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PREDICTING ADAPTATION UNDER MIGRATION LOAD: THE ROLE OF GENETIC SKEW

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Quantitative genetics models have been used to predict the constraints on local adaptation caused by gene flow between populations under migration–selection balance. One key assumption of this approach is that genetic values within a population are normally distributed. Gene flow, however, may generate distributions that are skewed toward the immigrant's mean value. If the response to selection from a skewed distribution is different from that expected under the assumption of normality, models may result in inaccurate predictions. We use individual-based computer simulations to explore this problem, comparing our results to a recent model developed by Hendry et al. (2001). We show that this model underestimates the equilibrium divergence between populations at migration–selection balance. The extent of this underestimation is correlated with the amount of genetic skew generated by migration and is partly explained by the fact that the analytical model ignores direct selection against hybrid phenotypes. We also show that all else being equal, response to selection in a population with a skewed distribution of genotypes is greater than in a population with normally distributed genotypes. The production of skew under migration–selection balance, however, is itself dependent upon the genetic architecture, with greater deviations from normality produced when alleles contributing to population differentiation have very different effect sizes. We find that both the skew and discrepancies between the models are greatest at intermediate migration rates and moderate to strong selection, which is exactly the region of parameter space that is most empirically relevant.

KEY WORDS: Continuum-of-allele model, diallelic model, divergent selection, Gaussian approximation, genetic architecture, quantitative trait.

Populations inhabiting heterogeneous environments tend to adapt to their local conditions, provided levels of gene flow are low enough for selection to cause allele frequencies to diverge. Because gene flow generally decreases with geographic distance, it is unusual to see local adaptation emerging at very fine spatial scales. If natural selection is sufficiently strong, some differentiation may be possible, even under very high rates of gene flow. The classic studies of adaptation to contaminated soils in mine tailings by McNeilly (1968) and Antonovics and Bradshaw (1970) provide a striking example of divergent selection causing adaptive responses over spatial scales small enough to allow considerable gene flow between environments. Although many studies have shown evidence of some level of local adaptation (Hedrick et al. 1976; Linhart and Grant 1996), it is unclear whether gene flow commonly constrains the amount of divergence between populations.

Unfortunately, implicating gene flow as the primary causal factor limiting local adaptation is difficult. Although several studies have found positive correlations between genetic distance and phenotypic divergence between pairs of populations inhabiting different environments (e.g., Smith et al. 1997; Lu and Bernatchez 1999; Hendry and Taylor 2004; Nosil and Crespi 2004), this alone does not prove that gene flow constrains adaptation because a similar pattern would be expected if divergence times differed between the population pairs. Furthermore, an opposite causal explanation is also possible, as reproductive isolation mechanisms that evolve as a byproduct of adaptation may constrain gene flow, resulting in a similar correlation (Räsänen and

Hendry 2008). Although reciprocal transplant experiments can help by demonstrating fitness differences between populations (or lack thereof), they do not rule out the possibility that any observed lack of divergence is simply the result of a nonequilibrium state as populations evolve toward their local optima following some perturbation (e.g., colonization or shift in environment). Other studies have used ecological factors that should be insensitive to demographic history, such as differences in patch size or connectivity to infer relative amounts of gene flow and test correlations between gene flow and trait divergence among populations (Dhondt et al. 1990; Riechert 1993; Sandoval 1994; Storfer et al. 1999). Combining these coarse estimates of contemporary gene flow with evidence from molecular divergence would strengthen the inferences drawn from these correlation-based studies. Even so, it is unclear whether a linear relationship between gene flow and trait differentiation should always be expected, especially in light of the nonlinear behavior seen in related population genetic models (e.g., Hendry et al. 2001; Spichtig and Kawecki 2004).

Another approach that can be used to test hypotheses about the role of gene flow is to compare empirical observations to predictions based on models that explicitly represent the evolutionary processes involved. Where variation in the trait of interest is driven by a single locus, population genetic models can be applied to predict allele frequencies under migration–selection balance (King and Lawson 1995; Lenormand et al. 1998; Bolnick and Nosil 2007). King and Lawson (1995) also derived a simple model of migration–selection balance in a quantitative trait where divergence did not depend upon the additive genetic variance. Hendry et al. (2001) corrected this model, providing a more general solution for predicting divergence between populations, which has been applied in several empirical studies (Hendry et al. 2002; Saint-Laurent et al. 2003; Moore et al. 2007). A similar model was derived by Lopez and colleagues (2008), who explored the effect of seed versus pollen migration on genetic load and divergence between populations. Both Hendry et al. (2001) and Lopez et al. (2008) suggested that deviations from a Gaussian distribution of breeding values might compromise the accuracy of this approach by violating the assumption of normality, but as yet, there has been no systematic exploration of this problem. Lopez et al. (2008) used simulations to test their analytical model and concluded that only the most heterogeneous environments resulted in inaccurate predictions. Although they suggested that any differences between simulations and analytical predictions were caused by deviations from a Gaussian distribution of breeding values, they did not explore this explanation in detail. Furthermore, they limited their study to very high mutation rates, which might be expected to counteract asymmetries caused by migration and increase the accuracy of the analytical model across a wider range of migration rates and strengths of selection.

Here, we revisit this problem, comparing the predictions of the Gaussian approximation model (hereafter GAM) developed by Hendry et al. (2001) to predictions from an individual-based simulation model. We show that the magnitude of the discrepancy between model predictions correlates with the amount of skew in the distribution of the breeding values, suggesting that departures from normality have important consequences for predicting evolutionary trajectories. We also explore the causal basis for these differences, showing that the response to selection within a single generation is greater from a skewed distribution of genotypes than from a normal distribution with the same mean and variance. High mutation rates can reduce the discrepancies between the GAM predictions and simulations, reproducing the patterns observed by Lopez et al. (2008). Observed discrepancies between predictions of the GAM and those of the simulations tend to be most extreme at intermediate-to-high migration rates ($10^{-4} < m < 10^{-1}$) and medium-to-strong selection, which is exactly the region of parameter space that is biologically most interesting as this is the region where adaptive constraint is predicted by the GAM.

