

# Predation and the Evolutionary Dynamics of Species Ranges

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**ABSTRACT:** Gene flow that hampers local adaptation can constrain species distributions and slow invasions. Predation as an ecological factor mainly limits prey species ranges, but a richer array of possibilities arises once one accounts for how predation alters the interplay of gene flow and selection. We extend previous single-species theory on the interplay of demography, gene flow, and selection by investigating how predation modifies the coupled demographic-evolutionary dynamics of the range and habitat use of prey. We consider a model for two discrete patches and a complementary model for species along continuous environmental gradients. We show that predation can strongly influence the evolutionary stability of prey habitat specialization and range limits. Predators can permit prey to expand in habitat or geographical range or, conversely, cause range collapses. Transient increases in predation can induce shifts in prey ranges that persist even if the predator itself later becomes extinct. Whether a predator tightens or loosens evolutionary constraints on the invasion speed and ultimate size of a prey range depends on the predator effectiveness, its mobility relative to its prey, and the prey's intraspecific density dependence, as well as the magnitude of environmental heterogeneity. Our results potentially provide a novel explanation for lags and reversals in invasions.

**Keywords:** range limits, gene flow, gradient, habitat specialization, predation.

## Introduction

The study of species range limits is pivotal to ecology, drawing on many different subfields and providing a unifying theme for a wide range of ecological investigations (Antonovics et al. 2006; Gaston 2009). Understanding the drivers of range limits is increasingly urgent, given the need to project how species and communities will respond to global change and to predict the ultimate distribution of invasive species. There is increasing interest in integrating evolutionary and ecological perspectives into explanations of range dynamics (Case et al. 2005; Bridle and

Vines 2007; Sexton et al. 2009). The interaction between demography and evolution—mediated in part by the interplay of gene flow and selection—has emerged as a potentially key driver of range dynamics (e.g., Kirkpatrick and Barton 1997; Gomulkiewicz et al. 1999; Kawecki 2008), local adaptation (Garant et al. 2007), and niche conservatism (Wiens et al. 2010).

Traced back to Haldane (1956) and Mayr (1963), the essential idea is that a flow of individuals from abundant (e.g., central) populations to sparsely populated range margins can restrain local adaptation at the margins, constraining geographical distributions. Asymmetric gene flow (potentially overwhelming selection) could be particularly strong when genetic variation is limited and low- and high-density populations are spatially close, for instance, along sharp environmental gradients (Kirkpatrick and Barton 1997; Barton 2001), or where distinct habitats are closely juxtaposed. Population density should reflect how well adapted a species is to local environments, so spatial variation in density could itself reflect the interplay of gene flow and selection. The implications of this positive feedback between demography and selection has been the focus of considerable attention for single species evolving in heterogeneous landscapes (e.g., Kirkpatrick and Barton 1997; Gomulkiewicz et al. 1999; Antonovics et al. 2001; Holt 2003; Filin et al. 2008; Polechova et al. 2009).

Yet few species live in isolation. How do interspecific interactions influence evolutionary range limitation by gene flow? Interspecific competition can constrain species' ranges (MacArthur 1972; MacLean and Holt 1979; Roughgarden 1979; Case et al. 2005) and, at times, magnify the impact of gene flow. Case and Taper (2000) adapted the model of Kirkpatrick and Barton (1997) to include a competitor and demonstrated that competition could sharpen spatial gradients in density and magnify the inhibitory impact of gene flow on selection, particularly given sharp or abrupt changes in selective optima along gradients. Species borders and replacements of related species often occur across transitions in vegetation structure (Boone and Krohn 2000), consistent with this prediction.

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Another major class of interspecific interactions influencing range limits consists of those between natural enemies and their victims, such as predator-prey and host-pathogen interactions. Prey and hosts are resources for their respective natural enemies, so the ranges of these resources obviously could constrain predator and pathogen distributions (Holt 1997a; Alexander et al. 2007). Conversely, generalist predators and other natural enemies can limit their victim's distributions (Case et al. 2005; Holt and Barfield 2009). This is not a new observation; in his classic article introducing the niche concept, Grinnell (1917) suggested that the California thrasher (*Toxostoma redividum*) is restricted to chaparral because of high predation from hawks in woodland. Hawks are generalist predators presumably sustained in their abundance by alternative woodland prey, permitting them to exclude this vulnerable bird species from a portion of its potential geographical range.

