The Relation of Density Regulation to Habitat Specialization, Evolution of a Species' Range, and the Dynamics of Biological Invasions

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ABSTRACT: Prior studies of the evolution of species' niches and ranges have identified the importance of within-population genetic variance, migration rate, and environmental heterogeneity in determining evolutionarily stable patterns of species' range and habitat use. Different combinations of these variables can produce either habitat specialists or generalists and cause either stable range limits or unbounded expansion. We examine the effect of density regulation on a species' range and habitat use within a landscape comprised of two discrete habitats and along continuous environmental gradients. Using the theta-logistic formulation, we demonstrate the following. (1) Spatially uniform density regulation generally weakens gene swamping and opposes habitat specialization and range limitation. (2) The form of density regulation should play an important role in determining whether the equilibrium species' range is limited by gene flow. (3) Even when no long-term limited-range equilibrium occurs, quasi-stable (or even contracting) range limits may be maintained for a long period during the initial phases of an invasion; the length of this period depends on the form of density regulation. (4) The steady state invasion speed in heterogeneous environments depends on the form of density regulation. Implications for the study of biological invasions are discussed, and directions for further exploration are sketched.

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Species' borders are a unifying theme in ecology (Holt and Keitt 2005; Antonovics et al. 2006). The study of range limits is strongly related to the study of other important ecological and evolutionary phenomena, such as niche conservatism, local adaptation, and biological invasions (e.g., Holt et al. 2005*a*; Wiens and Graham 2005; Bridle and Vines 2007). Species' range limits emerge from the interplay of many processes, both demographic and evolutionary, as well as history and chance events. Analyzing this interplay is central to understanding the many regularities observed in the study of species' ranges (e.g., Gaston 2003) and biological invasions (e.g., Shigesada and Kawasaki 1997).

A theoretical framework integrating evolution and ecology in the study of both range limits and invasions has been gradually developing over the past decade (see Holt and Gomulkiewicz 1997*a*; Kirkpatrick and Barton 1997; Gomulkiewicz et al. 1999; Case and Taper 2000; Holt and Keitt 2000; Barton 2001; Keitt et al. 2001; García-Ramos and Rodriguez 2002; Holt 2003; Peck and Welch 2004; Case et al. 2005; Holt et al. 2005*a*, 2005*b*; Travis et al. 2005). This framework stresses the interaction of ecological (demographic) processes with evolutionary processes in generating stable and quasi-stable (i.e., slowly changing), as well as rapidly changing, range limits. One such important (demographic) process is density-dependent population regulation. Several authors have suggested that regularities exist in the pattern of density dependence across species' ranges. Gaston (2003) reviewed several studies suggesting "that towards the edges of geographic ranges local populations … experience less density-dependent regulation" (Gaston 2003, p. 54). According to this hypothesis, density-independent processes (e.g., mortality due to abiotic stress) control the dynamics of peripheral populations. Maurer and Taper (2002), by contrast, provide examples in which density dependence (i.e., the mar-

ginal effect on per capita growth rates of a small change in density) is stronger near range edges.

Analyses of range limits and of biological invasions are closely related; a range limit occurs wherever an invasion stops, that is, where invasion speed drops to 0. The distinction between density-dependent dynamics at the center of the geographic distribution and density independence at the periphery is implicit in models of invasion dynamics (e.g., Holmes et al. 1994; Shigesada and Kawasaki 1997; Hastings et al. 2005). In these models, a species' invasion speed is determined solely by the (density-independent) dynamics at the periphery (i.e., at the invasion wave front). For purely negative density dependence, maximum per capita growth occurs at low densities, and populations at the invasion front are source populations with positive growth. Thus, invasion speed depends only on the maximum per capita growth rate and not on the specific form of density dependence.

By contrast, when an Allee effect is present, invasion speed may depend on the specifics of density dependence (e.g., Lewis and Kareiva 1993). The Allee effect depresses per capita growth at low densities and may cause populations at an invasion front to have a negative growth rate. The growth and advance of the invasion front then depend on immigration from higher-density populations within the species' range, where density dependence can significantly affect population growth, the flux of immigrants, and, therefore, indirectly, invasion speed (Lewis and Kareiva 1993). As we show in this article, source-sink dynamics also arise when peripheral populations are maladapted to their newly invaded environment, and so invasion dynamics can depend on the form of density dependence, even for purely negative density regulation.

Both theoretical and empirical work have shown that evolutionary processes can play an important role in invasion and range dynamics (e.g., Lee 2002; Lambrinos 2004; Holt et al. 2005*a* and references therein; Urban et al. 2007). During invasions, species may encounter novel and spatially changing environments, leading to selection for local adaptation. García-Ramos and Rodriguez (2002) studied invasion dynamics along an environmental gradient and showed that the expanding wave front of population density is followed by a similarly expanding wave front of local adaptation to the newly invaded environments. Invasion speed monotonically decreased as the gradient became steeper, until at some steepness threshold the invasion was stopped, and stable range limits arose. This invasion model builds on earlier work by Kirkpatrick and Barton (1997) on the evolution of a species' range (see also García-Ramos and Kirkpatrick 1997; Case and Taper 2000; Barton 2001; Case et al. 2005). Kirkpatrick and Barton (1997) explored how stable range limits may arise through the feedback between demography (population growth and dispersal) and evolution (adaptation and gene swamping). This feedback is at the heart of many theoretical studies on the evolution of species' ranges and niches (e.g., Pease et al. 1989; Holt and Gaines 1992; Holt and Gomulkiewicz 1997*b*; Barton 1999; Gomulkiewicz et al. 1999; Barton 2001; Holt et al. 2003, 2004*b*). An important point of contact between demography and evolution is through the process of gene swamping (e.g., Lenormand 2002). Asymmetric gene flow from high- to low-density populations can oppose local adaptation to peripheral environments and cause stable range limits (e.g., Kirkpatrick and Barton 1997; Barton 2001). The strength of gene swamping should increase as the spatial gradient in population density becomes steeper. Density dependence can influence this gradient and thus modulate the swamping effect of gene flow.

Our study focuses on how the form of density dependence influences niche conservatism and the dynamics of species' ranges and invasions. Although most of the studies cited above assumed some form of density regulation, the consequences of density dependence for niche and range evolution and for invasion dynamics have not yet been systematically explored. We first consider how density dependence influences the evolution of habitat specialization and generalization in a landscape comprised of two discrete habitat patches. We then turn to range edges along a continuous environmental gradient. The form of density regulation has major effects, qualitative as well as quantitative, on both the equilibrium outcome of range evolution and transient invasion dynamics. We show as well that some forms of density regulation may cause a phase of range contraction before range expansion.

The Evolution of Species' Ranges in Two-Habitat Landscapes

The geometry of species' ranges can be highly complex, exhibiting a patchwork of occupied areas and gaps, multiple abundance peaks, and ragged and at times ill-defined edges (e.g., Brown et al. 1995; Sagarin and Gaines 2002; Fortin et al. 2005). But it is useful in theoretical studies to first consider much simpler landscapes so as to begin to tease apart the action of different ecological and evolutionary forces acting on species' distributions. We start by examining the evolution of a species with a potential range comprised of two distinct habitats. The interplay of density dependence, dispersal, and selection can influence whether the species is a generalist, occupying both habitats in high numbers, or instead is a specialist, largely restricted to just one habitat.

Prior theoretical studies have examined how the reciprocal effects of demography, including density dependence, and local adaptation can mold the distribution of a species

in a two-habitat landscape (Holt 1996, 2003; Kawecki 2000; Ronce and Kirkpatrick 2001; Kawecki and Holt 2002; Kisdi 2002). Holt (1996), for instance, took an evolutionary game approach to examine adaptive evolution. He argued that increasing the strength of density dependence in a sink habitat (while leaving the source unchanged) should make specialization to the source more likely, because natural selection can be biased toward habitats with higher fitness, simply because more individuals occur there, and they have a higher reproductive value. Increasing negative density dependence in the sink magnifies this demographic asymmetry (see also Gomulkiewicz et al. 1999).