THE GAUSSIAN APPROXIMATION MODEL

Using quantitative genetics theory, the GAM solves for the equilibrium divergence in phenotype between two populations by finding when the change in mean trait value caused by the mixture of the immigrants and locals (Δz^m) is counterbalanced by the change caused by response to selection (Δz^s; Hendry et al. 2001). When migration is symmetrical and precedes selection, this yields the equilibrium divergence

$$D^* = D_\theta \left[\frac{V_G}{V_G(1-m) + (\omega^2 + V_P)m} \right], \qquad (1)$$

where D_θ is the difference between the optimal phenotype in each patch, m is the migration rate, V_P and V_G are the phenotypic and additive genetic variance, and ω^2 is the width of the fitness function (eq. 7 in Hendry et al. 2001). This model parallels Via and Lande's (1985) model for the evolution of phenotypic plasticity in heterogeneous environments. The basic assumption of this type of model is that the system can be accurately approximated by modeling the effect of migration on the mean population phenotype and ignoring its effects on the shape of the phenotypic distribution. Mixing of immigrants and locals changes the mean trait value (Δz^m), and the response to selection is calculated from this new average value under the assumption of constant and normally distributed genetic values (Fig. 1).

Migration followed by introgression, however, modifies the phenotypic composition of a population by adding distinct classes of individuals (e.g., immigrants, hybrids, backcrosses, etc., see Fig. 1), which results in a distribution of genetic values that is skewed toward the immigrant's trait value. Each of these classes

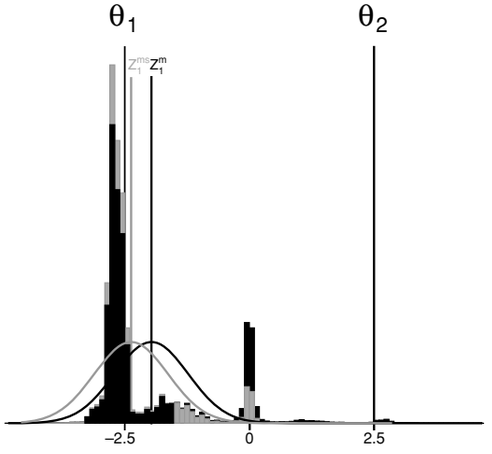

Figure 1. Histogram showing genotype frequencies in patch 1 at equilibrium following migration (black) and then selection (gray), for $\omega^2 = 2.5$ and $\theta = \pm 2.5$. Curves represent the Gaussian approximations used in the GAM, where immigration does not affect the variance, but changes the mean resident genotype by Δz^m, followed by a response to selection (Δz^s) toward local optimum θ_1, yielding z^{ms}. Equilibrium divergence is predicted when $\Delta z^m = -\Delta z^s$.

of individuals will then respond to selection differently, and by aggregating all of them into a single Gaussian distribution with a single mean, the GAM is likely to misrepresent the actual response to selection under certain circumstances. In particular, it ignores that selection is stronger on hybrids and immigrants, which may make up a large proportion of the total distribution prior to selection but be of little importance after selection, especially if selection is strong. To correct for this problem, we now present a model that partitions the total distribution of trait values into discrete phenotypic classes.

THE DISCRETE GAM MODEL

The model presented here follows closely the developments presented in Hendry et al. (2001). Our approach decomposes the total distribution of trait values in a population into three main phenotypic classes: the residents, the immigrants, and their hybrids, and then expresses the response to selection of the whole population as the sum of the responses of each phenotypic class weighted by their relative contributions based on the migration rate and relative fitness. The distribution of trait values within each class is still considered a Gaussian distribution and is represented in the model by its trait mean ($\bar{z}_{ik}$) and genetic variance V_{Gk}. For simplicity, each class k has the same genetic variance V_G in all populations. The average trait value in population i after mixing and random mating can thus be written as

$$\bar{z}_i^m = \sum_{k=1}^{3} p_{ik}\bar{z}_{ik}, \tag{2}$$

with

$$p_{ik}\begin{cases}(1-m)^2 & \text{for } k=1 \text{ (residents)},\\ 2m(1-m) & \text{for } k=2 \text{ (hybrids)},\\ m^2 & \text{for } k=3 \text{ (migrants)}.\end{cases} \tag{3}$$

We consider here only two populations, i and j, connected by migration at rate m. The average trait value for the different classes in population i is $(\bar{z}_i + \bar{z}_j)/2$ for the hybrid, $\bar{z}_i$ for the residents, and $\bar{z}_j$ for the immigrants. The mean trait value in population j is, by symmetry: $\bar{z}_j = \sum_{k=1}^{3} p_{jk}\bar{z}_{jk}$. The response to selection of class k in population i is then given by: $\bar{z}_{ik}^s = \bar{z}_{ik} + V_G\beta_i(\bar{z}_{ik})$, with $\beta(\bar{z}_{ik}) = (\theta_i - \bar{z}_{ik})/(\omega^2 + V_P)$ the selection gradient, and θ_i the trait optimum (substitute j for i for population j). The mean trait value after selection is then given by the sum of the contributions of each phenotypic class weighted by their relative fitness and frequency

$$\bar{z}_i^s = \frac{1}{\bar{W}_i}\sum_{k=1}^{3} p_{ik}\bar{w}_{ik}(\bar{z}_{ik} + V_G\beta_i(\bar{z}_{ik})), \tag{4}$$

with $\bar{w}_{ik} = \sqrt{\frac{\omega^2}{\omega^2+V_P}}\exp[-(\bar{z}_{ik}-\theta_i)^2/(\omega^2+V_P)]$, the mean fitness of phenotypic class k (following Via and Lande 1985), and $\bar{W}_i = \sum_{k=1}^{3} p_{ik}\bar{w}_{ik}$, the average fitness in population i.

We then proceed as Hendry et al. (2001) by finding the per generation changes in mean trait values in each population ($\Delta\bar{z} = \bar{z}^s - \bar{z}$) and in the divergence between those populations: $\Delta D = \Delta\bar{z}_j - \Delta\bar{z}_i$. The equilibrium phenotypic divergence between population i and j at migration–selection balance is then found by setting $\Delta D = 0$ and solving for the divergence $D = \bar{z}_j - \bar{z}_i$. However, as we consider a perfectly symmetrical system, for simplicity, we can substitute $-\bar{z}_i$ for $\bar{z}_j$ and $-\theta_i$ for θ_j overall, and solve $\Delta D = 0$ for $\bar{z}_i^*$, the equilibrium trait value in population i. The complete expression for ΔD at equilibrium is included in the Supplementary information. A first linear approximation of $\Delta D = 0$ can be found by using the assumption that both m and z_i are of order ε (small deviation) and finding the Taylor series expansion of order ε around $m = 0$ and $z_i = \theta_i$ yields:

$$\bar{z}_i^* = \theta_i - \frac{2mV_S}{V_G}\cdot\theta_i e^{-\frac{\theta_i^2}{2(V_S+V_G)}} \tag{5}$$

