Geographical patterns of adaptation within a species' range: interactions between drift and gene flow

M. ALLEAUME-BENHARIRA,* I. R. PEN*† & O. RONCE*

*Laboratoire Génétique et Environnement, Université de Montpellier II, Institut des Sciences de l'Évolution, Montpellier Cedex, France
†Theoretical Biology Group, Centre for Ecological and Evolutionary Studies, University of Groningen, Kerklaan, The Netherlands

Abstract

We use individual-based stochastic simulations and analytical deterministic predictions to investigate the interaction between drift, natural selection and gene flow on the patterns of local adaptation across a fragmented species' range under clinally varying selection. Migration between populations follows a stepping-stone pattern and density decreases from the centre to the periphery of the range. Increased migration worsens gene swamping in small marginal populations but mitigates the effect of drift by replenishing genetic variance and helping purge deleterious mutations. Contrary to the deterministic prediction that increased connectivity within the range always inhibits local adaptation, simulations show that low intermediate migration rates improve fitness in marginal populations and attenuate fitness heterogeneity across the range. Such migration rates are optimal in that they maximize the total mean fitness at the scale of the range. Optimal migration rates increase with shallower environmental gradients, smaller marginal populations and higher mutation rates affecting fitness.

Introduction

Understanding species' distributions has become critically important for biodiversity conservation (Gaston, 1996; Channell & Lomolino, 2000) in the context of a changing climate and an increasing fragmentation of natural habitats. Species living in a fragmented or discontinuous habitat form a web of interconnected entities. Migration guarantees the integrity of the species from both demographic and genetic perspective. In particular, connectivity permits recolonization after local extinction. The genetic consequences of migration at the scale of a species' range are however more ambiguous. Mayr (1963) emphasized that gene flow is an essential mechanism maintaining genetic and phenotypic homogeneity within a species. Yet, such homogeneity might not

Correspondence: Mariane Alleaume-Benharira, Laboratoire Génétique et Environnement, CNRS UMR 5554, Université de Montpellier II, Institut des Sciences de l'Évolution, CC065, USTL, Place Eugène Bataillon, FR-34095, Montpellier Cedex 05, France.
Tel.: +33-4-67144718; fax: +33-4-67143622;
e-mail: alleaume@isem.univ-montp2.fr

always be desirable for conservation purposes. In particular, if natural selection is spatially variable, genetic homogeneity induced by gene flow occurs at the cost of local maladaptation, which may potentially jeopardize the local or global persistence of the species. Local adaptation describes the adequacy between the phenotypes and the local environment. In this context, natural selection, which increases the frequency of locally adapted genes, interacts with gene flow, which introduces potentially maladapted genes that have been selected elsewhere (Lenormand, 2002). The abundant evidence of genotypic clines within species' ranges exemplify the fact that selection often opposes gene flow in maintaining population differentiation within the range (Ehrlich & Raven, 1969; Magiafoglou et al., 2002).

Several theoretical models have investigated the consequences of migration for the evolution of patterns of local adaptation within a species' range (starting with Haldane, 1948; for clines in quantitative characters see Felsenstein, 1977; Slatkin, 1978; Pease et al., 1989). Garcia-Ramos & Kirkpatrick (1997) describe a quantitative genetics model for a species continuously distributed

through space along some environmental gradient, which generates clinal selection on a phenotypic character. They assume that the species' density is fixed through time, but decreases from the centre to the margins of the range. Such distribution of density (sometimes referred as the 'abundant centre distribution') is both expected for theoretical reasons and empirically observed in many species' ranges (Brown, 1984; for a review see Brewer & Gaston, 2002; Lonn & Prentice, 2002). These demographical asymmetries lead to a net flow of individuals from core populations towards the margins. Garcia-Ramos & Kirkpatrick (1997) show that such asymmetric gene flow can result in phenotypic clines at the scale of the species' range deviating from optimal values. Increased migration then generates higher maladaptation in peripheral populations.

Gene swamping may however not be the single force opposing natural selection in small peripheral populations. Due to their small size, genetic drift may be important in such populations. There is circumstantial empirical evidence that genetic diversity is indeed reduced at the margins of several species' ranges (Cote *et al.*, 2002; Lonn & Prentice, 2002), or that drift could be a potent force in such populations due to both reduced and more variable density (Vucetich & Waite, 2003). Yet, the previous deterministic models of adaptation at the scale of a species' range (Garcia-Ramos & Kirkpatrick, 1997; Kirkpatrick & Barton, 1997) neglect genetic drift by considering local population of infinite size (but see Butlin *et al.*, 2003). Theoretical studies investigating the effect of drift on the evolution of gene frequency along environmental gradients with constant density (Hastings & Rohlf, 1974; Felsenstein, 1975; Slatkin & Maruyama, 1975; Nagylaki, 1978) have however shown that gene flow, rather than cancelling the effect of selection, could help mitigate the effect of drift and maintain smooth clines along such environmental gradients. Theoretical studies of source-sink systems taking into account both genetic drift and demographical asymmetries have also shed a different light on the effect of migration on local adaptation in heterogeneous environments. For example, Gomulkiewicz *et al.* (1999) study probabilities of establishment for beneficial alleles in sinks. They show that dispersal could have negative and positive effects on local adaptation. In particular, migration increases the probability of the appearance of fit alleles in the sink (see also Holt *et al.*, 2003), both by introducing variability from the source and by increasing the sink population size. However, the probability of long-term persistence of fit alleles in small sink populations decreases with migration (Gomulkiewicz *et al.*, 1999). Many of the latter models have focused on the conditions facilitating the spread of an advantageous allele at the margins and on the initial process of niche expansion (Holt *et al.*, 2003), rather than investigating long-term patterns of adaptation within a fixed range. As migration may have

antagonistic effects on the two phases of the adaptation process (initial increase and equilibrium), it is often difficult to conclude about the optimal rate of connection in the long term. To what extent can the predictions of source-sink models with only two patches be extrapolated to clinal variation within a fragmented range? Conversely, can the predictions derived from models of clines with constant density apply to situations where density is higher at the core of the range? Butlin *et al.* (2003) use individual-based stochastic simulations to investigate the evolution of a species' range with assumptions very close to the deterministic model of Kirkpatrick & Barton (1997). Although their simulations take into account finite population sizes, they do not compare quantitatively their predictions to those of models ignoring drift and provide no clear conclusion regarding optimal rates of migration.

In this paper, we investigate the evolution of geographical patterns of adaptation at the scale of a species' range taking into account natural selection, genetic drift, mutation and migration. An analytical approach of the question is extremely difficult (for approximations assuming constant density throughout the range and ignoring mutations, see Nagylaki, 1978). We therefore resort to individual-based stochastic simulations and systematically compare our stochastic simulations outcomes to analytical predictions derived by ignoring various consequences of drift. We investigate the evolution of both clines in mean phenotype and clines in mean fitness across the range and show that these two measures of local adaptation respond differently to variation in gene flow. Our model predicts that intermediate levels of connectivity among discrete and finite sized populations in a fragmented species' range minimize fitness differences between the core and marginal populations and maximise the total mean fitness at the scale of the range. Such level of connectivity corresponds to some optimal migration rate, because it is the most desirable from a conservation perspective. Our model therefore focuses on the consequences of gene flow but does not address the question of the evolution of migration rates at the scale of the range (for a distinction between optimal and evolutionarily stable migration rates in presence of local adaptation see Billiard & Lenormand, 2005).

