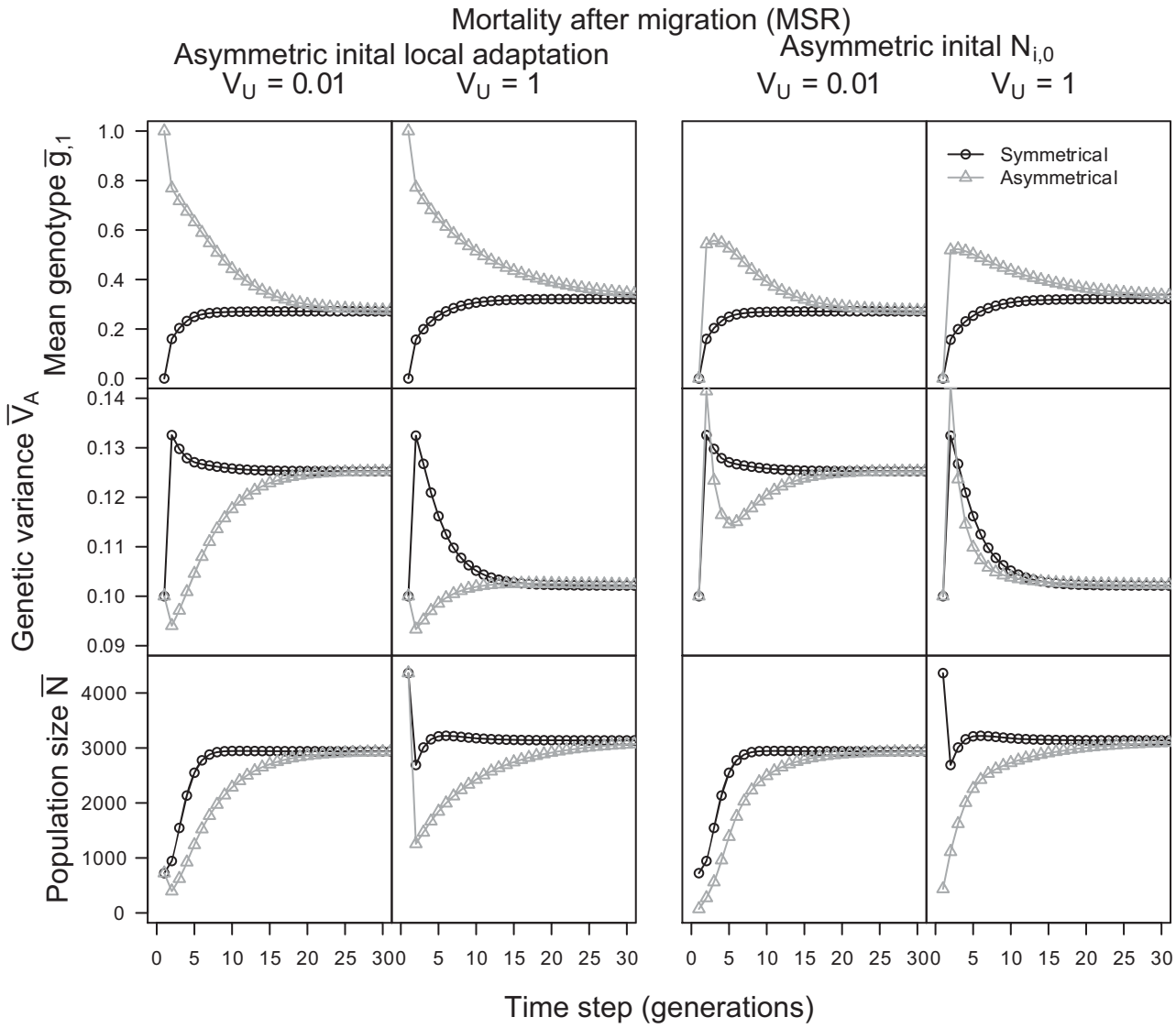

Figure 3. **Transient dynamics in patch 1 of the population mean genotypic value, population genetic variance, and population size under different strengths of frequency-dependent competition (V_U) with symmetrical (black lines; circles) or asymmetrical (gray lines; triangles) initial conditions for mean local adaptation (left panels) or initial population size (right panels) between the two patches. Initial mean genotypes reflected locally adapted populations (initial $\overline{g}_1 = \theta_1 = 0$; initial $\overline{g}_2 = \theta_2 = 1$) in the symmetrical case. In the asymmetrical case, population 1 was initially maladapted (initial $\overline{g}_1 = 1$, $\theta_1 = 0$) whereas only population 2 was locally adapted. Asymmetrical initial population sizes were created by setting sizes in patch 1 to 10% of that in patch 2, reflecting a potential gene "swamping" scenario. Default parameters used in all plots are $m = 0.2$, $V_E = 0.1$, $V_{LE} = 0.1$, $\theta_1 = 0$, $\theta_2 = 1$.**