with $V_S = \omega^2 + V_E$, the strength of stabilizing selection. Equation 5 simply shows that, first, the effect of migration on the equilibrium trait value is strongest when the response to selection after mixing is likely to be smallest (larger V_S means weaker selection), and second, that this response to selection depends on the phenotypic divergence between the two populations (θ); it first increases as θ increases from 0 to the critical value of $\theta = \sqrt{V_S + V_G}$ and then decreases. This dependence of the equilibrium mean trait value on the phenotypic optimum divergence between the two

populations captures the fact that as the two populations diverge, selection against the hybrid and immigrant individuals increases, which compensates for their increased phenotypic effect on the population equilibrium value. The original GAM does not take into account this balanced effect of divergence and increased selection against intermediate phenotypes and thus predicts that the amount of constraint is independent of the total divergence between habitats (see Results). Because of the assumption that m is of the order of ε around 0, equation 5 gives accurate predictions for small migration rates only and numerical solutions of $\Delta D = 0$ were used to compare predictions from this model with simulation results. The complete expression for $\Delta D = 0$ is given in the Supplementary information along with a more accurate solution ignoring the simplification that m is small.

THE SIMULATION MODEL

To test the predictions of the GAM and discrete GAM, we ran individual-based simulations using a modification of the Nemo platform (Guillaume and Rougemont 2006). Individuals inhabited an environment consisting of two patches with different local optima ($\pm\theta$) that were connected by migration at some specified rate (m). Offspring were created by drawing parental gametes from either patch ($Pr[\text{local}] = 1 - m$; $Pr[\text{nonlocal}] = m$), with recombination occurring within the parents prior to migration at rate r between adjacent loci ($n = 50$ loci). Newly created offspring with phenotype z experienced stabilizing viability selection around the local optimum with survival probability equal to their fitness (W) as defined by a Gaussian function with variance ω^2: $W = \exp[-(\theta - z)^2/2\omega^2]$. When some nonzero level of environmental variance (V_E) was included, a random value drawn from a Gaussian distribution with a mean of zero and variance equal to V_E was added to the individual's genotypic trait value to calculate its fitness. Offspring were created until filling the local patch to carrying capacity (N), such that the entire system effectively experienced soft selection following migration of the parental gametes. Individuals had diploid genotypes with n loci arranged on a single chromosome, with each locus contributing additively to the phenotype. At the initialization of the simulations, all loci were given allelic values of 0 with a 5% chance of mutating to provide some initial variability. All mutations were drawn from a Gaussian distribution with a standard deviation of α, with their values added to the existing allelic value at the locus (continuum-of-alleles model). Following initialization, mutations occurred at a per-locus rate of μ, such that mutational variance was $V_M = 2n\mu\alpha^2$. Simulations were run until there were no consistent directional changes in mean, variance, or skew for at least 20,000 generations (this typically required at least 100,000 generations, depending on the parameter values). We ran 20 replicates of each parameter set, taking all statistical measurements of the populations after selection on juveniles every 50 generations for at least 400 intervals during the stable period at the end of the simulation. Genetic skew was calculated as: $\sum[(z - \bar{z})^3/\sigma^3]/N$, where σ is the standard deviation of the genotypes and $\bar{z}$ is the mean genotype. To check that the simulations were yielding accurate results, we compared genetic variance maintained at equilibrium under a homogenous environment to the variation predicted by Bürger et al. (1989) for the stochastic house-of-cards model of mutation–selection balance, and we found a good agreement (see Fig. S1 Supporting Information for details). As currently formulated, these simulations represent only the classical effects of migration, selection, mutation, recombination, and drift and do not include any effects of gene flow that have the potential to facilitate adaptation (e.g., demographic rescue or reinforcement).

To compare simulation results with the predictions of the GAM (eq. 1), we used the parameter values from each set of simulations to parameterize the GAM. Because the GAM also requires V_G and V_P as parameters (but these are dynamic products of the simulations), we used the within-patch genetic variance from the simulation results for V_G, and calculated $V_P = V_G + V_E$, as V_P was not measured directly from the simulations. We also note that $\hat{m}$ from the GAM is equal to $2m$ in the simulations and that the simulations measure V_G following selection. We used the migration rate parameter from the simulations to parameterize the GAM, as this is congruent with the theoretical development of the analytical model. Empirical studies, however, typically use estimates of m based on the realized gene flow inferred from divergence at molecular markers (e.g., Moore et al. 2007), which represents successful migration events and will be lower than the overall migration rate, therefore predicting less constraint.

We sought to examine model behavior over a biologically reasonable range of parameter values for selection, migration, and mutation rate. We used two differences in optimum ($\theta = \pm 1$; ± 2.50) and three strengths of stabilizing selection ($\omega^2 = 2.5$, 5, 25), yielding the migrant and hybrid fitness shown in Table 1. Nosil et al. (2005) reviewed a range of estimates for the fitness of locally adapted individuals when transplanted into a different environment, finding estimates for migrant fitness ranging from 0.087 to 0.99, which roughly corresponds to the range shown

Table 1. Fitness of individuals with phenotype $z=\theta$ when inhabiting a patch with local optimum of $-\theta$ (fitness of hybrids with $z=0$ shown in brackets) for the three values of ω^2 used in the simulations.

		Local optimum ($\pm\theta$)	
		1	2.5
Width of fitness function (ω^2)	2.5	0.45 (0.82)	0.007 (0.28)
	5	0.67 (0.90)	0.08 (0.53)
	25	0.92 (0.98)	0.61 (0.88)

in Table 1 (with the exception of $\theta = 2.5$ and $\omega^2 = 2.5$, which results in stronger selection). A recent review by Hereford (2009) found similar fitness values. For each of these combinations of optimum and selection, we ran a range of migration rates from 10^{-6} up to 10^{-1} and a range of per-locus mutation rates from 10^{-6} up to 10^{-3}. Except where specifically indicated otherwise, all results shown below are for simulations with a patch size of $N = 1000$, a mutation effect size of $\alpha = 0.5$, a recombination rate of $r = 0.02$ between adjacent loci, $n = 50$ loci, and $V_E = 0.5$. Choosing a biologically representative level of V_E is complicated by trying to scale it both to V_S ($V_S = \omega^2 + V_E$) and to V_M. Reviews by Johnson and Barton (2005) and Stinchcombe et al. (2008) concluded that the strength of stabilizing selection is often stronger (i.e., V_S is smaller) than the values of V_S/V_E of 10–100 typically used in theoretical literature; whereas estimates of V_E/V_M are typically in the range of 100–1000 (Houle et al. 1996). Our choice of parameters for ω^2 yields values of V_S within this range, whereas our choice of mutational parameters yields $V_M = 25\mu$, resulting in $V_E = 2000\ V_M$ for $\mu = 10^{-5}$, but $V_E = 20\ V_M$ for $\mu = 10^{-3}$. In any case, these variables (N, r, n, α, and V_E) had very limited effects on the results of the simulations; as such, we limit our discussion here to the major effects caused by migration, mutation, and selection and discuss the effects of these other variables in the Supporting Information (Figs. S2–S6).