Model assumptions

Our aim is to understand the effect of genetic drift on the evolution of local adaptation within a species' range. The assumptions and explored parameters range are very similar to those of Garcia-Ramos & Kirkpatrick (1997), except that we consider discrete local populations with finite numbers of individuals and resort to individual-based simulations. The model describes the evolution of a quantitative trait in a species distributed patchily across a limited range. A fixed number of populations within the species' range are arranged linearly along some

environmental gradient. As observed in many species, the density decreases from the centre to the periphery of the range (Brown, 1984). More precisely, we assume that the carrying capacity of a population, $K(i)$, is fixed in time and declines with its distance from the centre of the range as a Gaussian function:

$$K(i) = K_{\min} + \Delta K e^{-\frac{x(i)^2}{2}}, \tag{1}$$

where $x(i)$ gives the spatial position of population i. The minimal carrying capacity in the two peripheral populations is $K_{\min}$. The maximum carrying capacity in the central population ($x(i) = 0$) is $K_{\max} = K_{\min} + \Delta K$ individuals. In practice, carrying capacities were rounded to the closest integer value to $K(i)$. In the simulations, the central population was kept to a constant size, but the size of the marginal populations ($K_{\min}$) was varied. Changes in density at the margins affect both the intensity of genetic drift and the spatial patterns of asymmetrical gene flow. In particular, if marginal populations are larger, we expect both gene swamping from central populations and genetic drift to be weaker forces compromising adaptation in such populations. In simulations, we always used the same total number of populations ($n_d = 27$ populations), the carrying capacity at the centre $K_{\max}$ was of 2100 adults and we contrasted situations where the maximal size at the margins $K_{\min}$ was either 10 or 100.

We are interested in the evolution of some phenotypic character z (for instance the flowering date or the resistance to desiccation stress) subject to heterogeneous selection within the species' range (see for instance Karan *et al.*, 1998; Rehfeldt *et al.*, 1999; Sgro & Blows, 2003). Environmental factors such as temperature or moisture usually change continuously across species' range (for instance, see Obrien, 1993). We assume that such environmental gradients result in the ecological optimum for the studied quantitative trait, varying as a linear function of space:

$$\theta(i) = x(i) \times b, \tag{2}$$

where b is the rate at which the ecological optimum changes as one moves across the range. Larger values of b correspond to more heterogeneous environments. Phenotypic optima do not vary with time in our model. In practice, b was fixed to either 0.1 or 1. Selection acts on the juvenile stage. The probability for an individual to recruit as an adult, $w_i(z)$, in population i, depends on the distance of its phenotype z to the local optimum $\theta(i)$. More precisely, fitness was modelled as a Gaussian function:

$$w_i(z) = e^{-\frac{(z-\theta(i))^2}{2\Omega}}, \tag{3}$$

where $1/\Omega$ measures selection intensity. High Ω results in weak selection, allowing phenotypes quite far from the local optimum to survive. Ω was fixed to 20.

The phenotype z of diploid individuals is determined by additive gene action at a single locus with many alleles.

There is no environmental effect on the expression of the phenotype. We used a random-walk model of mutation (Zeng & Cockerham, 1993). Mutation was modelled by assuming that, with probability μ, a random increment is subtracted or added to the current allelic value in the gamete. Mutational steps were drawn from a uniform distribution and take the maximal value $\delta\mu$. We considered this one locus phenotypic trait z to be equivalent to a polygenic trait which allowed us to study mutation rates per genome (μ) of $\mu = 10^{-3}, 10^{-2}$. The maximal mutational steps ($\delta\mu$) were 5 and 10. Preliminary comparisons with a biallelic multilocus simulation model gave qualitatively comparable results, i.e. intermediate migration rates maximized mean fitness (Frank Shaw, personal communication). Varying the number of loci may however have important quantitative consequences through effects on the genetic variance (see for instance Bürger & Gimelfarb, 1999), which we did not explore in the present paper.

The order of the events in the simulated life cycle was: (i) local reproduction, (ii) migration of the zygotes, (iii) selection, (iv) density regulation. Generations are nonoverlapping. Hermaphroditic mating occurs within the same local population under panmixia. Adults die after the reproductive period. Mutation of gametes occurs. New zygotes are formed and migrate after birth following a stepping stone model (for instance see Arnegard *et al.*, 1999) with absorbing boundaries (juveniles migrating out of the range limits disappear). In the simulations, the migration rate was 0, 0.5, 1, 2, 3, 4, 5, 7.5, 10 and 20%. Selection (genotype-dependant mortality) precedes density regulation (density-dependent, genotype-independent mortality). In the presented simulations, the level of adaptation does not influence the density of adults per population, which remains constant between generations. Therefore, in each deme, the population size was held at the carrying capacity.

Analysis of simulations

In contrast to deterministic models, in our individual-based simulations, once pseudo-equilibrium has been reached, population characteristics still fluctuate through time because of stochastic events (such as genetic drift). More than being disturbing background noise, such fluctuations have profound consequences for the evolution of adaptation within the range (see below). The simulation program recorded both spatial and temporal variation in population characteristics. For a given parameter set, a single simulation was run for 300 000 generations starting with random phenotypes across the range. The first 20 000 generations were not recorded. Every 100 generations, the mean phenotype $\bar{z}(x,t)$, the phenotypic variance $\sigma_z^2(x,t)$ and the mean fitness $\bar{w}(x,t)$ were recorded in every population, where t denotes the generation and x the location of the population. Those statistics were further averaged across generations to

obtain $\overline{\overline{z}}(x) = E_t[\overline{z}(x,t)]$, $\overline{\sigma}_z^2(x) = E_t[\sigma_z^2(x,t)]$ and $\overline{\overline{w}}(x) = E_t[\overline{w}(x,t)]$. We also computed the mean fitness at the scale of the range as: $W_u = \sum_{i=1}^{n_d} \overline{\overline{w}}(x_i)/n_d$. Such measure gives the same weight to large central populations and small peripheral ones. Alternatively, we computed a mean fitness measure where local demes are weighted by their density: $W_w = \sum_{i=1}^{n_d} \overline{\overline{w}}(x_i)K(i) \Big/ \sum_{i=1}^{n_d} K(i)$.

Validation methods

In order to validate the simulation outputs and better understand the role of drift, we compare our results to analytical predictions derived with deterministic models. To describe the geographical patterns of adaptation within the species' range, we focus on (i) the clines in mean phenotype across the range $\overline{\overline{z}}(x)$ and (ii) the patterns of spatial variation in mean fitness $\overline{\overline{w}}(x)$. While many empirical and theoretical studies have investigated patterns of phenotypic differentiation across species' range, variation in mean fitness is likely to be a better predictor of the potential demographic consequences of patterns of adaptation, which, however, we did not studied in the present paper.

Garcia-Ramos & Kirkpatrick (1997) predict that, under assumptions very similar to our model, the cline in mean phenotype across the species' range should be linear with slope:

$$b^* = \frac{b\sigma_A^2}{\sigma_A^2 + m(\Omega + \sigma_z^2)}, \qquad (4)$$

where σ_A^2 and σ_z^2 are, respectively, the local additive genetic variance and total phenotypic variance for the phenotypic trait involved in local adaptation. Equation (6) written by Garcia-Ramos & Kirkpatrick (1997) for the slope of the cline is slightly different because they standardize the phenotypic measures so that $\sigma_z^2 = 1$. Their model assumes that the additive genetic variance does not vary across the range. Such an assumption is violated in the simulations, where additive genetic variance varies with distance from the centre of the range. We replaced the variance terms in eqn 4 by the variance measured in the simulations, $\hat{\sigma}_z^2 = E_x[\overline{\sigma}_z^2(x)]$, averaged over the ten central populations (of a total of 27 populations, see above for how statistics were computed from the simulations), for which the variance was observed to be approximately constant in our simulations. Given that environmental effects on the phenotype are absent in the simulations and all gene action is purely additive ($\sigma_z^2 = \sigma_A^2$), the predicted slope for the cline in mean phenotype across the range is:

$$b_{\mathrm{pred}} = \frac{b\hat{\sigma}_z^2}{\hat{\sigma}_z^2 + m(\Omega + \hat{\sigma}_z^2)}, \qquad (5)$$

where the migration rate, m, and the inverse of selection intensity, Ω, are simulations input values. No hypothesis is made concerning phenotypic distributions in our simulations. Therefore, in particular, no assumptions of variance constancy and Gaussian distributions are made. The theoretical slope b_{pred} is compared to the slope estimated from the simulation results by a linear regression of observed mean phenotypes $\overline{z}(x)$ on spatial position, x. Such estimated slope will be noted b_{sim}. The predicted slope of the mean phenotype cline in mean phenotype is always smaller than the slope of environmental gradient, b. It informs us about the extent of mean genetic differentiation within the range. For the same environmental gradient, shallower slopes reflect more homogenous species and poorest levels of local adaptation at the periphery of the range.