By contrast, specialist predators at first glance seem unlikely to govern prey range limits. The reason is simple: beyond the prey range limit, such a predator has nothing to eat and so tends to become extinct. However, this first-order expectation of the unimportance of specialist enemies as a cause of range limits of their victims may not always hold (Antonovics 2009; Holt and Barfield 2009). For instance, dispersal by the predator from sites with productive, abundant prey can sustain predators in marginal populations of prey, imposing a range limit on the prey (Hochberg and Ives 1999; Owen and Lewis 2001; Holt and Barfield 2009).

Beyond such purely ecological effects, and pointing in the converse direction, Holt and Barfield (2009) conjectured that predators could alter the constraining influence of gene flow on prey range limits via effects on prey abundance, at times facilitating habitat generalization and range expansion in prey. Specialist predators can grow and depress prey numbers to a level that just keeps the predator's own growth in check. If attack rates and other predator parameters are spatially invariant, at equilibrium the predator should reduce its prey to uniform abundances everywhere the predator persists, flattening spatial variation in prey abundance that would otherwise exist. This could weaken asymmetries in net rates of prey movement, reduce the effect of gene flow relative to selection, and thereby indirectly permit a prey species to expand its habitat range or distribution along a gradient. The indirect evolutionary effect of predation on prey habitat use and ranges could thus qualitatively differ from its direct ecological effect.

Here we examine this conjecture in detail using models of coupled ecological and evolutionary dynamics. We extend models explored earlier (Kirkpatrick and Barton 1997; Ronce and Kirkpatrick 2001; Filin et al. 2008; Holt and Barfield 2009) by splicing a model of prey evolution

with models of coupled predator-prey population dynamics, which alter the interplay of demography, gene flow, and local adaptation in a prey species. We first consider how a predator can influence the evolutionarily stable habitat range of its prey in landscapes comprised of two discrete habitats. We then turn to predator-prey interactions along smooth gradients and explore how predation alters evolutionary stability of range limits, as well as transient range dynamics. The actual landscapes in which range dynamics unfold are complex. We seek to identify common features of coupled ecological and evolutionary dynamics in two idealized landscapes—a pair of linked discrete habitats, and smooth continuous gradients—in the hope of reaching robust conclusions about predator impacts on prey ranges.

#### The Evolution of Species Ranges in Two-Habitat Landscapes with Predation

Consider a landscape containing two distinct habitats of equal area coupled by dispersal. To examine how predation influences the evolution of prey habitat specialization and generalization, we use a model presented by Ronce and Kirkpatrick (2001) and extended by Filin et al. (2008) to encompass a broader range of nonlinear density dependence in prey growth. In this model, a species experiences stabilizing selection on a quantitative trait with different optima in each habitat. Specialists have trait values near one habitat's optimum, while generalists are nearer the average of both. A specialist has high density in the habitat to which it is adapted and low density in the other, while a generalist population has more nearly equal densities in both. In a specialist, the mating of immigrants and residents in the suboptimal habitat can overwhelm selection, constraining adaptation there and maintaining habitat specialization. This model predicts alternative equilibria, the likelihood of which varies with the degree of habitat heterogeneity, movement rates, and the nonlinear form of density dependence. Ronce and Kirkpatrick (2001) noted that habitat generalists may lose adaptation to one habitat as a result of the swamping effects of gene flow after numbers are perturbed to low levels, a phenomenon they called "migrational meltdown." Predation, by altering habitat-specific prey abundance, can shift the relative strengths of gene flow and selection and thus influence evolutionary stability of different patterns of prey habitat use.

We generalize the model developed by Ronce and Kirkpatrick (2001) in two respects (for more details, see appendix in the online edition of the *American Naturalist*): the prey can experience nonlinear density dependence and predation in one or both habitats  $i$  and  $j$ . Prey abundance in habitat  $i$  is  $N_i$ , and its maladaptation (the difference

between its phenotype and the habitat optimum) there is  $Y_i$ . The model for the prey habitat  $i$  is as follows:

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

$$\frac{dN_i}{dT} = (1 - N_i^\theta) N_i - \frac{\Gamma}{2} Y_i^2 N_i + M(N_j - N_i) - D_i N_i. \quad (2)$$

(The equations for habitat  $j$  are the same, with a switch of subscripts.) Equation (1) describes phenotypic evolution and matches equation (6a) of Ronce and Kirkpatrick (2001). The first term on the right is selection, driving  $Y_i$  toward 0 (and therefore the prey phenotype toward the habitat optimum). The quantity  $\Gamma$  is the rate of evolution and includes (fixed) genetic variation and the strength of selection. Without movement, the population in each habitat equilibrates at the local optimum. The two habitats differ in their optima by  $H$  (a measure of habitat heterogeneity). Per capita movement at rate  $M$  (the same in both directions) leads to maladaptive gene flow (the second term). This flux is biased toward the habitat with lower numbers, producing greater maladaptation there. The expectation of greater maladaptation in habitats with low abundance is a general prediction of models for evolution in heterogeneous landscapes (Kawecki 2008). Note that predation does not directly drive prey evolution. We assume the predator does not evolve.