Results

In all cases examined, the mean divergence in phenotypes in the simulations either equaled or exceeded the predictions of both the GAM and the discrete GAM, meaning that gene flow imposed less constraint on adaptation than predicted under the GAM. The discrete GAM generally predicted less constraint than the GAM, but still predicted more constraint than the simulations (Fig. 2). Models agreed in the less interesting set of parameters leading to nearly complete adaptation (with $m < 10^{-4}$) or constraint (with $m > 0.1$, $\omega^2 = 25$). The difference between the predictions was most pronounced in the region of parameter space where the GAMs predicted some intermediate level of constraint, with the difference depending heavily upon the migration (m) rate and the combination of the strength of selection (ω^2) and difference in optima. In some cases, the GAM predicted the phenotype would deviate from its local optimum by as much as ~25% (~15% for the discrete GAM), where the simulations predicted nearly perfect adaptation (Fig. 2A–C). At intermediate-to-strong selection ($\omega^2 \leq 5$) and larger differences in optimum, the greatest discrepancies were seen at $m > 10^{-3}$; at weaker strengths of selection ($\omega^2 = 25$) the greatest discrepancies were seen at intermediate migration rates, where $10^{-4} < m < 10^{-2}$ (Fig. 2A–C). The effect of mutation was less pronounced, but in all cases, higher mutation rates resulted in greater agreement between the two approaches (Fig. 2D–F).

The differences between the results of the approaches can be quantified by comparing the deviation from optimum predicted under the GAM relative to the deviation from optimum found in the simulations (hereafter referred to as the "discrepancy score")

$$\frac{\theta - Z^*_{GAM}}{\theta - Z^*_{sim}} \tag{6}$$

(although this measure exaggerates the degree of difference between the models, it provides a useful means to explore the causal basis for the differences). To select some illustrative examples, when $\mu = 10^{-5}$, $m = 10^{-3}$, and $\theta = \pm 2.5$, the GAM predicted 21.3, 17.9, and 20.0% deviation from the optimum for $\omega^2 = 2.5$, 5, and 25, respectively, whereas the simulations found deviations of 0.19, 0.65, and 2.1% for the same values (as shown in Fig. 2). By contrast, when $\mu = 10^{-3}$, the same choices of parameters gave GAM predictions of 0.58, 0.68, and 1.0% versus 0.06, 0.1, and 0.68% in the simulations. Stated as discrepancy scores, at $\mu = 10^{-5}$, the GAM predicted approximately 113, 27, and 10 times more constraint than the simulations, whereas at $\mu = 10^{-3}$, it predicted ~10, 7, and 1.6 times more constraint (parameter values as above).

We found a strong positive relationship between the genetic skew measured in the simulations and the discrepancy score (Fig. 3A), suggesting that skew is indeed important. This relationship persisted after correcting for the differing contributions of the migrant classes to the total population response to selection using the discrete GAM, suggesting that these differences in accounting cannot explain all of the discrepancies between the GAM and the simulations. The region of parameter space with the highest discrepancy scores corresponds to the region of highest genetic skew (i.e., at intermediate migration rates). Genetic skew then decreases at higher migration rates as population divergence at equilibrium decreases (Fig. 3B).

To explore the causal basis for the differences between the predictions of the GAM and the simulations, we simulated the change in mean due to migration and response to selection within a single generation using the actual distribution of genotypes sampled at the end point of each simulation replicate. We then compared this to the response to selection from a randomly generated normally distributed population with the same genotypic mean and variance as the postmigration distribution of genotypes. For each parameter set with $m > 0.001$ (i.e., with at least one migrant per generation), we performed 30 random draws of genotype values to generate postmigration distributions and then performed 30 replicate responses to selection on each of these distributions. These simulations showed that the change in mean genotype due to selection (Δz^s) within a single generation was greatly affected by the skew in the distribution of genotypes. Normally distributed populations had much lower variance in fitness (not shown) and response to selection (Fig. 3C) than the simulation-result