Here, we derive some simple predictions for the mean fitness as a function of spatial location assuming that spatial characteristics of populations do not fluctuate through time once equilibrium has been reached. By assuming a gaussian distribution of phenotypes within each population, one can write (for instance see Bürger & Lynch, 1995):

$$\overline{\overline{w}}(x) = \sqrt{\frac{\Omega}{\Omega + \sigma_z^2(x)}} \exp\left(\frac{-(\overline{\overline{z}}(x) - bx)^2}{2(\Omega + \sigma_z^2(x))}\right). \qquad (6)$$

Such distribution of fitness in space can be summarized by several statistics. Here, we chose to focus on the curvature of this distribution close to the centre of the range, which measures how fast mean fitness declines when moving towards the periphery of the range, reflecting the geographical pattern of adaptation. Assuming that the phenotypic variance does not vary spatially ($\overline{\sigma}_z^2(x) \approx \hat{\sigma}_z^2$) and replacing $\overline{\overline{z}}(x)$ by $b_{\mathrm{sim}}x$, one can approximate the function in (6) near the centre of the range (x close to 0) by a parabola:

$$\overline{\overline{w}}(x) = \sqrt{\frac{\Omega}{\Omega + \hat{\sigma}_z^2}} - \left(\frac{(b_{\mathrm{sim}} - b)^2 \Omega^{1/2}}{2(\Omega + \hat{\sigma}_z^2)^{3/2}}\right) x^2, \qquad (7)$$

whose curvature is:

$$c_{\mathrm{pred}} = -(b_{\mathrm{sim}} - b)^2 \frac{\Omega^{1/2}}{2(\Omega + \hat{\sigma}_z^2)^{3/2}}. \qquad (8)$$

The absolute value of the predicted curvature in (8) decreases with increasing match between mean phenotypes and the environment [smaller values of $(b_{\mathrm{sim}} - b)^2$], but also with higher local phenotypic variance $\hat{\sigma}_z^2$. Such analytical predictions however neglect the effect of temporal fluctuations in the mean phenotype and of phenotypic variance. If the mean phenotype at location x fluctuates around $\overline{\overline{z}}(x)$, then the average distance between the mean phenotype and the ecological optimum can be in practice much greater than $(\overline{\overline{z}}(x) - bx)^2$ and the average mean fitness much smaller than predicted by (6) and (7). We therefore estimated a curvature c_{sim}, from the simulations, by fitting a parabolic curve to the observed averaged mean fitness $\overline{\overline{w}}(x)$ as a function of x. Comparing estimated curvature to that predicted by (8) allows us to better understand the role of
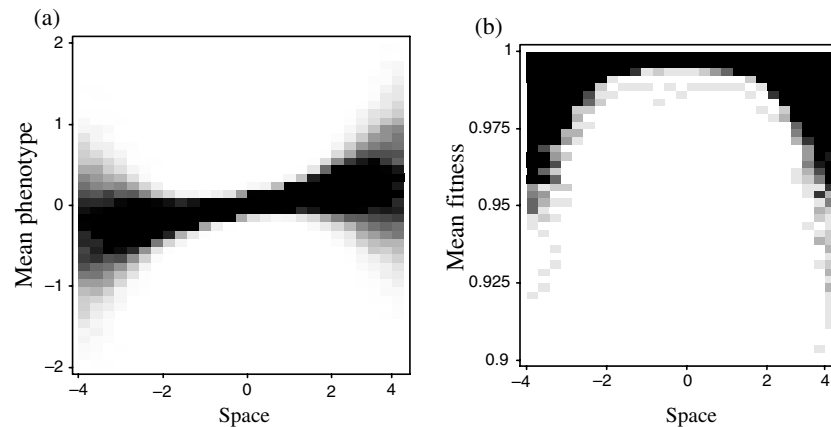
Fig. 1 Simulated distribution through time of mean phenotype and mean fitness across the range. (a) Mean phenotype and (b) mean fitness in populations as a function of space during 300 000 generations (recorded every 100 generations) for a given simulation with parameters: $K_{min} = 10$, $b = 0.1$, $\mu = 0.01$, $d\mu = 10$, for $m = 0.01$. Recorded values were pooled in intervals of size 0.1 for panel (a) and 0.0025 for panel (b); the grey level, in the corresponding cells, reflects the density of observed values. Note for panel A that optima ranged from $\theta(i) = -0.4$ up to $\theta(i) = 0.4$ for the two peripheral populations.

stochastic fluctuations due to drift in shaping the spatial patterns of variation in mean fitness.

Results

Figure 1 shows a typical example of a single simulation with the distribution of mean phenotypes and mean fitnesses per locality for 3000 censuses through time (spanning over 300 000 generations). Variation of mean phenotypes through time is larger at the periphery, with mean phenotypes in the same locality either much larger or much smaller than the optimum (Figure 1a). Population mean fitnesses (Figure 1b) are bounded up to one, whereby most simulated values lie, but, again, the temporal variability in mean fitness is much larger at the periphery than at the core. Measured fitness in marginal populations can therefore occasionally be much smaller than at the core. In the following, we present only results for temporal averages taken out over all censuses for the same parameter set, as explained in the Method section.

Simulated and predicted phenotypic and fitness clines

To explore the effects of drift, and more generally of stochastic variation in genotypic frequencies, we now compare the results of the simulations with the predictions of the deterministic models. To facilitate the comparison of such geographical data, we compare estimated and predicted slopes in mean phenotype and estimated and predicted curvature for mean fitness through space. We particularly address the effect of variation in migration rates on the evolution of spatial patterns within the range. Figure 2 shows, for two

different migration rates (i) the spatial patterns observed in the simulations, (ii) their fit to the predictions of a linear (for phenotypic clines) or parabolic (for fitness clines) variation through space, (iii) the predicted patterns using eqns 5 and 7. From simulated distributions, we can first observe that the mean phenotype in the central population is at its ecological optimum (Figure 2a, b). The distance between mean phenotype and local optimum increases as one moves away from the centre of the range and with increasing migration. For all populations, except the central one, gene flow is asymmetric and represents a directional force pushing the mean phenotype away from the ecological optimum. Gene swamping from large central populations then prevents relatively small peripheral populations from being close to their optimum. Second, the mean fitness declines with increasing distance from the centre (Figure 2c, d), consistent with increasing deviation of mean phenotype to optimum.