Ronce and Kirkpatrick (2001) considered a complementary model of the evolution of a quantitative trait determining fitness in a randomly mating species exposed to each of two habitats coupled by migration, with logistic density dependence in each. If a species (a specialist) is initially adapted to one habitat and maladapted to the other, random mating between immigrants and residents in the suboptimal habitat can lead to gene flow overwhelming selection there, thus preventing adaptation. Moreover, a species initially adapted to both habitats (a generalist) might be vulnerable to loss of adaptation in one habitat, so that the generalist contracts its range and becomes a specialist—a phenomenon Ronce and Kirkpatrick call "migrational meltdown." The interplay of gene flow, demography, and selection can readily lead to such alternative evolutionary states in heterogeneous landscapes (Kirkpatrick and Barton 1997; Holt 2003; Holt et al. 2004*b*).

Generalized Ronce-Kirkpatrick Model

We follow the notation of Ronce and Kirkpatrick (2001) and refer the reader to that article for the detailed derivation of the model, which we modify by generalizing the form of density dependence. There are two habitats (indexed by $i = 1, 2$) of equal area; for each, we track population density n_i and the mean phenotype $\bar{z}_i$. The fitness of an individual of phenotype z is given by the sum of a density-dependent growth term and a term due to stabilizing selection toward a habitat-specific phenotypic optimum (for mathematical expressions and derivations, see the appendix in the online edition of the *American Naturalist*). It is assumed that the intrinsic growth rate, carrying capacity, and strength of stabilizing selection are the same in both habitats and that phenotypes are normally distributed in each habitat with means $\bar{z}_i$. Phenotypic and genetic variances are fixed parameters, independent of habitat. The per capita rate of migration between habitats is m (assumed to be symmetrical; i.e., no habitat selection).

After rescaling into dimensionless variables (see appendix), the equation for trait evolution in habitat i is

$$\frac{dY_i}{dT} = -\Gamma Y_i + M\frac{N_j}{N_i}(H - Y_i - Y_j), \tag{1}$$

where j is the habitat opposite to i, Γ is the standardized intensity of selection (proportional to the genetic variance), H represents the difference in phenotypic optima between the habitats (a measure of environmental heterogeneity), and M is the rescaled migration parameter (for details, see appendix). The quantity Y_i in equation (1) is the maladaptation in habitat i, that is, the difference between the mean phenotype and the local optimum (divided by the genetic standard deviation; see appendix), and N_i is the normalized population density. The first term in equation (1) describes how natural selection acts on the trait in habitat i. In the absence of migration, the phenotype in each habitat should equilibrate at its respective local optimum ($Y_i = 0$). The second term describes how movement with random mating induces a migrational load because of trait admixture due to mating between immigrants and residents. The maladaptive influence of migration is greater in the habitat with lower abundance.

The equation for change in population size in habitat i is

$$\frac{dN_i}{dT} = U(N_i)N_i - \frac{\Gamma}{2}Y_i^2N_i + M(N_j - N_i). \tag{2}$$

The third term in equation (2) describes how migration changes local population size. There is a net flow of individuals from high- to low-density habitats, which in the absence of countervailing forces would equalize densities in both habitats. The second term in equation (2) is the demographic load in habitat i due to maladaptation, that is, the depression in local growth because of the difference between the mean trait value and its optimum. Finally, the first term in equation (2) is population growth including local density dependence $U(N_i)$, which for most results below is assumed to be theta-logistic, given by $U(N) = 1 - N^\theta$ (Gilpin and Ayala 1973; Gilpin and Case 1976; Diserud and Engen 2000; Sæther and Engen 2002; Sæther et al. 2002). The theta-logistic model is flexible and describes phenomenologically many different forms of density dependence, depending on the value of θ (linear for $\theta = 1$; convex for $0 < \theta < 1$; concave for $\theta > 1$). There are empirical estimates of θ in many natural populations (see "Discussion").

Ronce and Kirkpatrick (2001) used logistic density regulation ($U(N) = 1 - N$; $\theta = 1$). However, different mechanisms can lead to different forms of density depen-

dence. For instance, contest competition for territories may occur only at high abundances, so density dependence is weak at low N and increases at high N ($\theta > 1$). By contrast, in scramble competition for quickly depleted resources, density dependence can be strong at low density and weaker near carrying capacity (so $\theta < 1$). We examine how the behavior of model equations (1) and (2) is altered by different forms of density dependence, assuming the same value of θ in both habitats. Density dependence does not directly enter into equation (1), so it does not directly alter how selection drives trait evolution. Instead, nonlinearities in density dependence affect population density (eq. [2]), which in turn indirectly affects the strength of gene flow acting against selection in equation (1). Because the force of gene flow is determined by the fraction of individuals comprised of immigrants each generation, any factor changing local population size can modify the strength of gene flow.

The above model can have a symmetric equilibrium (representing a generalist) for which population size and degree of maladaptation are spatially uniform at

$$Y_1^* = Y_2^* \equiv Y^* = \frac{HM}{2M + \Gamma}, \tag{3a}$$

$$N_1^* = N_2^* \equiv N^* = U^{-1}\left(\frac{\Gamma}{2}Y^{*2}\right) = \left(1 - \frac{\Gamma}{2}Y^{*2}\right)^{1/\theta} \tag{3b}$$

(for the theta-logistic), where U^{-1} indicates the inverse of function U. As Ronce and Kirkpatrick (2001) note, the uniform generalist population is unviable (i.e., $N^* < 0$) if the difference in habitat optima is too large; by adapting equally to both habitats, the species may be too poorly adapted to persist. A necessary condition for demographic persistence is that there is a positive N for which $U(N^*) = \Gamma Y^{*2}/2$. For the theta-logistic form, this reduces to $H < 2^{1/2}(2M + \Gamma)/(M\Gamma^{1/2})$ (the same as in Ronce and Kirkpatrick 2001), which is independent of θ (as the threshold H is approached, N^* approaches 0, so density dependence becomes negligible). Therefore, nonlinear density dependence does not affect the existence of a symmetric equilibrium. It does, however, affect the condition for local stability of this equilibrium. Using standard local stability analysis (as in app. 2 in Ronce and Kirkpatrick 2001), the symmetric equilibrium can be shown to be stable if and only if $M > 2U(N^*) + (N^*/2)dU(N)/dN|_{N=N^*}$, where N^* is the general solution in equation (3b). All else being equal, increasing the strength of density dependence at the equilibrium reduces the right-hand side and so enhances the stability of the symmetric equilibrium. For the theta-logistic, this stability condition reduces to $H < [2(2M + \theta)]^{1/2}(2M + \Gamma)/\{M[\Gamma(4 + \theta)]^{1/2}\}$ (fig. 1). If the symmetric equilibrium exists but is not stable, the system will go to an asymmetric equilibrium, in which the population is specialized to one of the habitats. Thus, evolutionarily stable generalization is easier with higher θ (in fig. 1, note that symmetric equilibria are stable below the solid lines).