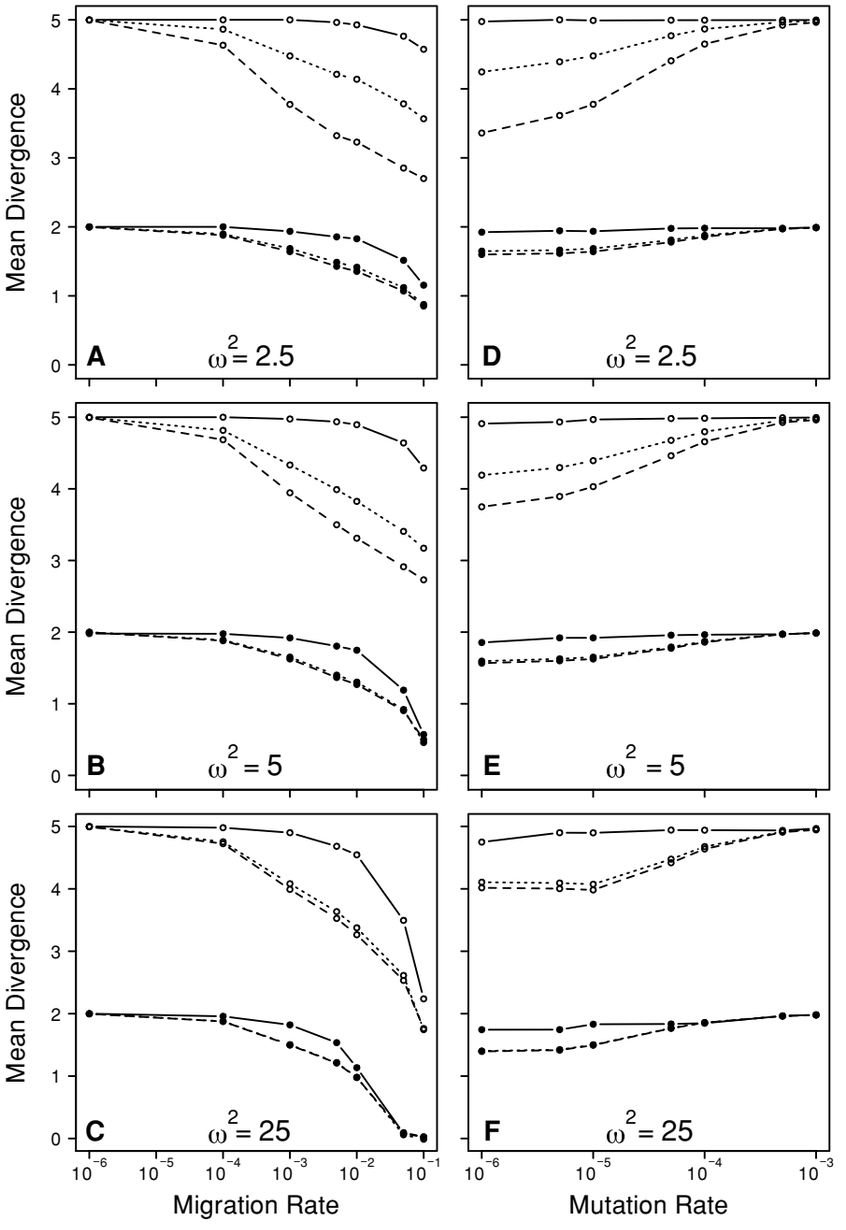

Figure 2. **Mean divergence as a function of migration rate (A–C) and mutation rate (D–F) for the simulations (solid lines) and the corresponding GAM (dashed lines) and discrete GAM predictions (dotted lines). Optimum values are $\theta = \pm 2.5$ (open circles) and $\theta = \pm 1$ (filled circles); strength of stabilizing selection within each patch is: 2.5 (A and D); 5 (B and E); 25 (C and F). For (A–C), the per locus mutation rate (μ) is 10^{-5}; for (D–F), the migration rate (m) is 10^{-3}. Each simulated point represents the average of 20 replicates (SE bars too small to show clearly).**

populations with large amounts of skew but the same mean and genetic variance. These results conform with the general expectation under Fisher's fundamental theorem, wherein the rate of change in fitness of a population is equal to its genetic variance in fitness (Fisher 1930). In all of the simulations, skew was generated with the opposite sign as the direction of selection (i.e., in the direction of immigration); in principle, skew could also reduce the magnitude of the selection differential when in the same direction as selection, depending on the curvature of the fitness surface, distribution of phenotypes, and resulting variance in fitness (T. Day, pers. comm., 2009). Finally, all else being equal, as mutation rate increased, genetic variance increased and skew in the distribution of genotypes decreased, due to changes in the relative contributions of mutation and migration to genetic variation.

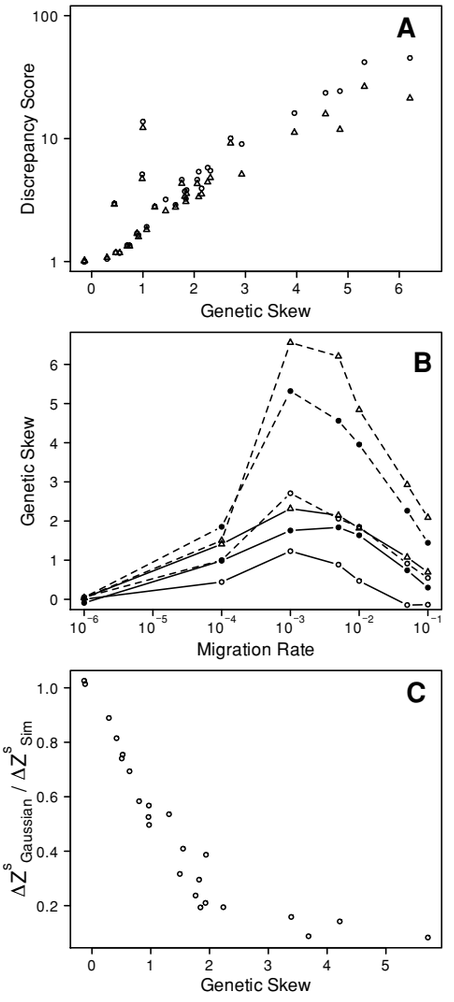

Figure 3. **Relationship between (A) genetic skew and the discrepancy score for predicted constraint at equilibrium (from eq. 6); (B) migration rate and genetic skew; (C) genetic skew and the ratio of response to selection for simulated normally distributed populations versus the distributions from the individual-based simulations. Symbols in (B) represent values of ω^2 of 2.5 (triangles), 5 (filled circles), and 25 (open circles); optimum values are $\theta = \pm 1$ (solid lines) and $\theta = \pm 2.5$ (dashed lines). Triangles in A are for the discrete GAM, open circles are for the GAM. Points in (A and C) are taken from the same parameter sets used in Figure 2A–C, excluding all points where mean genotype in the simulations was not significantly different from the optimum (A) or where $m \leq 0.001$ (C).**

Thus, the greatest differences between the predictions of the GAM and the simulations are seen when migration causes substantial deviations from normality in the distribution of breeding values, and this is typically most pronounced at lower mutation rates. At higher mutation rates, mutation contributes relatively more to genetic variance and the skewing effect of migration is less pronounced, resulting in a better agreement between the predictions of the two approaches.

GAM VERSUS DISCRETE GAM

The multiple-class version of the GAM we derived captures a part of the effect of the departure from normality caused by intermediate migration and strong selection (Fig. 2). The discrepancy between the GAM and the discrete GAM thus correlates with genetic skew (not shown) and is caused by strong selection against hybrid and immigrant genotypes that the GAM ignores. Under weaker selection and small migration, the trait difference between the different phenotypic classes is reduced, which reduces the amount of genetic skew, and the discrete GAM reaches the same predictions as the GAM, closer to the simulation results (Fig. 2). The discrete GAM also corrects for another problem inherent in the GAM, which predicts that the equilibrium divergence (D^*) should scale linearly with D_θ for a given value of ω^2; the amount of constraint is thus independent of the total divergence between habitats assuming constant variances. By contrast, the discrete GAM accounts for the fact that migrants (and to a lesser degree the hybrids) should have a fitness that tends toward zero as the local optima diverge, for a constant curvature of the selection surface. To illustrate this effect, under a fitness surface of $\omega^2 = 10$, $V_G = V_P = 1$, and $m = 0.01$, the GAM predicts a migration load of 9% of D_θ, regardless of whether $\theta = \pm 0.1$ or ± 10. In the former case, a perfectly adapted individual in patch 1 would have fitness equal to 0.996 in patch 2 whereas in the latter case, its fitness would be nearly 0. The discrete GAM accordingly predicts a migration load of 17% and 0.002% for the first and second case, respectively.