When comparing simulations and analytical predictions, we can observe that the linear and parabolic models do not entirely describe the spatial patterns. In particular, the characteristics of the populations at the edge of the range often deviate from those models. Yet, for most parameter sets (as illustrated in Figure 2) the overall fit is very good as analytical predictions describe quite accurately the mean characteristics of the simulated cline (the fraction of spatial variation in averaged mean phenotype explained by a linear model ranged from 98.92% to 99.87% and for the averaged mean fitness, variation explained by the parabolic model ranged from 89.01% to 96.09%). Note however that, at any point in time, the characteristics of the cline may deviate strongly from this average behaviour (see Fig. 1).
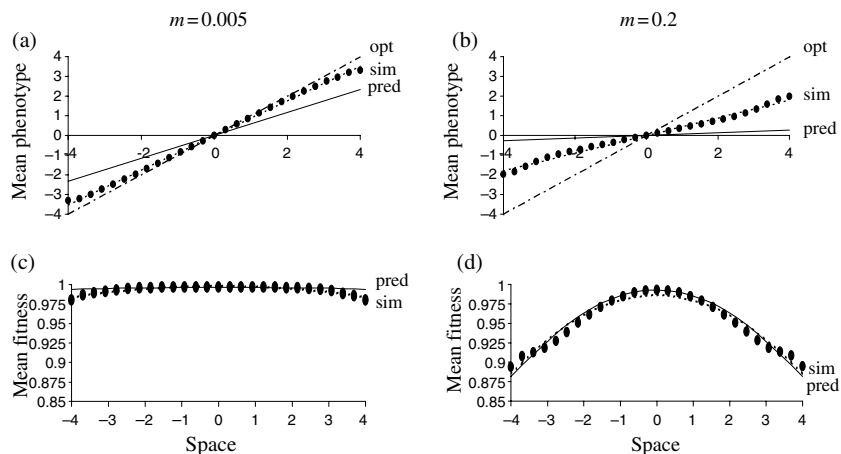
Fig. 2 Comparison of simulated and predicted clines in mean phenotype and mean fitness for two migration rates in the same landscape. (a, b) Clines for mean phenotype $\bar{z}(x)$ and optimal phenotype (dashed line); (c, d) Clines for mean fitness $\bar{w}(x)$, as a function of space. Closed circles: simulated value. Dotted line: estimated value by linear (a, b) or parabolic (c, d) regression. Solid line: predicted value by, respectively, eqn 4 in a, b and by eqn 7 in c and d. (a–c) small migration rate ($m = 0.005$) and (b–d) large migration rate ($m = 0.2$). $K_{min} = 10$, $b = 1$, $\mu = 0.01$, $d\mu = 10$.

Effect of migration on the slope of phenotypic clines

Figure 3 shows variations of estimated and predicted mean phenotype slopes when the migration varies. Both slopes decrease with increasing migration rate (Figs 2 and 3). More intense gene flow results in homogenisation of phenotypes across the range. Note, that the migration rate m affects the value of the slope directly (see eqn 4) but also indirectly through its effects on the estimated phenotypic variance $\hat{\sigma}_z^2$ (see eqn 4). The estimated slope from the simulation is always higher than the predicted slope (Figs 2 and 3). The simulated mean phenotype cline is therefore closer to the environmental gradient than predicted. Such discrepancy is unlikely to be
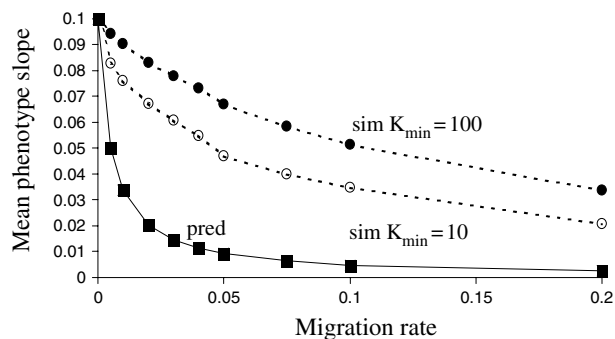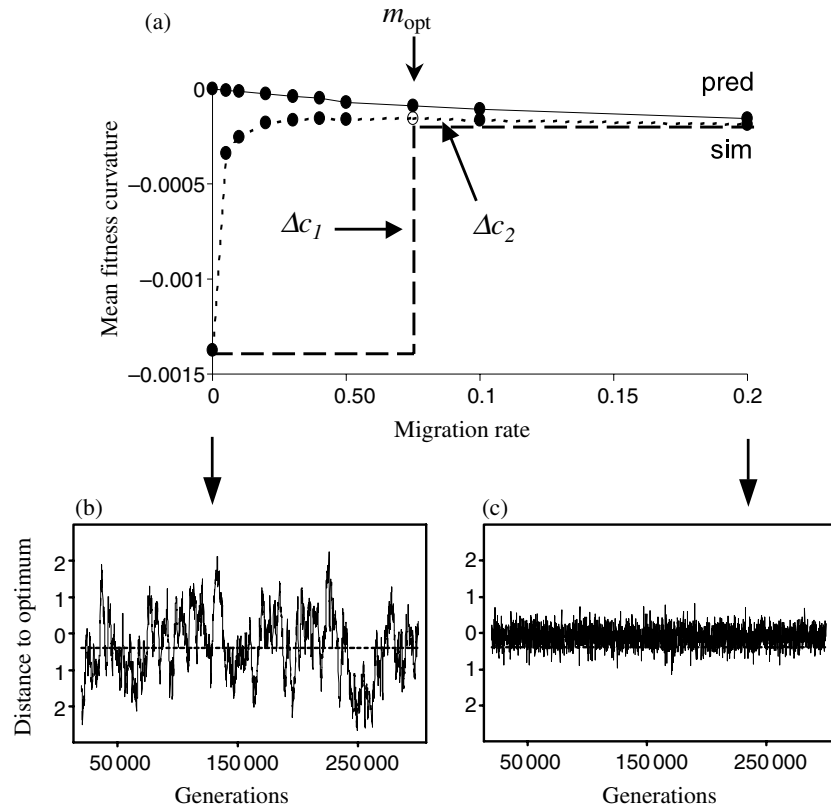

Fig. 3 Variation of the estimated and predicted slope of the cline for the mean phenotype with the migration rate. Estimated (circles, dotted line) and predicted (squares, solid line) slope of clines for mean phenotype as a function of the migration rate. Open and closed circles respectively for small ($K_{min} = 10$) and large ($K_{min} = 100$) peripheral populations. $b = 0.1$, $\mu = 0.01$, $d\mu = 10$.

explained by drift alone. Indeed drift is not a directional force and there is little reason why drift would systematically ameliorate local adaptation throughout the range. In fact, Slatkin & Maruyama (1975) predict that drift should decrease the value of the slope in a cline. Deviations from a normal distribution of phenotypes within populations, which may result in increased selection efficiency (see Ronce & Kirkpatrick, 2001) and the sharp edges of the simulated range, which decrease gene flow asymmetry within the range (see Garcia-Ramos & Kirkpatrick, 1997), might be responsible for the greater slope for the cline in the simulations than predicted by our analytical model. Such explanations are consistent with the fact that (i) both estimated and predicted slopes converge to the same value (corresponding to b, the slope in phenotypic optima) when the migration rate tends toward 0, (ii) the estimated slope is higher when peripheral populations are larger (see Fig. 3) and the distribution of density within the range more platykurtic.

Effect of migration on the curvature in mean fitness

Predicted curvature in mean fitness across space is negative and decreases with increasing migration rates (see for instance Fig. 4a). Such an effect is expected since the deviation between observed slope and environmental gradient [$(b_{sim} - b)^2$ in eqn 4] increases with increasing gene flow. The consequences of the indirect effect of migration on phenotypic variance for the evolution of predicted fitness clines are less apparent. The estimated curvature from the simulations seems to follow the same decreasing trend with increasing migration for most examined migration rates

Fig. 4 Variation of the estimated and predicted curvature of the clines for the mean fitness with the migration rate. (a) Estimated (dotted line) and predicted (solid line) curvature of clines for the mean fitness as a function of the migration rate. The simulated optimal migration rate is highlighted by the open circle pointed by an arrow (m_{opt} = 0.075). (b, c) For $m = 0$ (b) and $m = 0.2$ (c), the mean phenotype in one marginal population is plotted across time. The local optimum is shown by the dashed line ($\theta(i) = -0.4$). $K_{min} = 10$, $b = 0.1$, $\mu = 0.01$, d$\mu = 10$.

(Fig. 4a). Yet, for low to very low migration rates, the estimated curvature is in general more negative than predicted (Fig. 4a). As a result, the variation of the simulated curvature in mean fitness as a function of migration is nonmonotonic with a minimum absolute value in curvature for some low intermediate migration rates (shown by the arrow and open symbol in Fig. 4a), which the deterministic model does not predict. Such migration rates correspond to those that minimize the differences in fitness throughout the range.