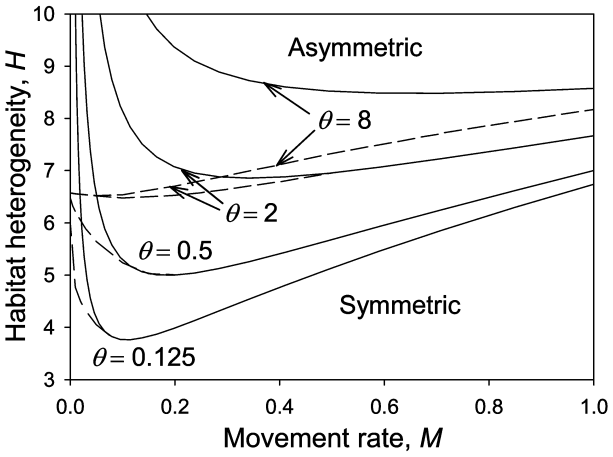

Figure 1: Stability of equilibria as influenced by density dependence at low numbers. At symmetric equilibria, the species is a generalist, adapted equally to both habitats. At asymmetric equilibria, the species is a specialist, well adapted (and near carrying capacity) in one habitat and poorly adapted (and at low abundance) in the other. The solid lines are the maximal degree of habitat heterogeneity for which the symmetric equilibria are locally stable in the two-habitat model for four different values of θ. Below the solid lines, the symmetric equilibria are locally stable, and above them, they are unstable. The dashed line separates stability domains for asymmetric equilibria. Above the dashed line (which, note, converges on the solid line as M increases), the asymmetric equilibria are locally stable, whereas below the dashed line, they are unstable. Between the solid and dashed lines for a given θ, both kinds of equilibria are locally stable, so the system can exhibit alternative stable states. The figure shows that making density dependence stronger at lower abundances (decreasing θ) increases the range of parameter space over which the asymmetric equilibria alone are stable. The intensity of selection $\Gamma = 0.1$.

Density dependence strongly affects the conditions for an asymmetric equilibrium in which the species is a specialist largely adapted to one of the two habitats. Because no analytical solution was possible, we identified asymmetric equilibria numerically by setting the derivatives in equations (1) and (2) to 0 and solving. Local stability of these equilibria was then determined by simulating the system (and evaluating the eigenvalues numerically). (An approximate analytical solution is possible assuming small M, as in Ronce and Kirkpatrick 2001. Including theta-logistic density dependence has very little effect on this limiting case.) The minimum difference in the phenotypic optima (H) required for asymmetric equilibria increases with higher θ, making specialization harder (in fig. 1, below

the dashed line for a given θ, a system that starts at a specialized asymmetric equilibrium [if it exists], if perturbed, will evolve toward habitat generalization). For sufficiently large M, the two boundaries coincide, so a species will always evolve to be either a generalist or a specialist, regardless of initial conditions; whichever one occurs depends on H. At low M, the asymmetric boundary is lower, which allows alternative stable equilibria (i.e., a species could end up either a generalist or a specialist to either habitat) for parameter combinations between the two boundaries (this area is larger for higher θ; fig. 1).

Figure 2 displays examples of symmetric and asymmetric equilibria in local abundances and trait values as a function of the movement rate M for different values of θ. Consider figures 2*A* and 2*B*, which show the equilibria for various values of M, when $H = 7$ (a fairly strong degree of spatial heterogeneity in the adaptive optimum) and $\theta = 8$ (density dependence is weak until density nears carrying capacity). In this example, above a value of M of around 0.34, only a symmetric equilibrium exists, with equal numbers in both habitats and equal degrees of maladaptation. Movement slightly depresses population size below that observed in an isolated, adapted population ($M = 0$). Below $M = 0.34$, there are in addition equilibria with one habitat well adapted and at high density (e.g., denoted by the X) and the other maladapted and at low density (denoted by the asterisk). These equilibria can either be stable (*solid lines*) or unstable (*dashed lines*). In this region, a species that starts out specialized to one habitat will likely stay specialized, and a generalist is likely to remain a generalist (unless there is a substantial perturbation). For asymmetric equilibria, the maladaptation in one habitat is high and approximately independent of M. The existence of maladaptation is determined by gene flow, but the magnitude of such maladaptation at equilibrium may not greatly depend on the rate of movement (for another example, see Holt et al. 2004*b*). The population size at this equilibrium is low and increases directly with M; because the population in this habitat is maintained by migration, an increase in migration boosts its numbers. The evolutionary force of gene flow is determined by the ratio M/N, so the numerical effect of movement on population size in effect moderates the genetic effect.

In figure 2*C*–2*F*, θ is progressively decreased, making density dependence stronger at low densities. The symmetric equilibrium now becomes unstable for a range of movement rates M, and this range is widened as θ is decreased. The region of alternative stable equilibria (a generalist vs. a specialist for either habitat) is narrowed to values of $M < 0.05$ for $\theta = 0.5$ as a result of the instability of the symmetric equilibrium. Asymmetric equilibria are present for an increasing range of M as θ is decreased. Overall, the effect of a low θ in strongly heterogeneous environments is that over most movement rates, one observes only asymmetric equilibria, which have one habitat with high densities and a mean trait value near the local optimum and the other with low numbers and substantial maladaptation due to recurrent gene flow (a source-sink population structure). Thus, strong density dependence at low densities (low θ) is likely to result in a distribution that is evolutionarily restricted largely to a single habitat, because the impact of gene flow is magnified. Weakening density dependence at low densities (high θ), by contrast, makes it harder for gene flow to inhibit local adaptation sufficiently to prevent a species from adapting to both habitats. When density dependence is weak, an increase in immigration boosts population size proportionally, so that the fraction of the population comprised of immigrants each generation is largely unchanged; hence, the force of gene flow is not greater (even though immigration may be). By contrast, with strong density dependence, an increase in immigration does not change abundance much but does increase the fraction of the population made up of migrants and so directly increases gene flow.

The Evolution of Species' Ranges along a Continuous Environmental Gradient

Basic Formulation

Kirkpatrick and Barton (1997) provided a model of the joint evolution of population density and the mean of a quantitative trait along continuous environmental gradients. Their model can be viewed as a continuous space version of the Ronce-Kirkpatrick model analyzed above. The genetic variance is also a fixed parameter of the Kirkpatrick and Barton model, and the intrinsic growth rate, carrying capacity, and strength of stabilizing selection are assumed to be uniform across space (for additional details, see appendix). In addition, the Kirkpatrick and Barton model considers space explicitly, which introduces the space variable x and its dimensionless form X (see appendix). We now modify the dimensionless Kirkpatrick and Barton equations (eqq. [9], [10] in Kirkpatrick and Barton 1997; for details, see appendix) to account for general density dependence (rather than the specific logistic or logarithmic forms of Kirkpatrick and Barton 1997 or Barton 2001), leading to the following dynamical equations:

$$\frac{\partial N}{\partial T} = \frac{\partial^2 N}{\partial X^2} + \left[U(N) - \frac{1}{2}(\overline{Z} - Z_{\text{opt}})^2\right] \times N, \qquad (4a)$$

$$\frac{\partial \overline{Z}}{\partial T} = \frac{\partial^2 \overline{Z}}{\partial X^2} + 2\frac{\partial \ln N}{\partial X} \times \frac{\partial \overline{Z}}{\partial X} - A(\overline{Z} - Z_{\text{opt}}), \qquad (4b)$$