EFFECT OF GENETIC ARCHITECTURE

All of the previous simulations draw mutations from a Gaussian distribution and thus allow mutations of a broad range of sizes to arise and contribute to adaptation (continuum-of-alleles model). Even so, in most cases, very few of the 50 loci in the model actually contributed to population differentiation at equilibrium for migration rates above $\sim Nm = 1$ ($m > 0.001$; Fig. 4). As a preliminary test of the effect of genetic architecture on our results, we reran a subset of the above simulations, constraining mutations at all loci to have the same effect size (± 0.1, with equal probability) and replacing the previous allelic value with the new mutation (rather than adding the new value to the old, as above). This diallelic model of genetic architecture is expected to generate predictions closer to the GAM (Tufto 2000). Our results confirm this expectation as the diallelic model generated considerably less skew than under the continuum-of-alleles model (Fig. 5A) and resulted in less divergence between the populations, matching the results from the discrete GAM closely when loci are unlinked (Fig. 5B). Linkage between loci in the diallelic mutation model can create

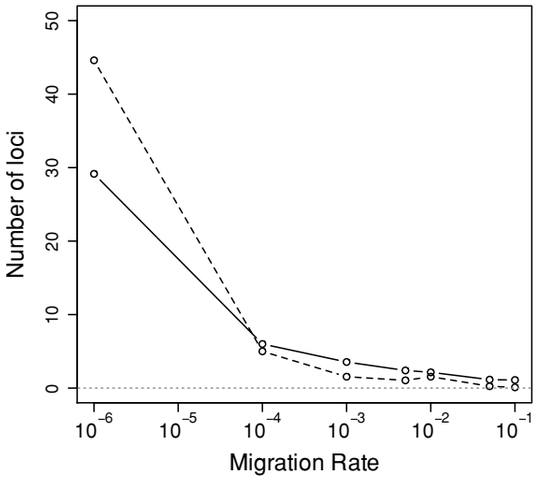

Figure 4. Average number of loci contributing to divergence between populations (i.e., where the most common allele in one patch differs from that in the other patch) for $\omega^2 = 2.5$ (solid line) and 25 (dashed line) and optimum values of $\theta = \pm 1$. All other parameters are as in Figure 2A–C.

groups that function effectively more like a single large locus and thus tend to behave more like the continuum-of-alleles model. As such, lower recombination rates in the diallelic mutation model resulted in more skew and a poorer agreement with the GAM predictions (Fig. 5B). By contrast, recombination had less effect on the results for the continuum-of-alleles model, where single large alleles can occur by mutation alone (Fig. 5B). A broad comparison across a range of strengths of selection and differences in optimum shows a close agreement between the predictions of the discrete GAM and the diallelic model, in contrast to the relatively poor agreement between either GAM and the continuum-of-alleles model. This demonstrates that discrepancies are generated by ignoring both selection on hybrids (in the GAM) and departure from normality in the distribution of genetic values (in the GAM and discrete GAM). This also illustrates the importance of genetic architecture and the difficulty of accurately modeling migration–selection processes using the Gaussian approximation. Biologically realistic genetic architectures will likely fall somewhere between the two extremes modeled here.

Discussion

The aim of the GAM is to provide a means to quantitatively assess whether gene flow is responsible for the apparent deviations from optimal local adaptation often found in natural populations. Based on the results of our analysis, it will be difficult to have confidence in the application of models based on the Gaussian approximation to generate quantitative predictions for natural populations in many cases of empirical relevance. The predictions of the GAM were most accurate in cases that generated very little genetic skew: at very low ($m \ll 0.0001$) and very high migration rates ($m \gg 0.05$) and at weak strengths of selection and small differences in optimum. Intermediate migration rates and stronger selection can generate more genetic skew (Fig. 3B), which decreases the accuracy of the Gaussian approximation for predicting selection within a single generation (Fig. 3C). This resulted in GAM predictions that were much less accurate (Fig. 3C) and depended heavily upon the mutation rate (Fig. 2D–F) and genetic architecture (Figs. 5 and 6). These results are consistent with the hypothesis that the GAM would underestimate the response to selection most severely when the change in mean phenotype due to migration (Δz^m) is large relative to the standing genetic variation (V_G; see Fig. 1; Tufto 2000). The ratio $\Delta z^m/V_G$, should be largest under low mutation (low genetic variance) and intermediate migration rates that cause high skew (large within-generation

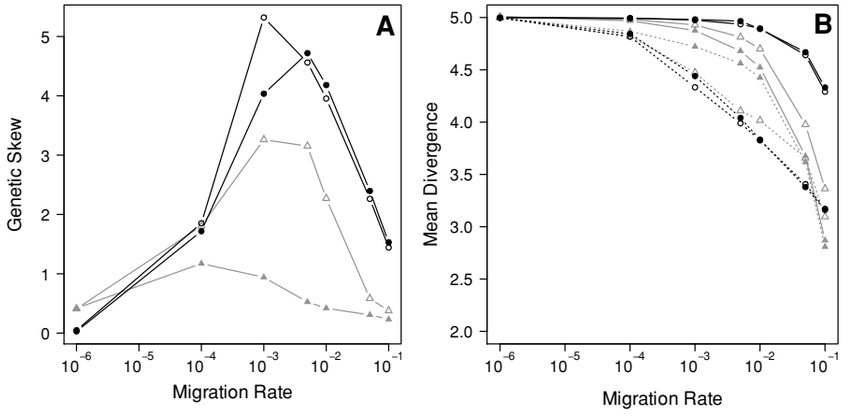

Figure 5. Genetic skew in patch 1 (A) and the mean divergence (B) as a function of migration for the diallelic mutation model (gray; triangles) and the continuum-of-alleles mutation model (black; circles) for $r = 0.02$ (open symbols) and $r = 0.5$ (filled symbols). Solid lines in B are for simulations, dotted lines are for the discrete GAM predictions; $\omega^2 = 5$, $\theta = \pm 2.5$, $\mu = 0.00001$.