We found that, in the simulations, for a given parameter set, the migration rate corresponding to the lowest absolute curvature in fitness through space not only minimized fitness heterogeneity throughout the range but also maximized the mean fitness at the range scale, whether or not local populations are weighted by their density (Fig. 5). This result can be partly explained by a relative insensitivity of the central population fitness to variation in migration rates (compare for instance Fig. 2c, d). This migration rate, both minimizing the fitness heterogeneity and maximizing the mean fitness, will further be called the optimal migration rate. The relevance and position of such intermediate optimal migration rates are discussed for different parameter sets further in the Result section (Table 1) and the discussion.

The discrepancy between theoretical predictions based on infinite population size and simulations can be due to two factors. First, genetic variance drops when approaching the edge of the range (results not shown, see also Butlin *et al.*, 2003), while the deterministic model assumes equal variance across the range and therefore overestimates the ability of such marginal population to respond to selection. Increasing migration rates might increase the genetic variance in such populations (see Effect of migration on phenotypic variance and distance to optimum section). Second, and more importantly, stochastic variation in mean phenotype due to drift may explain the poor fitness in marginal populations. Indeed, even though the averaged mean phenotype lies close to its optimal value at low migration rates, at any point of time, it might be far away from such value (Fig. 4b), which significantly decreases the averaged mean fitness when such stochastic fluctuations are taken into account. When the migration rate increases, such stochastic fluctuations in mean phenotype are dampened (Fig. 4c), while the average value of the mean phenotype gets further from the optimum because of gene swamping from central population (Figs 3 and 4c). The optimal migration rate corresponds to the best compromise between this reduction in temporal variability and increasing distance to the optimum.

(a)

(b)
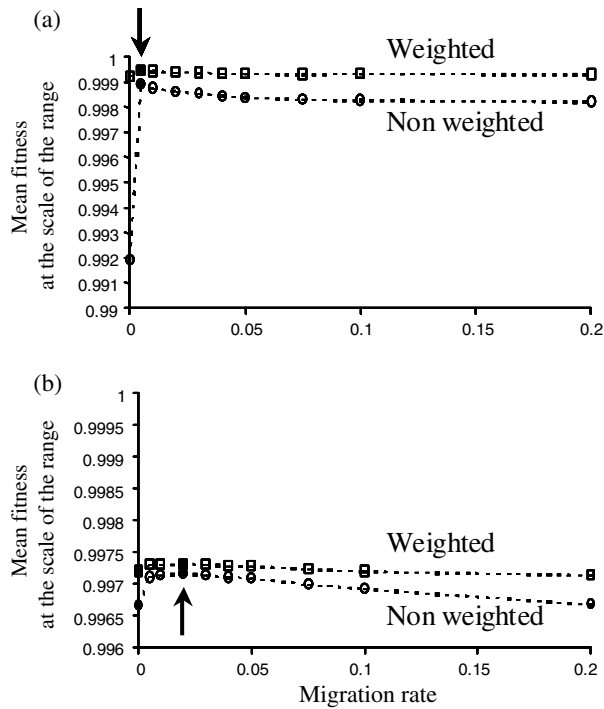
Fig. 5 Nonweighted and weighted mean fitness at the scale of the range as a function of migration rate for two parameter sets. Nonweighted (circles) and weighted (squares) mean fitness at the scale of the range corresponding to W_u and W_w (see *Analysis of simulations*). The arrow pointing a closed symbols highlights the migration rate minimizing the mean fitness function curvature at $m = 0.005$ for (a) $K_{min} = 10$, $b = 0.1$, $\mu = 0.001$, $d\mu = 5$ and $m = 0.02$ for (b) $K_{min} = 100$, $b = 0.1$, $\mu = 0.01$, $d\mu = 10$.

Effect of migration on phenotypic variance and distance to optimum

In order to give support to the proposed explanations for the existence of optimal intermediate migration rate, we have explored such situations in more details. Figs 6 shows the effect of migration rate on the average distance between mean phenotype and optimum at the margin, i.e. $E_t \left[\sqrt{(\bar{z}(x_{max}, t) - bx_{max})^2} \right]$ and average phenotypic variance at the margin, $\overline{\sigma}_z^2(x_{max})$. Contrary to $\overline{\overline{z}}(x_{max}) - bx_{max}$, which always increases in the margin as migration rate increases, the average distance of mean phenotype to the optimum varies nonmonotonically (Fig. 6a). The average distance between mean phenotype and optimum is minimal for some intermediate value of the migration rate, which is close to optimal migration rate minimizing the absolute value of the curvature in mean fitness. Phenotypic variance always increases at the margin as migration rate increases (at least for the range of explored migration rates, see Fig. 6b).

Table 1 Optimal migration rate, positive effect (Δc_1) and negative effect (Δc_2) of migration rate.

K_{min}	b	$d\mu$	μ	m_{opt}*	Δc_1[†]	Δc_2[†]
10	0.1	5	10^{-3}	0.005	17.8	1
10	0.1	5	10^{-2}	0.02	13.4	0.7
10	0.1	10	10^{-3}	0.02	18.5	0.7
10	0.1	10	10^{-2}	0.075	11.5	0.3
10	1	5	10^{-3}	0	0	83.7
10	1	5	10^{-2}	0	0	69.1
10	1	10	10^{-3}	0.005	1.4	69.4
10	1	10	10^{-2}	0.005	2.1	54.1
100	0.1	5	10^{-3}	0.01	1.06	1.46
100	0.1	5	10^{-2}	0.01	1.02	1.02
100	0.1	10	10^{-3}	0.01	1.26	1.36
100	0.1	10	10^{-2}	0.03	0.93	0.63
100	1	5	10^{-3}	0	0	78.7
100	1	5	10^{-2}	0	0	75
100	1	10	10^{-3}	0	0	78.8
100	1	10	10^{-2}	0	0	69.3

*The optimal migration rate is given with a precision related to the explored values of migration rate, i.e $m = 0$, 0.005, 0.01, 0.02, 0.03, 0.04, 0.05, 0.075, 0.1 and 0.2.
[†]Fitness curvatures were multiplied by -10^4 for convenient comparisons between Δc. See the Results section for the calculation of Δc_1 and Δc_2 (eqns 9 and 10).

Variation in optimal migration rates

We now explore how the value of the optimal migration rate varies with the parameters of the model (see Table 1). To better quantify the potential positive effects and negative effects of increasing gene flow, we define two new metrics, which summarize the variation in curvature in mean fitness as a function of migration such as in Fig. 4a. The first measures the positive effects of an initial increase in the migration rate, i.e. the decrease in the absolute value of curvature in mean fitness from a situation where local populations within the range are isolated to a situation corresponding to the optimal migration rate (see Fig. 4a):

$$\Delta c_1 = c_{sim}(m_{opt}) - c_{sim}(0), \qquad (9)$$

where m_{opt} is the optimal migration rate measured in our simulations for this parameter set. The second measures the negative effects of a further increase in migration and gene swamping, i.e. the increase in the absolute value of curvature in mean fitness from a situation where the migration rate is optimal to a situation where the migration rate is large (here arbitrarily fixed to 20%, see Fig. 4a):

$$\Delta c_2 = c_{sim}(m_{opt}) - c_{sim}(0.2). \qquad (10)$$

The optimal migration rate is null or very low when the environmental gradient, measured by b, is steep (Table 1). Positive effects of migration, measured by Δc_1, are then low or nonexistent. In such situations, the negative effects of gene flow, measured by Δc_2, are also

(a)