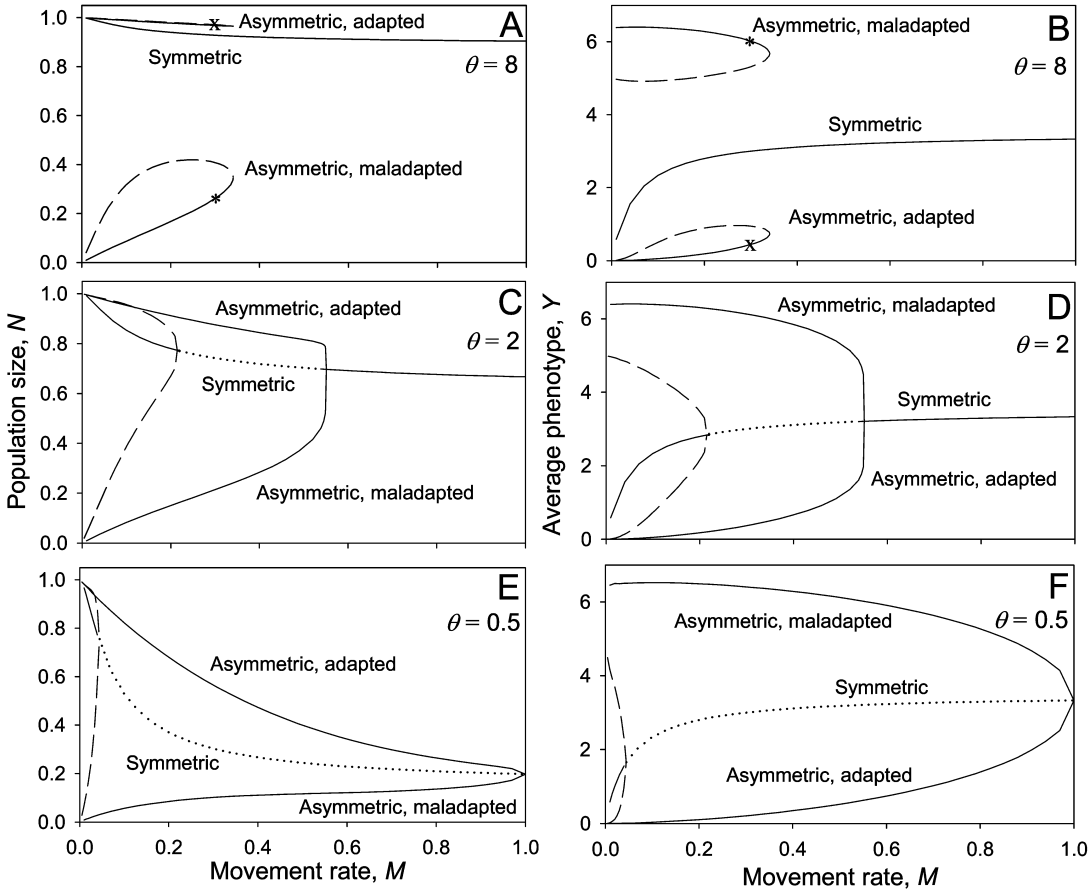

Figure 2: Symmetric and asymmetric equilibria for the generalized Ronce-Kirkpatrick model for differing degrees of nonlinearity in density dependence. In each row, the left side shows the equilibrial population size and the right side the degree of maladaptation for equilibria of equations (1) and (2) as a function of M, the per capita rate of movement between the two habitats. The difference in phenotypic optima between the two habitats is $H = 7$ (a relatively large difference), and the intensity of selection is $\Gamma = 0.1$. Solid lines denote locally stable equilibria (i.e., stable to small perturbations), and dashed and dotted lines are unstable asymmetric and symmetric equilibria, respectively. The symmetric equilibria are given by equations (3). For the stable asymmetric equilibria, one population will be at each of the two levels. The values of θ in the figures are 8 (*A*, *B*), 2 (*C*, *D*), and 0.5 (*E*, *F*). Standard logistic growth is $\theta = 1$. Decreasing θ corresponds to increasing the strength of density dependence at low densities. Overall, an increase in density dependence makes an asymmetrical equilibrium more likely. We show on the abscissa $M \leq 1$, because when $M = 1$, movement occurs at the same rate as the intrinsic growth rate of the species; at higher movement rates, it seems more appropriate to view the system as a single, fairly well-mixed population. For $\theta = 8$ and $M = 0.3$, one asymmetric equilibrium is indicated by an X and the other by an asterisk. There is a very narrow region of alternative stable equilibria near $M = 0.55$ for $\theta = 2$.

where T, X, Z, and N represent dimensionless variables of time, space, phenotype, and population density, respectively (see appendix).

Equations (4a) and (4b) are analogous to equations (2) and (1), respectively, of the two-habitat model. Equation (4a) describes demographic dynamics of local population density, $N(X, T)$, and equation (4b) represents evolutionary dynamics of the local mean phenotype, $\overline{Z}(X, T)$. Demographic dynamics (eq. [4a]) are determined by random dispersal (first term on right-hand side) and local population growth (second term on right-hand side). The local per capita growth rate is the sum of a density-dependent component, $U(N)$, and a maladaptation component that arises because of the deviation of the local mean phenotype, $\overline{Z}(X, T)$, from the spatially varying local optimum phenotype, $Z_{\text{opt}}(X)$.

Local evolutionary dynamics (eq. [4b]) are determined by the symmetric component of gene flow (due to random dispersal; first term on right-hand side), by gene flow asymmetry (which causes gene swamping; second term on right-hand side), and by stabilizing selection toward the local optimum $Z_{\text{opt}}(X)$ (third term on right-hand side).

Note that gene swamping (second term on right-hand side of eq. [4b]) depends on spatial gradients in the logarithm of population density, causing net gene flow from high- to low-density populations.

The effect of stabilizing selection on the evolution of local mean phenotype is given by A, which is called the genetic potential for adaptation (Kirkpatrick and Barton 1997; Case and Taper 2000; Barton 2001). The quantity A combines the genetic variance within a local population and the intensity of selection (see also Barton 2001) and is identical to Γ of the two-habitat model (see appendix). In this model, within-population genetic variance is assumed to be a fixed parameter. Consequences of the evolution of genetic variance itself were investigated by Barton (2001) and are referred to in "Discussion."

Finally, the dimensionless function $U(N)$ represents density dependence of the per capita growth rate. We consider here only negative density dependence; that is, $U(N)$ is monotonically decreasing with density N. (Consequences of inverse density dependence and Allee effects for invasion and range dynamics have been explored by, to cite only a few, Lewis and Kareiva [1993], Keitt et al. [2001], Wang and Kot [2001], Holt et al. [2004*a*], and Taylor and Hastings [2005].) At present, we assume no specific form of density dependence. We later explore the model using the theta-logistic equation.

A Gaussian Approximation

Intuitively, density regulation causes high-density populations to grow more slowly than low-density populations. When density dependence is spatially uniform, this has the effect of making spatial density gradients more shallow, thus weakening gene swamping (the $\partial \ln N/\partial X$ term in eq. [4b] becomes smaller). Consequently, we expect density regulation to oppose the maladaptive force of gene swamping and to facilitate local adaptation and expansion of the species' range. We can demonstrate this effect analytically by employing an approximation and verify our conclusion with direct numerical solutions of equations (4) (see below).

We consider a linear environmental gradient: $Z_{\text{opt}}(X) = B \times X$, where the dimensionless parameter B measures the steepness of the gradient. Additionally, we approximate the population density spatial profile as a Gaussian: $N(X) \approx N_0 \exp(-\kappa X^2/2)$, where κ is a dimensionless measure of range limitation (the higher the value of κ, the more confined is the range). (With no loss of generality, we assume that the center of the species' range is at $X = 0$.) The motivation for such an approximation comes from previous studies that also used a Gaussian density profile (e.g., Kirkpatrick and Barton 1997) and from the fact that such a Gaussian profile is an exact solution of equations (4) in the density-independent case (given $Z_{\text{opt}} = B \times X$; see also "Unregulated Populations") as well as for logarithmic density regulation (Barton 2001).