Equation (2) describes prey population dynamics. Time is scaled by the prey intrinsic growth rate and density by its carrying capacity. The first term is inherent prey growth (including nonlinear density dependence), the second expresses demographic load from maladaptation, the third gives the direct effect of dispersal on population size, and the last is mortality from predation at a habitat-specific per capita rate of  $D_i$ . The form of density dependence is theta logistic (Sæther and Engen 2002; Filin et al. 2008). The larger  $\theta$  is, the weaker density dependence is at low density. Equation (2) generalizes equation (6b) of Ronce and Kirkpatrick (2001) to include nonlinear density dependence and mortality due to predation.

We first consider the impact of predation acting simply as a density-independent source of mortality (fixed  $D_i$ ) and then allow predator numbers to vary dynamically with prey availability, driving variation in  $D_i$ . The former assumption could describe a generalist predator, sustained by alternative prey. The latter is appropriate if the predator is a specialist, dependent on our focal prey species.

#### *Predation as a Density-Independent Mortality Factor*

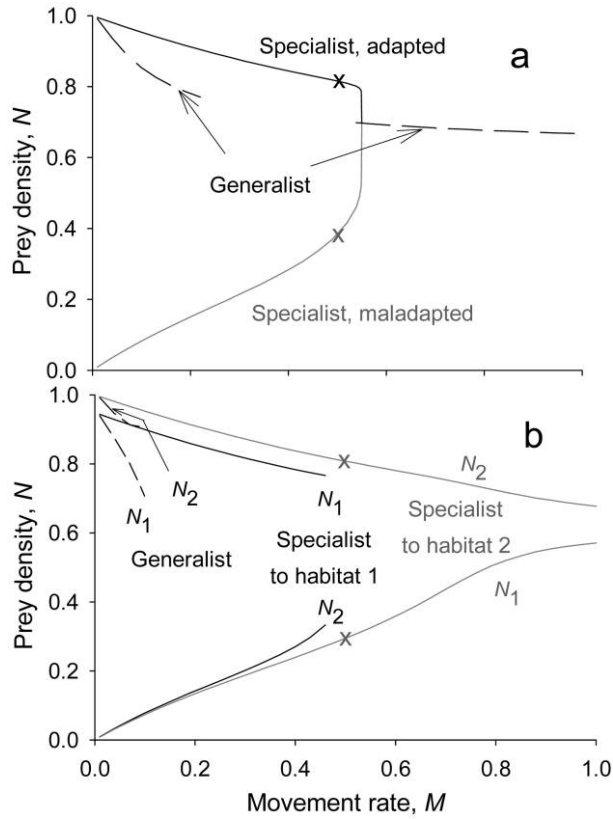
Predation can alter the likelihood of habitat generalization or specialization in a prey species. With fixed predation, the above model can have multiple equilibria, including

generalists and specialists. For some parameters, especially at low  $M$ , both kinds of equilibria can be evolutionarily stable alternative states, while for others, only one type of equilibrium is stable (generalist for low  $H$ , specialist for high  $H$ ). With symmetric predation, one can solve explicitly for the symmetric equilibria (with equal abundances and degrees of maladaptation in both habitats; i.e., generalists). Asymmetric equilibria are populations specialized to some extent on one habitat. At high  $M$ , some equilibria have modest differences in numbers and maladaptation between habitats. In these cases, it may not be clear whether the prey should be considered a generalist or a slight specialist.

Both predation and prey movement strongly affect whether the prey evolves to be a habitat specialist or a generalist. As an example, figure 1a shows evolutionarily stable equilibria of prey density as a function of movement rate without predation at  $H = 7$  (a moderately large difference in the phenotypic optima; this difference is 7 genetic standard deviations, 3.5 phenotypic standard deviations for a heritability of 0.25). The system always goes to one of the stable equilibria (discontinuities result where a stable equilibrium becomes unstable). The degree of maladaptation  $Y_i$  varies inversely with local density (not shown). Low movement ( $M < 0.22$ ) permits three stable equilibria: a generalist (dashed line is density in each habitat) and two specialist equilibria (solid black line is density in the habitat where the species is better adapted, gray line is its density in the other). At intermediate movement, the only stable equilibria are specialists (solid lines), but at high  $M$ , the species is a generalist (dashed lines).