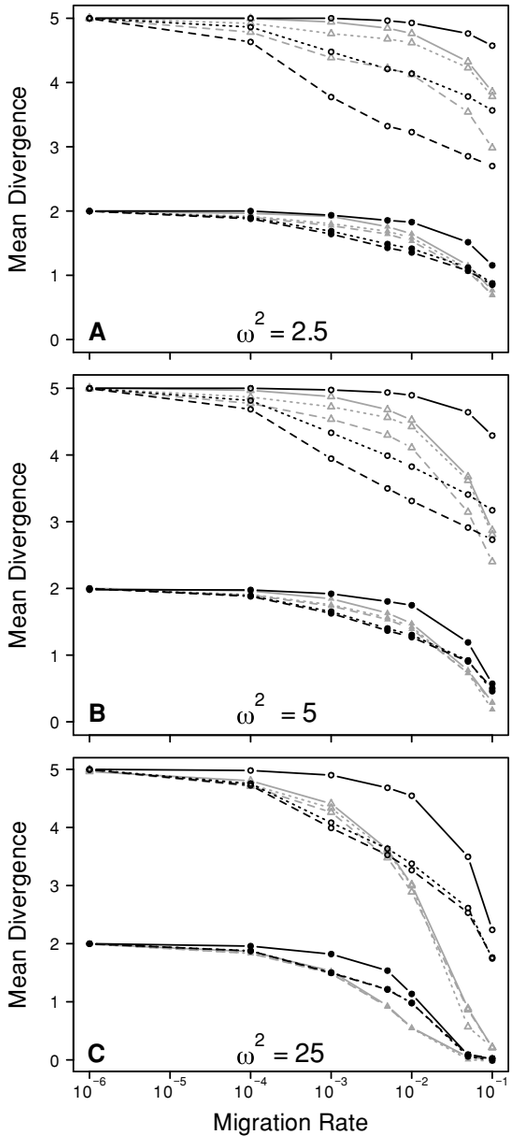

Figure 6. Mean divergence as a function of migration rate for the continuum-of-alleles model (black; circles) and the diallelic mutation model with $r = 0.5$ (gray; triangles), with the corresponding GAM (dashed) and discrete GAM (dotted) predictions. Filled symbols are $\theta = \pm 1$; open symbols are $\theta = \pm 2.5$. All other parameters are identical to those in Figure 2 A–C.

change in mean), which is where the response to selection is most poorly approximated by a normal distribution (Fig. 3C), and where the discrepancy scores are maximized (Fig. 3A). The corrections made to account for the differing contributions of migrant classes (the discrete GAM) provided an improved fit to the simulation results but were unable to resolve all of the discrepancies generated under the continuum-of-alleles model. Taken together, our results suggest that the role of migration in constraining adaptation may often be less important than expected under models that employ a Gaussian approximation to represent genetic variation. The Gaussian approximation works reasonably well when there is either near-perfect adaptation or near-perfect constraint, but is much less reliable when dealing with the more interesting cases with intermediate constraint, where its accuracy is heavily dependent upon the mutation rate and genetic architecture of the trait. It is not clear how these problems would affect modifications of the model to include habitat choice (e.g., Bolnick et al. 2009), but qualitative conclusions about the effect of habitat choice would likely be robust to genetic architecture.

Although Lopez et al. (2008) used simulations to test an analytical model similar to the GAM, they concluded that departures from a Gaussian distribution of breeding values caused by migration would only result in inaccurate predictions under the most heterogeneous environments and highest migration rates. By contrast, we find considerable discrepancies between the simulations and GAM predictions across a wide range of strengths of selection and migration rates. The principal difference between the present study and that of Lopez et al. (2008) is the range of mutation rates considered; Lopez et al. examined dynamics under high mutation rates ($\mu = 0.005$ and 0.0005; $n = 10$; yielding a per-trait mutation rate of $U = 10^{-2}$ and 10^{-1}) whereas our study used a much broader range of mutation rates ($10^{-6} < \mu < 10^{-3}$; $n = 50$; yielding per-trait mutation rates of $10^{-4} < U < 10^{-1}$). Like Lopez and colleagues, we found a good agreement between predictions generated under high mutation rates ($U = 0.1$; $\mu = 10^{-3}$), but found a much poorer agreement at lower mutation rates ($U \leq 0.01$; $\mu \leq 10^{-4}$). This is because Gaussian mutation generates variation in both directions, opposing the directional variation introduced by skew when migration rates are low relative to mutation rates. At the present time, our understanding of biologically reasonable mutation parameters is very limited and faced with seemingly contradictory lines of evidence; per-locus mutation rates estimated from allozyme polymorphism, visible, or lethal mutations typically seem to be on the order of 10^{-5} or 10^{-6} whereas per-trait mutation rates are on the order of 10^{-2}, which would seem to require an unrealistically large number of loci per trait or extensive pleiotropy (Barton and Turelli 1989). If the estimations of per-trait mutation rate are more accurate than per-locus estimates, then there are less serious problems with applying the GAM to natural populations. If, however, a substantial fraction of mutations affecting quantitative traits have unconditionally deleterious pleiotropic side effects, the effective rate of mutations contributing to trait variance in the manner modeled by both the simulations and the analytical theory discussed here would be much lower than empirical estimates (which likely include mutations with deleterious fitness consequences). Regardless of whether per-locus or per-trait estimates of mutation are more accurate, the results of this study clearly illustrate the sensitivity of the Gaussian approximation to these variables across

biologically realistic regions of parameter space, undermining the application of such models to natural populations.

Taking a step back from the specific problems associated with the GAM, the comparison illustrated in this study raises important questions about how to accurately model evolution in quantitative traits, given the inadequacy of the Gaussian approximation under a large region of parameter space. Turelli and Barton (1994) found that the Gaussian approximation provided a reasonably accurate description of evolutionary change under the infinitesimal assumption (i.e., infinite number of loci of small effect) for a range of selection regimes (without migration), suggesting that departures from normality produced by linkage disequilibria are unlikely to be important for traits with this genetic architecture. They performed numerical tests of their results using multilocus diallelic models with equal effect size alleles and generally found a good agreement with the analytical approximations. They also suggested, however, that departures from the infinitesimal assumption (i.e., fewer loci with heterogeneous effect sizes) could cause departures from normality to be more important, but did not test this explicitly. Tufto (2000) also compared an analytical model using the Gaussian approximation to the multilocus diallelic model for a single population experiencing stabilizing selection and immigration of individuals with nonlocal genotypes. Tufto concluded that discrepancies between the analytical and the diallelic model are "a result mainly of the increased genetic variance generated by migration and only to a small extent a result of deviations from normality" (Tufto 2000, p. 291). He showed that deviations from normality were unlikely to be important except under very strong selection or large deviations in migrant genotype. Tufto also noted that large differences in allele frequency among loci could also affect the accuracy of the Gaussian approximation, but suggested that these were unlikely to be important at migration–selection equilibrium. In the present study, we found a relatively good agreement between the discrete GAM and the diallelic mutation model (which is similar to the diallelic models described above) but found a poor agreement between the GAMs and the continuum-of-alleles model. The discrete GAM is thus a good approximation of the effect of migration on traits that are well behaved in regard to the Gaussian assumption. However, when genetic architecture was characterized by a few large effect mutations or linkage groups contributing most of the variation between populations (Fig. 4), migration could generate considerable genetic skew (Fig. 5), resulting in larger discrepancies between the discrete GAM and the simulations (Fig. 6). Essentially, the discrete GAM fails because it cannot properly approximate the shape of the genotypic distribution generated under the continuum-of-alleles model; the diallelic model generates less-complex distributions, and as such, can be more accurately approximated. Migration–selection balance seems to favor local adaptations based on genetic architectures characterized by divergence at only a few large loci. This is likely due to the homogenizing effect of gene flow, which would be more strongly opposed by the larger selection coefficient of a single large locus (compared to several unlinked loci with small coefficients). The diallelic model was more sensitive to recombination rate (Fig. 5) because it is limited by allele effect size; the only way that evolution can "build" a large effect locus under this model is through linkage under a reduced recombination rate. The continuum-of-alleles model is not as limited by mutations and is thus able to "build" single large alleles without reduced recombination and linkage; as such, it is less affected by recombination rate than the diallelic model and leads to a far smaller constraining impact of gene flow on adaptation.