(b)
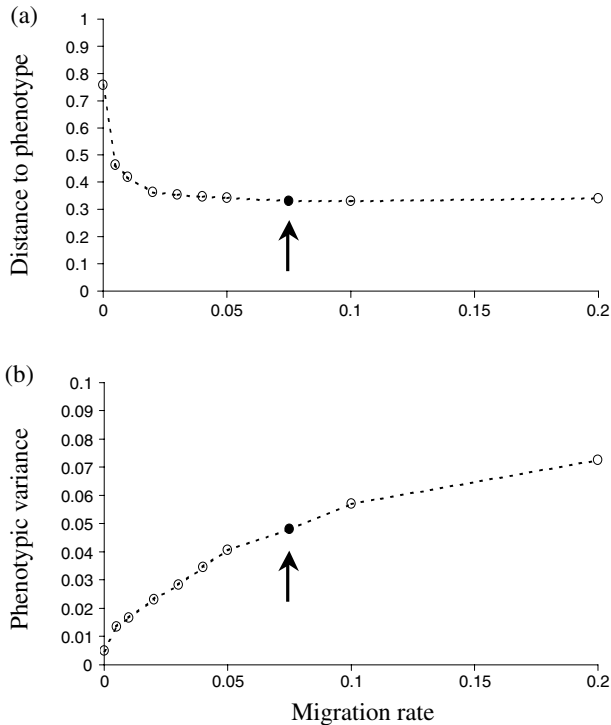
Fig. 6 Average distance between mean and optimal phenotypes and average phenotypic variance as a function of migration rate in a marginal population. (a) Average distance between the mean phenotype and the local phenotypic optima and (b) average phenotypic variance as a function of migration rate in a marginal population. The arrow pointing a closed circle highlights the optimal migration rate ($m_{opt} = 0.075$). $K_{min} = 10$, $b = 0.1$, $\mu = 0.01$, $d\mu = 10$.

the largest (Table 1). When the environmental gradient is shallower, the optimal migration rate increases when the size of peripheral populations decreases (Table 1). The positive effect of an initial increase in migration (Δc_1) is also larger when peripheral populations are smaller and drift is a potent force in such populations, while density at the margins has little effect on the negative effects of a further increase in migration (Δc_2). We also performed simulations with $K_{min} = 1000$ and observed that an optimal migration rate persists (but is smaller when compared to the ones detected for $K_{min} = 100$ and all the more for $K_{min} = 10$), while both Δc_1 and Δc_2 are close to 0 (results not shown).

Mutation is another process able to restore genetic variability lost by drift. Increasing the mutation rate or the size of the mutational step might have the same positive effect as increasing migration on the fitness of marginal populations. Contrary to this expectation, the optimal migration rate generally increases with both increasing mutation rate and increasing mutational steps (Table 1). A higher influx of migrants throughout the range seems then necessary to maximize adaptation at the scale of the species' range, which suggests that

migration and mutation do not play equivalent roles. However, both negative and positive effects of migration on the heterogeneity of fitness within the range (Δc_1 and Δc_2) are also smaller when the mutation rate is large (see Table 1).

Discussion

How does gene flow interact with drift at the scale of the species' range?

The small size of peripheral populations represents a double handicap: they are more sensitive to both genetic drift and gene swamping from larger more central populations. Both gene swamping and drift reduce genetic variability in such populations, impede their response to selection and can lead to the fixation of locally maladapted genotypes. Note, by now, some essential differences between the two processes. First, gene flow may result in the local fixation of a genotype that deviates from the local optimum, but this genotype was at least positively selected somewhere else in the range, while drift is a random process which may result in the local fixation of a genotype that may be deleterious everywhere in the range of the species. Second, gene flow has directional effects and will leave a consistent signature across generations in the local adaptation of peripheral populations as long as the environmental gradient does not vary too much in time. On the contrary, drift effects are not directional.

Contrary to predictions of deterministic models of species' range (Garcia-Ramos & Kirkpatrick, 1997; Kirkpatrick & Barton, 1997, see also the present analytical predictions), we found that, in presence of genetic drift, increasing gene flow may attenuate fitness heterogeneity within the range, resulting in higher total fitness. Positive effects of gene flow were detected when the environmental gradient was moderate, peripheral populations were small, mutation rates high and for very low migration rates among adjacent populations. Such an effect of migration on the evolution of mean fitness across the range could be described as a genetic rescue effect (Richards, 2000), by analogy with the demographic rescue effect, first described by Brown & Kodric-Brown (1977) (see also Hanski et al., 1996; cited in Richards, 2000). Similar genetic rescue effects have been documented both theoretically (Whitlock et al., 2000; Glémin et al., 2003) and empirically (Byers, 1998), in the context of homogeneous selection (Tallmon et al., 2004). What our model shows is that such rescue effects are expected even in the context of heterogeneous selection regimes (Robert et al., 2003) and at the scale of a species' range. Little exchange between populations could enhance natural selection and counteract drift, without preventing population differentiation (see experimental evidence by Newman & Tallmon, 2001). Optimal intermediate migration rates have also been predicted

by two habitat patches, source-sink models, incorporating demographic feedbacks between gene flow and selection (Gomulkiewicz *et al.*, 1999; Kawecki, 2000) and genetic drift (Gomulkiewicz *et al.*, 1999; Holt *et al.*, 2003). Models incorporating temporally varying selection, such as induced by co evolution with pathogens, have also predicted that increased migration rates could lead to higher level of local adaptation (Gandon *et al.*, 1996; Gandon & Michalakis, 2002).

What are the exact mechanisms of the genetic rescue effect in our model?

First, moderate levels of migration may help restore genetic variability eroded by drift. Migration affects the evolution of local genetic variance in heterogeneous environments even in the absence of drift (Haldane, 1948; Barton, 1999). With assumptions very similar to ours, Barton (2001) then found that the positive effect of migration on genetic variance more than counterbalanced its negative effects through gene swamping, allowing the species to always spread indefinitely in absence of physical barriers or other demographic constraints, contrary to the prediction made by Kirkpatrick & Barton (1997) of a limited range. Yet, Barton's (2001) model does not take drift into account. Using individual-based simulations incorporating drift among other stochastic effects, Butlin *et al.* (2003) found situations where gene swamping nonetheless caused the evolution of a limited range. This suggests that drift might constrain the effect of migration on the evolution of genetic variance. Our deterministic predictions take into account the effect of migration on the evolution of genetic variance in presence of drift, through the use of measured levels of variance in the stochastic simulations. Yet, despite a very good quantitative fit for the ranges explored for most parameters, our deterministic model fails to predict the 'genetic rescue effect' observed in the simulations for shallow environmental gradients. The average effect of migration on genetic variance therefore does not suffice to explain the positive effect of migration on fitness heterogeneity across the range. In this respect, the mechanisms of the genetic rescue effect of migration in our model differ from those involving temporally variable selection (Gandon & Michalakis, 2002) where the main role of migration is to replenish genetic variation eroded by selection.

Second, migration among local populations reduces stochastic variation of the mean phenotype the population around optima that are due to drift. A similar result has been obtained by models of interaction between selection, migration and drift along clines with constant density and no mutation (Hastings & Rohlf, 1974; Felsenstein, 1975; Slatkin & Maruyama, 1975). In that respect, migration might ameliorate the mean fitness in peripheral populations, just by dampening the local fluctuations of the mean phenotype. In our model, this dampening effect of migration might be accentuated by the density gradient between core and periphery of the range. Gene flow can modify the process of stochastic differentiation for small drifting populations by reducing the genetic distance between them (Gaggiotti, 1996). When the environmental gradient is not too steep, immigrants from bigger adjacent populations may be closer to the local optimum than residents in peripheral isolated populations experiencing stronger drift. Migration can thus help to purge deleterious mutations, which would be fixed by small isolated populations. Our simulations suggest that the optimal migration rate maximizing adaptation at the scale of the range is higher when the mutation rate is high (note however that we found nonnull optimal migration rates even for low mutation rate as low as $\mu = 10^{-5}$, results not shown) and the influx of deleterious mutation is of concern, which differs from results of models of local adaptation under temporally varying selection. In the latter, mutation and migration play the same role in replenishing genetic variance (Gandon & Michalakis, 2002).