In contrast to the analysis by Kirkpatrick and Barton (1997), we do the approximation around the center point of the species' range (i.e., around $X = 0$), where population density is maximal, rather than in the range's periphery, where population density is very low (and thus density dependence can be ignored). It can be shown (appendix) that the mean phenotype cline around the range center is also approximated by a linear form: $\overline{Z}(X) = \beta X$. Consequently, a case of unlimited geographic range and perfect adaptation across the entire gradient is described by $\kappa = 0$ and $\beta = B$, while a case of limited geographic range and increasing levels of maladaptation is described by a positive value of κ and $\beta \neq B$. If κ is large, the species' abundance is very sharply peaked around a single point on the gradient (i.e., the range center $X = 0$).

We can now derive the following equation for the dynamics of range limitation κ:

$$\frac{\partial \kappa}{\partial T} = -\phi(N_0) \times \kappa - 2\kappa^2 + (\beta - B)^2 \qquad (5)$$

(details in appendix). Density dependence is incorporated in equation (5) via the term

$$\phi(N_0) = -N_0 \times \left.\frac{dU}{dN}\right|_{N=N_0}, \qquad (6)$$

where N_0 is the population density at the range center. Thus, ϕ is the strength of density regulation at the range center ($X = 0$), where density is maximal. This expression (eq. [6]) was previously derived as a measure of the strength of density regulation in purely demographic models of stochastic population dynamics (e.g., eq. [6] is equivalent to $-\partial \ln \lambda/\partial \ln N$ of Lande et al. [2002] and Sæther et al. [2002]). This same measure is implicated here in the evolutionary dynamics of species' ranges. Values of ϕ are positive, because we consider only negative density dependence (i.e., $dU/dN < 0$).

Equation (5) represents the tension between "forces" that oppose range limitation and those that promote it. On the one hand, random dispersal ($-2\kappa^2$) and density regulation ($-\phi(N_0)\kappa$) strive to homogenize densities across space (decrease κ), thus opposing range limitation. On the other hand, local maladaptation (i.e., $(\beta - B)^2$) creates spatial differences in population growth rates along the gradient, thus resulting in spatial density gradients and asymmetric gene flow that works to maintain and increase maladaptation and, consequently, range limitation. The

balance between those opposing processes determines the equilibrium range limitation κ_{eq}, that is, whether the range is limited at all and, if so, to what extent.

We have demonstrated that, in general, spatially uniform density regulation opposes the limitation of a species' range and facilitates local adaptation along an environmental gradient. Next, we describe conditions for limited-range equilibria under different forms of density regulation, and then we move to consider the effect of density regulation on evolutionary invasion dynamics. The numerical results and figures presented are based on numerical integration of the original dynamical equations (eqq. [4]; for an explanation of the numerical procedure, see appendix).

Unregulated Populations

During the establishment phase, a newly invading species may have a population density low enough that its dynamics are effectively unregulated. The density-independent case of equations (4) (i.e., $U(N) = 1$ and $\phi = 0$) was analyzed by Kirkpatrick and Barton (1997), who showed that the outcome of range evolution is governed by the dimensionless parameters A and B. For a given selection intensity A, if the environmental gradient is not too steep, so that $B < B_L = A/2^{1/2}$, the species may expand indefinitely. However, if $B > B_L$, stable range limits arise and the species exhibits a limited-range equilibrium. In addition, if the environmental gradient is even steeper, so that $B > B_U = (A + 2)/2^{1/2}$, the species cannot persist and becomes globally extinct. If it persists, because population growth is unregulated, the species grows exponentially. In the unlimited-range case, the steady state exponential growth rate is $\partial \ln N/\partial T = 1$ (i.e., the steady state solution is given by $N(T, X) = N_0 e^T$), which is the maximal rate (using the dimensionless variables of eqq. [4]). In the limited-range case, the steady state exponential growth rate is given by

$$\frac{\partial \ln N}{\partial T} = \frac{B_U - B}{\sqrt{2}}, \tag{7}$$

which is lower than 1 for limited-range solutions (i.e., for $B > B_L$) and negative when $B > B_U$ (as expected, because the species becomes globally extinct in the latter case). Therefore, a population with a limited range will also initially grow more slowly than one with an unlimited range. This happens because of the source-sink dynamics that develop in the limited-range equilibrium; central populations suffer a loss of individuals as a result of emigration into peripheral environments where the species is maladapted. As shown below, this expression (eq. [7]) has important consequences for invasion dynamics.

Regulated Populations

Equation (5) captures the effects of density regulation (ϕ) on the dynamics of range limitation (κ). The strength of density regulation depends on central population density, N_0 (eq. [6]). Therefore, the form of density dependence ($U(N)$) is potentially important in determining the equilibria, the limited-range threshold B_L, and the dynamics of the species' range. For example, Barton (2001) demonstrates how the limited-range threshold is increased (relative to $B_L = A/2^{1/2}$ of the unregulated case), given logistic or logarithmic density regulation (for the latter, ϕ is independent of density). Therefore, with density regulation, steeper environmental gradients are needed for limited-range equilibria to be maintained by gene flow.

Next, to provide a more concrete example, we considered the theta-logistic used in the two-habitat model ($U(N) = 1 - N^\theta$) and numerically solved equations (4) for different combinations of A, B, and θ (for more details, see appendix). Figure 3 presents how B_L (i.e., the limited-range threshold) and B_U (i.e., the extinction threshold) vary with the selection intensity A for several values of θ. First, the extinction threshold curve is independent of the value of θ and is still given by $B_U = (A + 2)/2^{1/2}$ (i.e., the expression derived for unregulated dynamics; more details in appendix). Note that for the two-habitat model above, the condition for persistence was also independent of θ.

Figure 3 also demonstrates that as θ increases, the limited-range threshold is increasingly pushed upward. For a given value of A, steeper environmental gradients are required to limit a species' range as θ increases. Overall, as θ increases, there is a shrunken range of the A-B parameter space that permits stable range limits. Why is that? In the appendix, we demonstrate that the strength of density regulation at equilibrium is given by

$$\phi_{eq} = \theta \times (1 - \kappa_{eq}) \tag{8}$$

(using the previously described Gaussian approximation). Hence, the strength of density regulation at equilibrium increases with θ (see also Sæther and Engen 2002), opposing range limitation more strongly (see eq. [5]) and causing a higher limited-range threshold. Another way of thinking about this is suggested by figure 4. Close to the limited-range threshold (both above and below it), the equilibrium population density is practically $N = 1$, that is, at the carrying capacity. Figure 4 shows that at high densities (close to $N = 1$), the strength of density regulation, ϕ, indeed increases with θ, causing a higher limited-range threshold.

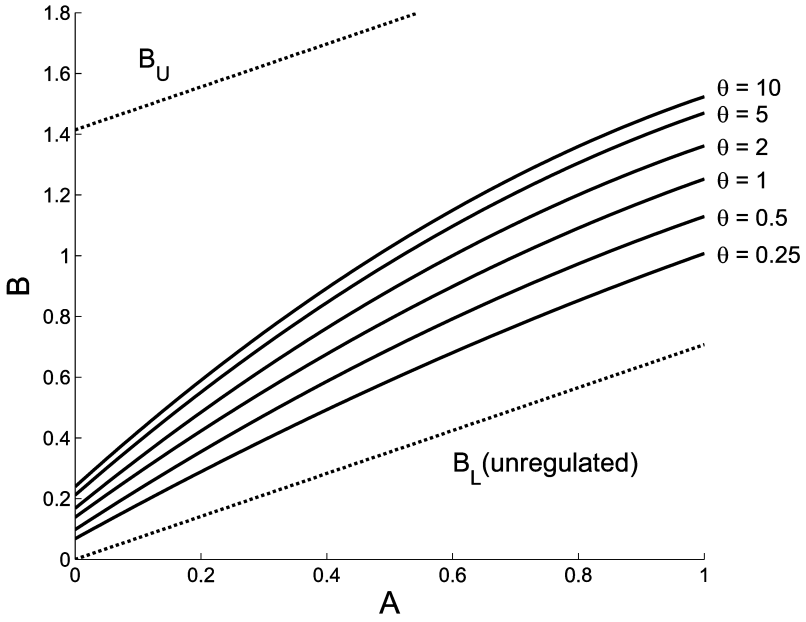

Figure 3: Limited-range threshold B_L for different forms of density regulation (i.e., for unregulated dynamics and for different values of θ) given in the *A*-*B* parameter space. Combinations of *A* (genetic potential for adaptation) and *B* (steepness of environmental gradient) that cause unlimited expansion are below that threshold curve. Combinations that cause stable range limits are above it. Combinations that end in global extinction lie above the extinction threshold curve B_U.