Although many studies of migration–selection balance assume diallelic loci of equal effect size to model evolutionary processes (e.g., Phillips 1996; Lythgoe 1997; Spichtig and Kawecki 2004), nature is unlikely to be so uniform. Orr (1998) showed that the distribution of mutation effect sizes fixed during adaptive evolution (without migration) would be approximately exponential under many different mutation models. Griswold (2006) showed that adaptation under migration–selection balance would result in fewer loci of small effect than predicted by Orr, but there was nevertheless considerable heterogeneity in mutation effect sizes contributing to divergence. Although it is unclear how closely these models resemble reality, they suggest that the assumption of infinitely many alleles of small effect does not have strong theoretical support. Drawing on evidence from the natural environment, many QTL studies have found a large proportion of trait variation between closely related species or subspecies can be explained by a few linkage groups with large effects (Orr 2001), although we still know very little about the variation in genetic architecture across a wide range of organisms. Given the prevalence of genetic architectures that depart from the infinitesimal assumption and the predictions of the adaptation theory described above, our study suggests that accounting for genetic architecture is indeed important in modeling any evolutionary process that could generate genetic skew (not to mention the higher moments of the distributions, which were not considered here but could be equally important). As such, it may be worthwhile to revisit other related models of migration–selection processes that have used the Gaussian approximation to see if their findings are robust to different types of genetic architecture (e.g., Garcia-Ramos and Kirkpatrick 1997; Kirkpatrick and Barton 1997; Filin et al. 2008).

It is important to note that even with an exact model, any predictions will only be as accurate as the parameters used in their calculation. Both the GAM and the simulations require the estimation of several parameters, each of which typically has large margins of error. Without an estimate of the compounded error from these terms, it is not possible to assess the confidence we have in any estimate of the concordance between the model predictions

and empirical observations. Furthermore, because a considerable variation in population divergence may be expected from one generation to the next due to the stochastic nature of the processes involved, inferences based on observations from a single pair of populations over a short time scale will likely have extremely large confidence intervals. Finally, it should be emphasized that the systems under study may be far away from the equilibrium state implied by the modeling approach. In many cases, the local adaptation process may be incomplete due to recent extirpation and recolonization events or changes in the environment. Depending on the efficacy of selection and the standing genetic variation, adaptation following such disturbances could take many generations to reach equilibrium, even in the absence of migration load. Hendry et al. (2001) showed that in many cases populations will evolve to within 10% of their expected mean phenotype at equilibrium in less than 50 generations, but their analysis was based on strong selection and high migration rates, which accelerate the approach to equilibrium. These considerations have not typically been addressed in the studies that have applied the GAM to testing hypotheses in nature. For example, the parameters estimated in Moore et al. (2007; $0.0005 < m < 0.0116$; $V_P = 1.04$; $V_G = 0.3$; $51 < \omega^2 < 3999$) result in times to approach within 10% of the expected equilibrium phenotype ranging from 132 to 4004 generations, using equation (13) from Hendry et al. (2001). It is difficult to reject the alternative nonequilibrium hypothesis without performing manipulation experiments (e.g., Riechert 1993), observations of populations following known changes in gene flow (Nosil 2009), or measuring (lack of) change in mean phenotype over many generations.

In summary, we have shown that the assumption of normally distributed genetic values introduces unrealistic constraints into models of adaptation under migration load, causing them to misrepresent the distribution of phenotypes that responds to selection under realistic assumptions about the genetic architecture of adaptive traits in nature. This can cause substantial inaccuracies when predicting response to selection and adaptive divergence at equilibrium, especially at intermediate migration rates and medium-to-strong selection, where gene flow can generate substantial genetic skew. This is precisely the region of parameter space where evolutionary constraints are expected and where accurate predictions are critical to testing hypotheses. Nevertheless, as a general approach, the discrete GAM can give better first approximations than the classic GAM. Although it is tempting to suggest simulations as an alternative means of formulating quantitative predictions, the complications involved in representing genetic architecture (e.g., Figs. 5 and 6) and our poor understanding of biologically reasonable parameters greatly limit our confidence in any such predictions. When confronting systems with many variables, we feel that experimental approaches are far superior to those based on model-fitting and observation. That said, it is clear that the modeling approach greatly helped us identify the key factors causing evolutionary constraints on adaptation under divergent selection and gene flow. In particular, given the results presented here, effects of the genetic architecture should be investigated when possible to explain discrepancies with theoretical results.

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Supporting Information

The following supporting information is available for this article:

Figure S1. Genetic variance in a homogenous population at equilibrium.

Figure S2. Predicted values of the mean genotype in patch 1 as a function of migration rate with different amounts of environmental variance (V_E).

Figure S3. Predicted values of the mean genotype in patch 1 as a function of mutation rate with different amounts of environmental variance (V_E).

Figure S4. Predicted mean genotype (A) and genetic variance (B) as a function of migration rate with different rates of recombination.

Figure S5. Mean genotype from the simulations versus GAM predictions as a function of migration rate (A) and mutation rate (B) with different mutation effect sizes (α).

Figure S6. Equilibrium phenotypic divergence as given by numerically solving equation S1 (dots) and by the approximation S2 (lines) for $V_E = 0.5$, $V_S = \omega^2 + V_E = 2.5$ (red) and $V_S = 25$ (blue), and for $\theta_i = \pm 1$ and ± 2.5 (with $V_P = 1$, and 2, respectively).

Supporting Information may be found in the online version of this article.
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