Optimal levels of connectivity

The present model allows us to better specify the range of conditions for which increased migration improves mean fitness at the scale of a species' range. First a few percent of migration per generation among adjacent populations are sufficient to counteract the effect of drift in completely isolated margins. Higher levels of migration is generally deleterious as migration then introduces alleles selected further away in the core of the range. Second, the optimal migration rate generally decreases with (i) increasingly steep gradients, (ii) lower mutation rates and (iii) higher density at the margins. Finally, in the set of conditions where the optimal migration rate is larger, i.e. in presence of a shallow environmental gradient, the variation of mean fitness across the range is moderate in any case, so the improvement of mean fitness through changes in connectivity might be minute. For a large range of parameters, the predictions of our deterministic model performed in general very well when compared to the averaged outputs of our stochastic individual-based model. In such situations, stochasticity affects very little the evolution of adaptation within the range and that the main effect of migration on patterns of adaptation is increased maladaptation due to gene swamping.

Critical assumptions

Models having explored the purging effect of gene flow in presence of drift (see for instance Couvet, 2002) have generally focused on the accumulation of unconditionally deleterious mutations, whose effects do not depend on the local environment. Other models (see for instance

Kawecki, 2000) investigating the effect of gene swamping at a local adaptation locus have often excluded the occurrence of mutations with deleterious effects in every environment. Our simulation model shows that both phenomena may affect the evolution of allelic frequencies at the same local adaptation locus. It would be interesting to investigate further the optimal levels of connectivity when some loci harbour mutations with environment-specific effects and some do not. More generally, a more thorough examination of the robustness of our predictions with respect to details of the genetic architecture of fitness would be necessary, though out of the scope of the present paper.

Both increased migration and mutation load at the margins are the direct consequences of the smaller size of peripheral populations. After decades of predominance of the 'abundant centre' rule (Brown, 1984; Lawton, 1993), some authors have questioned the generality of this bio geographical pattern, suggesting more asymmetrical distribution of density across the range (Sagarin & Gaines, 2002a,b). Yet, even such patterns of distribution will generate asymmetrical gene flow throughout the range and lead to stronger drift in the sparser margins, so that we expect our prediction about optimal intermediate migration rate still to hold. By assuming a stepping-stone model for dispersal within the range, we forbid very long-distance migration events in our simulations, which would tend to worsen the gene swamping effects and attenuate the beneficial effects of migration. Such an assumption might however be reasonable when studying movements at the scale of the species' range since migration between adjacent populations may already involve considerable distances and long-distance events are rare (Cain et al., 2000). Genetic variation for migration propensities has been documented in many species (Roff & Fairbairn, 2001) and the migration rate within a species' range is likely to be an evolving trait as well. The present model does not allow making predictions about the selected migration rates at the scale of the range, which would constitute an interesting perspective for the present work (for a model of selection on a migration modifier in presence of local adaptation in a two patches system, see Billiard & Lenormand, 2005). Evolutionary stable migration rates favoured by natural selection may often depart strongly from the optimal migration rates maximizing mean fitness or total population size (see Olivieri & Gouyon, 1997; Billiard & Lenormand, 2005).

By assuming fixed population sizes, the present model does not allow the consideration all potential effects of migration on adaptation within a species' range. Migration may also act on the intensity of drift by affecting local population size (see for instance Gomulkiewicz et al., 1999; Holt et al., 2003). Demographic stochasticity and Allee effects in small peripheral populations are likely to be critically important for the interplay between gene flow and local adaptation, as suggested

by recent theoretical studies (Butlin et al., 2003; see also Holt et al., 2004). More generally, predicting optimal levels of connectivity at the scale of the species' range would imply to integrate both genetic and demographic consequences of migration in a single theoretical framework. By ignoring feedbacks between the evolution of local adaptation and the evolution of density distribution (contrarily to Kirkpatrick & Barton, 1997), the present model does not allow predictions about how interactions between gene flow and drift might shape the extent of the species' range (see Butlin et al., 2003). Our predictions therefore apply more readily to those species which range is constrained by physical barriers or interspecific interactions (Case & Taper, 2000), rather than by the only interplay of natural selection, gene flow and drift.

Implications for conservation biology

Small peripheral populations are both very vulnerable and unique (Vucetich & Waite, 2003), as they occur in relatively extreme environmental conditions and may shelter unique adaptations. The fate of such marginal populations is also very important in the context of climatic change as they might be the theatre of evolutionary change, face increasing extinction risk, or be the source of migrants to colonize new areas at expanding margins. It is therefore critical to identify the different sources of maladaptation in such populations, to propose efficient conservation measures. Depending on whether drift or gene flow is mainly responsible for local maladaptation, recommendations should be completely opposite, i.e. improve or reduce connectivity between populations. However, our model also suggests that it might be empirically difficult to diagnose the exact origin of the low adequacy of populations with the environmental conditions. In both discussed cases, we expect small genetic variance and low fitness in peripheral populations. Spatial and temporal patterns in mean phenotypes across the range might be more informative about the causes of maladaptation, even though such information might be costly to collect. Drift would generate a strong spatial and temporal variance for the mean phenotypes, with increasing variability further from the core of the range, while gene swamping would result in phenotypic homogeneity across the range and less variation through time. A safer strategy in this context may be to improve, if feasible, population density at the margins, as increased population size may help mitigate both gene swamping and drift.

Acknowledgments

References

Arnegard, M.E., Markert, J.A., Danley, P.D., Stauffer, J.R., Ambali, A.J. & Kocher, T.D. 1999. Population structure and colour variation of the cichlid fish *Labeotropheus fuelleborni Ahl* along a recently formed archipelago of rocky habitat patches in southern Lake Malawi. *Proc. Roy. Soc. Lond. B Bio.* **266**: 119–130.

Barton, N.H. 1999. Clines in polygenic traits. *Genet. Res.* **74**: 223–236.

Barton, N.H. 2001. Adaptation at the edge of a species' range. In: *Integrating Ecology And Evolution In A Spatial Context* (J. Silvertown & J. Antonovics, eds), pp. 365–392. Blackwell Science, New York.

Billiard, S. & Lenormand, T. 2005. Evolution of migration under kin selection and local adaptation. *Evolution*, in press.

Brewer, A.M. & Gaston, K.J. 2002. The geographical range structure of the holly leaf-miner. 1. Population density. *J. Anim. Ecol.* **71**: 99–111.

Brown, J.H. 1984. On the relationship between abundance and distribution of species. *Am. Nat.* **124**: 255–279.

Brown, J.H. & Kodric-Brown, A. 1977. Turnover rates in insular biogeography – effect of immigration on extinction. *Ecology* **58**: 445–449.

Bürger, R. & Lynch, M. 1995. Evolution and extinction in a changing environment: a quantitative-genetic analysis. *Evolution* **49**: 151–163.

Bürger, R. & Gimelfarb, A. 1999. Genetic variation maintained in multilocus models of additive quantitative traits under stabilizing selection. *Genetics* **152**: 807–820.

Butlin, R.K., Bridle, J.R. & Kawata, M. 2003. Genetics and the boundaries of species' distributions. In: *Macroecology: Concepts and Consequences* (T. Blackburn & K. J. Gaston, eds), pp. 274–295. Blackwell Science, Oxford.

Byers, D.L. 1998. Effect of cross proximity on progeny fitness in a rare and a common species of *Eupatorium* (*Asteraceae*). *Am. J. Bot.* **85**: 644–653.

Cain, M.L., Milligan, B.G. & Strand, A.E. 2000. Long-distance seed dispersal in plant populations. *Am. J. Bot.* **87**: 1217–1227.

Case, T.J. & Taper, M.L. 2000. Interspecific competition, environmental gradients, gene flow, and the coevolution of species' borders. *Am. Nat.* **155**: 583–605.

Channell, R. & Lomolino, M.V. 2000. Dynamic biogeography and conservation of endangered species. *Nature* **403**: 84–86.

Cote, S.D., Dallas, J.F., Marshall, F., Irvine, R.J., Langvatn, R. & Albon, S.D. 2002. Microsatellite DNA evidence for genetic drift and philopatry in Svalbard reindeer. *Mol. Ecol.* **11**: 1923–1930.