Comparison with the Two-Habitat Model

Both the previous two-habitat analysis and this analysis of a continuous environmental gradient point to the same effect of negative density dependence. As density dependence is increasingly pushed into higher densities (e.g., as θ increases in the theta-logistic model), it becomes more difficult to obtain stable range limits and habitat specialization. This effect of the form of density regulation is evident both in figure 1 for the two-habitat model and in figure 3 for the continuous gradient model. Asymmetric solutions (i.e., habitat specialization) in the two-habitat model require larger differences among habitat types (larger *H*) as θ increases. Similarly, in the continuous case, stable range limits require steeper environmental gradients (higher *B*) for greater values of θ. Thus, despite differences between the two models (most notably, the symmetric solutions of the two-habitat model involve a uniform level of maladaptation across all habitats; this can never be a stable solution in the continuous case), the negative effect of strong spatially uniform density regulation near the population equilibria on habitat specialization and limitation of a species' range seems to be a robust result. Conversely, if density dependence near equilibrium is weak (low θ), it is easier for evolution to constrain species' niches and geographical ranges.

Implications for Biological Invasions

García-Ramos and Rodriguez (2002) studied biological invasions using the equations of Kirkpatrick and Barton (1997; i.e., eqq. [4] with logistic density dependence). They demonstrated that because of local maladaptation in the advancing wave front, the species' invasion proceeds more slowly than in the comparable purely demographic model of Fisher (1937) and Skellam (1951). As the environmental gradient becomes steeper, the invasion speed gradually decreases until it becomes 0 when *B* exceeds B_L. However, before reaching a steady state (of either range expansion at a constant rate or a stable range limit), there is a period of transient dynamics in which quite different behaviors may be observed. This is the focus of the next section.

Quasi-Stable Range Limits

The steady state behavior of García-Ramos and Rodriguez (2002) is obtained once the central population of the newly established species reaches carrying capacity (i.e., *N* very close to 1). However, during the initial time period, for example, shortly after introduction, all populations are low in density. Hence, the species will exhibit effectively unregulated population growth for some time. The period of time for such effectively unregulated behavior depends on the form of density dependence (here the value of θ).

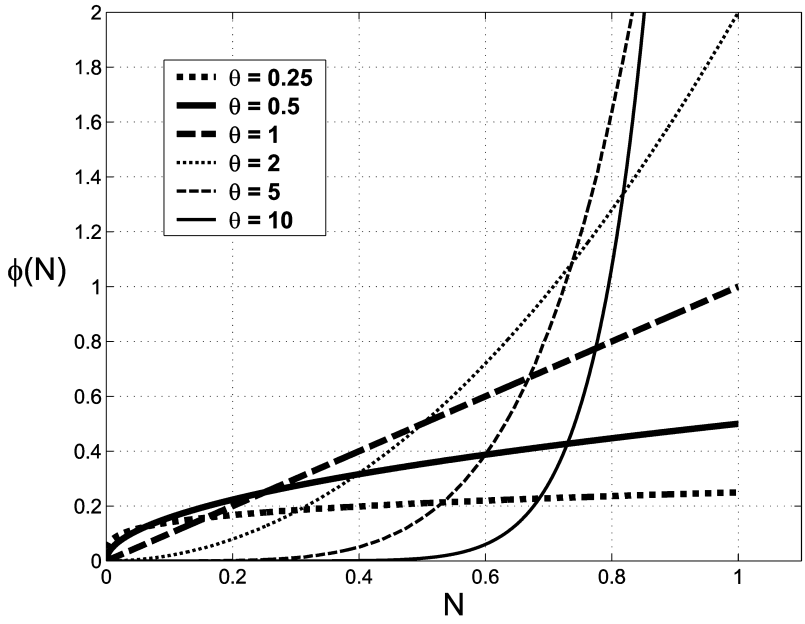

Figure 4: Strength of density regulation ϕ as a function of density N. Given equation (6) for ϕ and theta-logistic density dependence, $\phi(N) = \theta N^{\theta}$, which is plotted for several values of θ. Note that for $\theta < 1$ (convex forms of density dependence), the strength of density regulation rises sharply at low densities and then continues to grow slowly as density approaches carrying capacity. For $\theta > 1$ (concave forms), the strength of density regulation is effectively 0 at low densities and rises sharply only for intermediate and high densities. The range of densities in which such unregulated dynamics ($\phi \approx 0$) is operating increases with θ.

As θ increases, density dependence is pushed into increasingly higher densities, below which population growth is effectively unregulated (see fig. 4).

In figure 3, consider the region in A-B parameter space that lies above the unregulated limited-range threshold (i.e., $B > A/2^{1/2}$) but below that for density-regulated dynamics (curves labeled with values of θ). Within this region, unregulated dynamics dictate limited-range equilibria, but density-dependent dynamics cause unlimited expansion (i.e., invasion). The overall resulting dynamics include transient quasi-stable range limits during the initial low-density phase of a biological invasion (i.e., shortly after introduction). This quasi-stable phase lasts until density dependence is strong enough to initiate the steady state phase with its constant rate of expansion. We note that as θ increases and B_L increases (fig. 3), the region in A-B parameter space with quasi-stable behavior expands. Thus, as θ increases, there are more opportunities (combinations of A and B) for steady state unlimited expansion but also more opportunities for quasi-stable range limits during the initial low-density phase of an invasion.

Biologically speaking, if the environmental gradient is steep enough, the species may be confined to a limited range during the initial phase of the invasion, because gene swamping is strong enough to prevent local adaptation to a new environment. The limited-range quasi equilibrium is maintained until population densities become high enough for density regulation to weaken gene swamping, thus potentially "releasing" the species from the constraints of gene flow impeding local selection, initiating an unlimited expansion.

Typical trajectories of a species' range after introduction are shown in figure 5*A*–5*C*. We adopt an operational definition of range "size" based on statistical analyses of species' ranges (Gaston 2003, pp. 23–24; Fortin et al. 2005 and references therein). Range limits are defined as the boundary outside which local population density drops below 1% of the maximum. During the initial low-density phase of the invasion, the species' range tends toward the quasi-stable equilibrium (denoted by κ_{qs}). However, when density regulation finally becomes strong enough, the dynamics change (and quite abruptly for high values of θ) into steady state unlimited expansion (invasion). Also, note that if the initial species' range is wider than the quasi-stable range size (fig. 5*A*), then initially the range actually contracts. Initial dynamics of the species' range may not represent its eventual steady state invasion dynamics, and thus monitoring the initial range change of a species in a novel biome may lead to a very poor prediction of its ultimate invasive potential.