types of resources (Bolnick et al. 2003; Rueffler et al. 2006; File et al. 2011). For example, cichlid fish with similar jaw morphology compete more strongly for the same type of food associated with a particular microhabitat, and the relative proportion of microhabitats varies among pools so different jaw morphologies are favored (Swanson et al. 2003). A trait influencing local fitness need not be the same trait that influences frequency-dependent competitive ability. If there are phenotypic correlations between one trait influencing competitive ability and another trait influencing local fitness, spatially varying selection can act directly on the trait influencing local fitness and indirectly on the trait influencing competitive ability. Genetic correlations between the two traits will then result in a correlated response to selection (Lande and Arnold 1983). Tufto (2010) found that allowing joint evolutionary changes in such additional traits reduces migration load, but the effect was small, suggesting that the simpler one-trait model that we used is at worst a slight underestimation of how much frequency-dependent competition influences adaptation.

Other studies have explored how frequency-dependent competition and gene flow across environmental gradients influence

species range limits and character displacement (Case and Taper 2000; Goldberg and Lande 2006; Heinz et al. 2009). Frequency-dependent competition in previous models typically occurs between two species, so individuals that compete for a resource do not interbreed. For example, Case and Taper (2000) showed that interspecific competition between two species decreases local adaptation, ultimately generating a range limit where the species distributions overlap, because the lowered population sizes resulting from competition increase the negative effects of gene flow from central to marginal areas within each species range. Frequency-dependent competition in our model occurs between individuals of the same species that have different phenotypes. Individuals that compete for a resource later interbreed. Extending the Case and Taper (2000) model, Goldberg and Lande (2006) showed that hybridization (where hybrids are inviable) between two species results in a narrower geographic area over which the two species co-occur because interbreeding with phenotypically dissimilar species lowers population size more so than when the species just compete. In our model, we expect assortative mating to modify the extent to which phenotypically different competitors interbreed and may further reduce the extent to which gene flow reduces population size.

As with models of ecological character displacement and niche width (e.g., Taper and Case 1985), we assumed that the "competitive neighborhood" of interactions on the phenotypic scale was symmetrical and that there was no adaptive phenotypic plasticity. For some traits that influence competitive ability however, frequency-dependent resource competition is likely to be asymmetrical. For example, given two plants of different sizes (or growth rates) competing for sunlight, the larger plant might affect the smaller plant more than vice versa. Furthermore, the shape of the resource carrying capacity distribution relative to the resource utilization distribution can generate asymmetrical interactions between different phenotypes (see Abrams 1998; Ackermann and Doebeli 2004). Other functional forms of frequency-dependent competition would be needed to explore the interactions between gene flow and selection when frequency-dependent competition is asymmetrical or the neighborhood width depends on the phenotype (see Case and Taper 2000). We expect asymmetrical frequency-dependent competition to generate asymmetrical patterns of adaptation between habitats, even when habitats have similar per capita density dependence and migration rates. Phenotypic plasticity could also alter the interactions between gene flow, selection, and frequency-dependent competition. An especially valuable next step would therefore be to empirically and theoretically explore the strength and functional form of selection at different population densities in different environments (Wade and Kalisz 1990; Bolnick 2004; Rueffler et al. 2006; File et al. 2011), while allowing for plasticity, to better understand how frequency-dependent competition can influence adaptation and spatial population structure.

In addition to informing a basic understanding of spatial population dynamics, our results can be applied to conservation and management questions related to the genetic and demographic effects of artificial propagation programs, which are common tools in a wide variety of fishery, forestry, agriculture, and wildlife management programs (Laikre et al. 2010). In this case, the populations experiencing gene flow and different selective regimes are the natural, wild populations, and the artificially cultured populations (Lynch and O'Hely 2001; Ford 2002; Tufto 2010; Baskett and Waples 2013). The potential for frequency-dependent competition to affect adaptation and population size shown here suggests that it could also influence both the positive and negative effects of artificial propagation programs on wild populations. In particular, our results suggest that selection in captivity, whether purposeful or accidental, on traits that influence competitive ability in natural environments has the potential to reduce the demographic consequences of negative genetic effects from artificial propagation programs on wild populations. Our results also suggest that maintaining individual-level difference in resource use and competitive ability in programs focused on production could increase population sizes and help to buffer productivity from the negative genetic effects of gene flow.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Section 1. Nondimensionalization of the strength of stabilizing selection.