Couvet, D. 2002. Deleterious effects of restricted gene flow in fragmented populations. *Conserv. Biol.* **16**: 369–376.

Ehrlich, P.R. & Raven, P.H. 1969. Differentiation of Populations. *Science* **165**: 1228–1232.

Felsenstein, J. 1975. Genetic drift in clines which are maintained by migration and natural selection. *Genetics* **81**: 191–207.

Felsenstein, J. 1977. Multivariate normal genetic model with a finite number of loci. In: *Proceedings of The International Conference On Quantitative Genetics* (E. Pollak, O. Kempthorne & T. B. J. Bailey, eds), pp. 227–246. Iowa University Press, Ames.

Gaggiotti, O.E. 1996. Population genetic models of source-sink metapopulations. *Theor. Pop. Biol.* **5**: 178–208.

Gandon, S., Capowiez, Y., Dubois, Y., Michalakis, Y. & Olivieri, I. 1996. Local adaptation and gene-for-gene coevolution in a metapopulation model. Proc. *Roy. Soc. Lond. B Bio.* **263**: 1003–1009.

Gandon, S. & Michalakis, Y. 2002. Local adaptation, evolutionary potential and host-parasite coevolution: interactions between migration, mutation, population size and generation time. *J. Evol. Biol.* **15**: 451–462.

Garcia-Ramos, G. & Kirkpatrick, M. 1997. Genetic models of adaptation and gene flow in peripheral populations. *Evolution* **51**: 21–28.

Gaston, K.J. 1996. Species-range-size distributions: patterns, mechanisms and implications. *Trends Ecol. Evol.* **11**: 197–201.

Glémin, S., Ronfort, J. & Bataillon, T. 2003. Patterns of inbreeding depression and architecture of the load in subdivided populations. *Genetics* **165**: 2193–2212.

Gomulkiewicz, R., Holt, R.D. & Barfield, M. 1999. The effects of density dependence and immigration on local adaptation and niche evolution in a black hole sink environment. *Theor. Pop. Biol.* **55**: 283–296.

Haldane, J.B.S. 1948. The theory of a cline. *J. Genet.* **48**: 277–284.

Hanski, I., Moilanen, A. & Gyllenberg, M. 1996. Minimum viable metapopulation size. *Am. Nat.* **147**: 527–541.

Hastings, A. & Rohlf, F.J. 1974. Gene flow – effect in stochastic models of differentiation. *Am. Nat.* **108**: 701–705.

Holt, R.D., Gomulkiewicz, R. & Barfield, M. 2003. The phenomology of niche evolution via quantitave traits in a 'black-hole' sink. *Proc. Roy. Soc. Lond. B Bio.* **270**: 215–224.

Holt, R.D., Knight, T.M. & Barfield, M. 2004. Allee effects, immigration, and the evolution of species' niches. *Am. Nat.* **163**: 253.

Karan, D., Dahiya, N., Munjal, A.K., Gibert, P., Moreteau, B., Parkash, R. & David, J.R. 1998. Desiccation and starvation tolerance of adult *Drosophila*: opposite latitudinal clines in natural populations of three different species. *Evolution* **52**: 825–831.

Kawecki, T.J. 2000. Adaptation to marginal habitats: contrasting influence of dispersal rate on the fate of alleles with small and large effects. *Proc. Roy. Soc. Lond. B Bio.* **267**: 1315–1320.

Kirkpatrick, M. & Barton, N.H. 1997. Evolution of a species' range. *Am. Nat.* **150**: 1–23.

Lawton, J.H. 1993. Range, Population Abundance and Conservation. *Trends Ecol. Evol.* **8**: 409–413.

Lenormand, T. 2002. Gene flow and the limits to natural selection. *Trends Ecol. Evol.* **17**: 183–189.

Lonn, M. & Prentice, H.C. 2002. Gene diversity and demographic turnover in central and peripheral populations of the perennial herb *Gypsophila fastigiata*. *Oikos* **99**: 489–498.

Magiafoglou, A., Carew, M.E. & Hoffmann, A.A. 2002. Shifting clinal patterns and microsatellite variation in *Drosophila serrata* populations: a comparison of populations near the southern border of the species' range. *J. Evol. Ecol.* **15**: 763–774.

Mayr, E. 1963. *Animal Species and Evolution*. Belknap Press, Harvard, Cambridge.

Nagylaki, T. 1978. Random genetic drift in a cline. *Proc. Natl. Acad. Sci. USA* **75**: 423–426.

Newman, D. & Tallmon, D.A. 2001. Experimental evidence for beneficial fitness effects of gene flow in recently isolated populations. *Conserv. Biol.* **15**: 1054–1063.

Obrien, E.M. 1993. Climatic gradients in woody plant-species richness – Towards an explanation based on an analysis of Southern Africa woody flora. *J. Biogeogr.* **20**: 181–198.

Olivieri, I. & Gouyon, P.-H. 1997. Evolution of the migration rate and other traits: the metapopulation effect. In: *Metapopulation Biology* (I. Hanski & M. E. Gilpin, eds), pp. 293–323. Academic Press, San Diego, London.

Pease, C.M., Lande, R. & Bull, J.J. 1989. A model of population-growth, dispersal and evolution in a changing environment. *Ecology* **70**: 1657–1664.

Rehfeldt, G.E., Ying, C.C., Spittlehouse, D.L. & Hamilton, D.A. 1999. Genetic responses to climate in *Pinus contorta*: niche breadth, climate change, and reforestation. *Ecol. Monogr.* **69**: 375–407.

Richards, C.M. 2000. Inbreeding depression and genetic rescue in a plant metapopulation. *Am. Nat.* **155**: 383–394.

Robert, A., Couvet, D. & Sarrazin, F. 2003. Bottlenecks in large populations: the effect of immigration on population viability. *Evol. Ecol.* **17**: 213–231.

Roff, D.A. & Fairbairn, D.A. 2001. The genetic basis of dispersal and migration, and its consequences for the evolution of correlated traits. In: *Dispersal* (J. Clobert, E. Danchin, A. A. Dhondt & J. D. Nichols, eds), pp. 191–202. Oxford University Press, New York.

Ronce, O. & Kirkpatrick, M. 2001. When sources become sinks: migrational meltdown in heterogeneous habitats. *Evolution* **55**: 1520–1531.

Sagarin, R.D. & Gaines, S.D. 2002a. The 'abundant centre' distribution: to what extent is it a biogeographical rule? *Ecol. Lett.* **5**: 137–147.

Sagarin, R.D. & Gaines, S.D. 2002b. Geographical abundance distributions of coastal invertebrates: using one-dimensional ranges to test biogeographic hypotheses. *J. Biogeogr.* **29**: 985–997.

Sgro, C.M. & Blows, M.W. 2003. Evolution of additive and nonadditive genetic variance in development time along a cline in *Drosophila serrata*. *Evolution* **57**: 1846–1851.

Slatkin, M. 1978. Spatial patterns in distributions of polygenic characters. *J. Theor. Biol.* **70**: 213–228.

Slatkin, M. & Maruyama, T. 1975. Genetic drift in a cline. *Genetics* **81**: 209–222.

Tallmon, D.A., Luikart, G. & Waples, R.S. 2004. The alluring simplicity and complex reality of genetic rescue. *Trends Ecol. Evol.* **19**: 489–496.

Vucetich, J.A. & Waite, T.A. 2003. Spatial patterns of demography and genetic processes across the species' range: null hypotheses for landscape conservation genetics. *Conserv. Genet.* **4**: 639–645.

Whitlock, M.C., Ingvarsson, P.K. & Hatfield, T. 2000. Local drift load and the heterosis of interconnected populations. *Heredity* **84**: 452–457.

Zeng, Z.B. & Cockerham, C.C. 1993. Mutation models and quantitative genetic-variation. *Genetics* **133**: 729–736.