As discussed above, the duration of this quasi-stable initial phase is longer for higher values of θ. Recall that

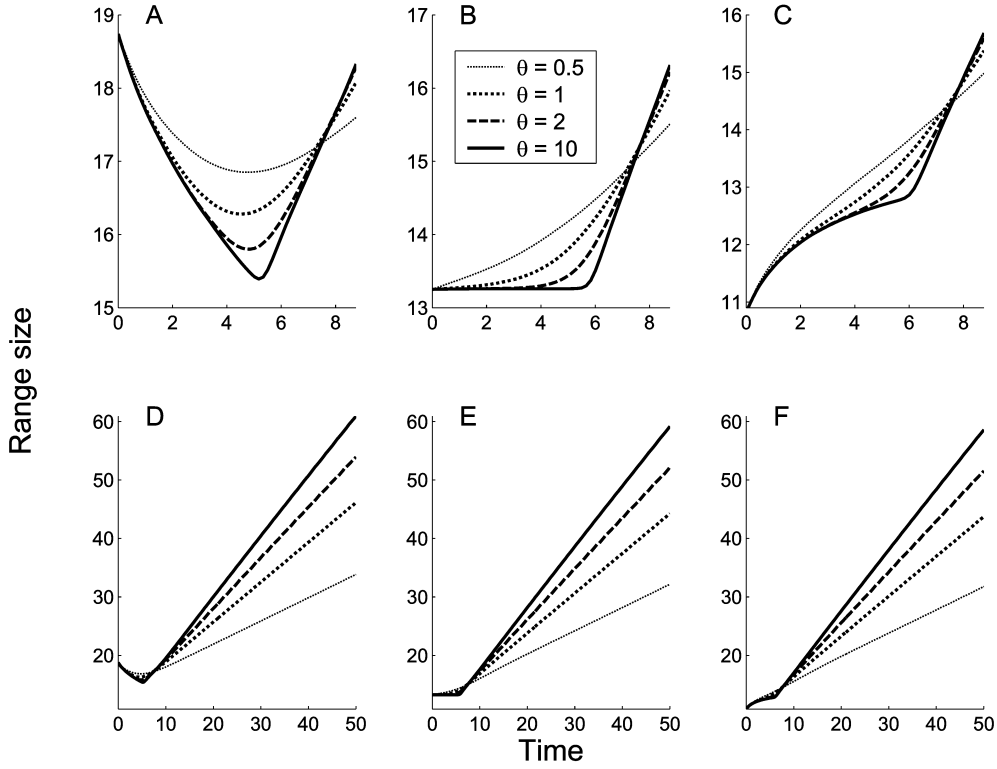

Figure 5: Dynamics of range size, given four different forms of density regulation (i.e., values of θ) and three different initial range sizes: $\kappa(t = 0) = 0.5\kappa_{qs}$, that is, initial range is wider than the quasi-stable range size (*A*, *D*); $\kappa(t = 0) = \kappa_{qs}$, that is, initial range is equal to the quasi-stable range size (*B*, *E*); and $\kappa(t = 0) = 1.5\kappa_{qs}$, that is, initial range is narrower than the quasi-stable range size (*C*, *F*). *A*–*C* present the range dynamics during the initial quasi-stable phase. *D*–*F* extend the time axis to present the steady state behavior of range expansion. Note in *A* that because the initial range is wider than the quasi-stable range size, then initially the species' range actually contracts until density dependence becomes strong enough to reverse this trend. The duration of this initial range contraction is longer for higher values of θ. In *B*, because range size is initialized at the quasi-stable level, then range size remains constant for a period of time that increases with θ. In *C*, because initial range size is narrower than the quasi-stable size, then range size initially approaches the quasi-stable size but in a decelerating manner until density dependence becomes strong enough to initiate unlimited expansion. *D*–*F* demonstrate how the invasion speed at steady state increases with the value of θ. In all cases, the trajectories are obtained for $A = 0.5$ and $B = 0.65$. The quasi-stable range limitation κ_{qs} is 0.21. Initial population size at range center is $N_0(t = 0) = 0.01$.

the exponential growth rate of the unregulated limited-range equilibrium decreases linearly as the environmental gradient becomes steeper (i.e., as B increases; eq. [7]). Hence, the duration of the quasi-stable phase not only increases with θ but also should be longer for steeper environmental gradients (i.e., $T \sim (B_U - B)^{-1}$; eq. [7]). Other processes that can further depress the per capita growth rate, such as demographic stochasticity and Allee effects (both expected to operate at low population densities), could also increase the duration of the quasi-stable phase.

Consequences for Accelerating and Steady State Speeds of Invasion

During the initial phase of quasi-stable range limits, range size may contract (if $\kappa_{t=0} < \kappa_{qs}$), remain constant for a while, or expand in a decelerating manner (fig. 5*A*–5*C*). The transition into steady state expansion involves an increase in the invasion speed; this is quite abrupt at large values of θ (e.g., $\theta = 10$). This rapid transition arises because when density dependence finally begins to operate, for large θ values it does so very strongly (see fig. 4; eq. [5]). Figure 5*D*–5*F* extends the curves in figure 5*A*–5*C* to show a trend in the steady state speed of invasion. As θ increases, invasion speed also increases as a result of the interplay of local adaptation and demography in newly invaded sites along the gradient. Differences in steady state invasion speed between different forms of density regulation disappear in a homogenous selective environment (i.e., when $B = 0$), as expected (fig. 6). Finally, as noted above, García-Ramos and Rodriguez (2002) found that as the environmental gradient is steepened, steady state in-

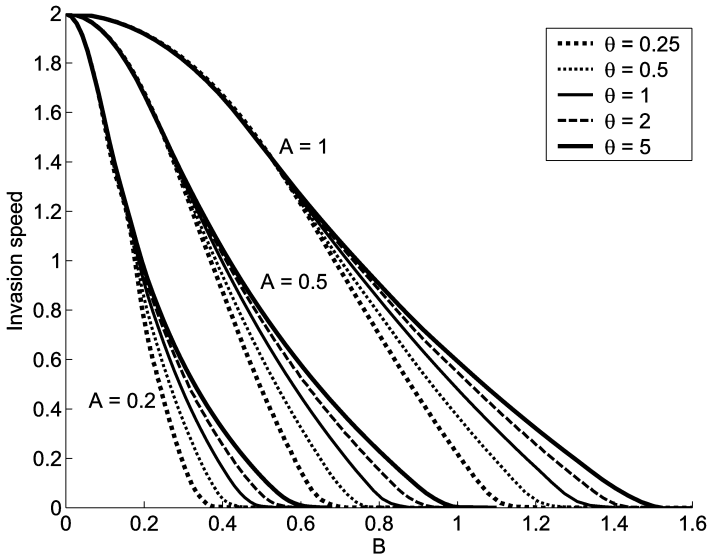

Figure 6: Steady state invasion speed as a function of the steepness of the environmental gradient B, given three different values of A (genetic potential for adaptation) and five different forms of density regulation (i.e., values of θ). Invasion speed is given in rescaled, dimensionless space time variables (see explanation of eqq. [4]). Therefore, its maximal value is 2 (see also García-Ramos and Rodriguez 2002). Note that as the environment becomes increasingly heterogeneous (as B increases), differences among forms of density regulation become more pronounced. Invasion speeds are the same (equal to 2) when the environment is homogenous ($B = 0$). For each curve, the B-axis intercept defines the limited-range threshold B_L for those values of A and θ.

vasion speed decreases, becoming 0 when B equals B_L. Figure 6 demonstrates this effect for several values of A and θ. In addition to the effect of the gradient, figure 6 also shows the effect of the form of density regulation (i.e., value of θ) on the steady state invasion speed; increasing θ leads to faster invasion along steeper (higher B) gradients.

Discussion

We have explored how the form of density regulation affects the interplay between demography and evolution that determines both invasion dynamics and the potential for evolutionarily stable ranges. We demonstrate that, in heterogeneous environments (either a mosaic of two discrete habitats or a continuous gradient), the specific form of negative density regulation influences steady state invasion speed, and the appearance and duration of initial invasion lags. Such effects of negative density dependence on invasion dynamics are in sharp contrast to the results of purely demographic models, for which invasion speed depends only on the maximal intrinsic growth rate (e.g., Shigesada and Kawasaki 1997). The form of density regulation also affects conditions for being either a habitat specialist or a generalist and conditions for stable range limits to occur. Thus, when analyzing rates and patterns of species invasions or range boundaries, it is important to consider density dependence. General patterns of invasions (e.g., initial lags and accelerating rates), as well as the invasive potential of specific species, may very well be connected to the form of the density dependence (see below).

Using a two-habitat model, we demonstrated that habitat specialization becomes less likely as density regulation becomes increasingly concave (i.e., density dependence that is weak at low densities and strong at high densities, corresponding to increasing θ in the theta-logistic model). Larger differences between habitats are likewise required for migrational meltdown to occur with more concave patterns of regulation (fig. 1). Similarly, for a continuous environmental gradient, we demonstrated that as density regulation becomes increasingly concave, so that density dependence is strong near equilibrium, there is less opportunity for stable range limits to arise, because steeper gradients are needed to prevent unlimited invasion (see fig. 3).

We have also demonstrated how gene swamping might cause quasi-stable range limits, or even phases of range collapse, during the initial phases of a biological invasion, even when the ultimate range is unbounded. This transient phase is followed by a phase of accelerating expansion; the eventual steady state invasion speed depends on the form of density regulation (e.g., in the theta-logistic model, higher values of θ lead to faster invasions). Differences in steady state invasion speed in our model are not an out-

come of different forms of density regulation per se but rather are caused by the feedback between demography and evolution in heterogeneous environments, which in turn is altered by the form of density regulation.

One effect of a quasi-stable phase of invasion is an initial time lag. Such lags are often observed in biological invasions (e.g., Crooks 2005). Several alternative mechanisms have been proposed to explain these lags. An Allee effect may cause long initial periods of sluggish growth and difficulty in adapting to novel environments (Lewis and Kareiva 1993; Keitt et al. 2001; Wang and Kot 2001; Holt et al. 2004*a*; Taylor and Hastings 2005). The level of initial maladaptation to the novel environment at the point of introduction can also influence establishment success (Holt et al. 2005*a*; see also Holt and Gomulkiewicz 1997*a*). Our results suggest a different mechanism. Gene swamping during an initial phase of unregulated population growth temporarily maintains habitat specialization, causing quasi-stable range limits that eventually dissolve when populations approach carrying capacity. This process depends on the form of density dependence at intermediate and high population densities and assumes the importance of local adaptation along a gradient, unlike Allee effect–based explanations, which depend on density dependence at low densities and pertain even in spatially homogeneous environments. The process we have identified may delay species' invasion, even when population density grows and the Allee effect is no longer operating. Note that in the specific scenarios we have explored, the central population of the invading species is assumed to be locally adapted. Initial maladaptation at the site of introduction is thus not required for time lags to occur in invasion; our suggested mechanism can cause time lags even in the absence of initial maladaptation or Allee effects. Of course, all these distinct explanations may operate in a complementary, mutually reinforcing fashion.

Our models suggest a novel mechanism leading to accelerating rates of invasion. The two current demographic explanations for accelerating rates of invasion are Allee effects and fat-tailed dispersal kernels (i.e., long-distance dispersal; Hastings et al. 2005; Taylor and Hastings 2005). These demographic processes operate in both homogenous and heterogeneous environments. Holt et al. (2005*a*) suggested an evolutionary mechanism for accelerating invasion rates in homogenous environments. An initially maladapted species gradually adapts to the novel environment; its per capita growth rate (r) thus increases, resulting in increasingly faster invasion speeds. We suggest that in heterogeneous environments, the feedback between demography and evolution can also cause initially low invasion rates, followed by acceleration to higher steady state invasion speeds.

Prior studies of the Kirkpatrick and Barton model have focused on equilibrium solutions (Kirkpatrick and Barton 1997; Case and Taper 2000; Barton 2001) or steady state behavior (García-Ramos and Rodriguez 2002), with little attention given to transient behaviors. Moreover, previous analyses of the model used logarithmic (Barton 2001) or logistic (i.e., $\theta = 1$; Kirkpatrick and Barton 1997) density regulation, for which a quasi-stable phase does not occur or is very brief (see fig. 5 for the logistic form). Allowing density dependence to be weak at low densities and strong at high densities permits quasi-stable range limits. Finally, our models assumed fixed genetic variance. By explicitly including also the dynamics of genetic variance, Barton (2001) showed that initially low genetic variance may maintain a limited species' range for long periods, until enough additive genetic variance builds up to facilitate adaptation and expansion (see also Lee 2002; Blows and Hoffmann 2005). It would be interesting to examine how the form of density regulation affects invasion and range dynamics when the dynamics of the genetic variance are also taken into account.

Other possible extensions of the models presented here include spatial variation in the form of density regulation, in addition to the spatial heterogeneity in selective optima considered in this article. If marginal populations also have unusually strong density dependence at low densities, then the model of Holt (1996) and Gomulkiewicz et al. (1999) suggests that gene flow may be particularly effective at limiting local adaptation. An important open question is analyzing coupled evolutionary and demographic dynamics along gradients for species with age/size/stage structure. For example, it may be that selection mostly occurs at the juvenile phase, while density-dependent effects are most strongly exerted by mature individuals that are more likely to have locally favored genotypes. If traits that lead to local adaptation also permit individuals to be superior in competition before reproducing, selection against immigrants can be amplified by density dependence. This should make it more difficult for gene flow to prevent niche evolution and range expansion.

Sæther and Engen (2002) give estimates of θ ranging from 0.15 to 11.17 for 11 bird species. The R variable of Fowler (1988) can be readily translated into estimates of θ that fall between 0 and ~20 (for more details, see appendix). Many studies provide evidence for strongly nonlinear forms of density regulation (e.g., Fowler 1981; Sibly and Hone 2002; Sinclair 2003), including both convex ($\theta < 1$) and concave ($\theta > 1$) forms. In the face of these wide ranges of estimated values of θ from natural populations, all of the different dynamical behaviors described in this article are potentially feasible in nature. Specifically, the potential for quasi-stable range limits, transient range contraction, and acceleration in invasion speeds would

likely vary among species because of their correspondingly different values of θ.

In addition, the form of density regulation may reflect life-history syndromes. For instance, Fowler (1988) and Sæther and Engen (2002) find significant negative relationships between θ (or R in Fowler's study; see appendix) and the intrinsic (i.e., maximal) per capita growth rate, r (Sæther and Engen 2002), or the rate of increase per generation (rT, where T denotes here generation time; Fowler 1988). These correlations suggest that the form of density regulation is related to other life-history characteristics (e.g., the "slow-fast continuum" of Sæther and Engen 2002). Clades that differ in these life-history variables thus might systematically differ in the likelihood of evolutionary constraints on range limits.

Predicting the invasion potential of species, based on life-history traits and environmental characteristics, is the Holy Grail of invasion biology (e.g., Kolar and Lodge 2001; Grotkopp et al. 2002; Facon et al. 2006). So far, this has been an elusive goal (e.g., Kolar and Lodge 2001). We suggest that the form of density regulation may play an underappreciated role in determining invasiveness (see also Holt et al. 2004*a*). As noted above, life-history traits related to invasiveness (e.g., Kolar and Lodge 2001) are correlated with the form of density regulation. However, much residual variation exists around those correlations (e.g., Sæther and Engen 2002). Examining the form of density regulation may help to sharpen differences between invasive and noninvasive species. We suggest that the predictions outlined in this article concerning the role of density regulation in biological invasions involving adaptation to novel heterogeneous environments open up a new path for comparative, empirical, and field investigations of species invasiveness and evolutionarily stable range limits.

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