Now add a generalist predator to habitat 1, where it imposes density-independent mortality at a per capita rate  $D_1$ . The symmetry is broken, so equilibria of equations (1) and (2) are found numerically. Figure 1b shows how predation in habitat 1 (at a per capita rate of 0.1) alters prey habitat use. Equilibrium densities differ between habitats. As makes intuitive sense, predation restricted to habitat 1 decreases the range of parameters favoring generalization (dashed lines) in favor of specialization to the predator-free habitat (habitat 2; gray lines), including at high movement (although for increasing  $M$ , the degree of specialization decreases). In this example, specialization to habitat 2 is evolutionarily stable at much higher movement rates than without predation; specialization to habitat 1 (solid black lines) can still occur with the predator present but only for  $M$  up to 0.46, while it occurs for  $M$  up to 0.55 without predation.

Localized predation can cause the generalist strategy to disappear for many parameter values. Predation often converts a generalist to a specialist on whichever habitat lacks predation (e.g., cf. fig. 1a, 1b for  $M > 0.55$ ). Figure 2a shows an example of the effect of gradually increasing

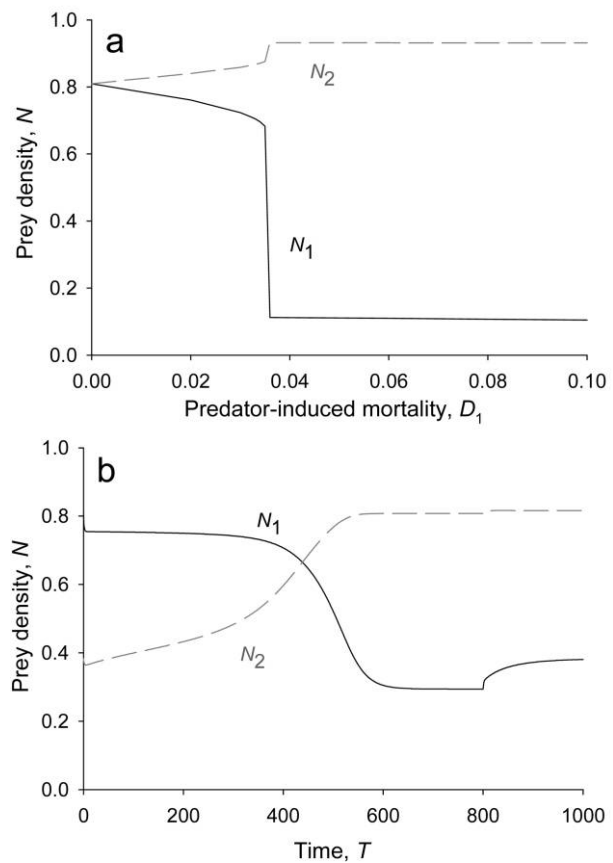


**Figure 1:** Population sizes for habitat generalists and specialists for the generalized Ronce-Kirkpatrick model when a generalist predator is absent (a) or present only in habitat 1 (b). Stable equilibria of equations (1) and (2) are shown as a function of  $M$  (per capita rate of prey movement). “Stable” here denotes evolutionary as well as ecological stability. The difference in phenotypic optima between the two habitats is  $H = 7$ ; intensity of selection is  $\Gamma = 0.1$ ; density dependence has  $\theta = 2$ . Predator-induced mortality in b is  $D_1 = 0.1$ . a, For low movement (and a tiny range of moderate movement), the prey has both stable generalist (dashed line) and specialist equilibria (solid black line for habitat to which prey is adapted, solid gray line for the other). At other movement rates, there are stable equilibria of one type or the other (specialization can be to either habitat). b, Predation eliminates the generalist equilibrium at high  $M$ , replacing it with a specialist to habitat 2 (solid gray lines). A generalist occurs only for very low  $M$  and is asymmetric (in terms of abundance and maladaptation). A specialist to habitat 1 occurs for a smaller range of movement rates than does a specialist to habitat 2.

predation (restricted to habitat 1) for a low movement rate. The prey exhibits habitat generalization in the absence of predation ( $D_1 = 0$ ). As predation increases, the generalist equilibrium remains but with shrunken numbers in habitat 1 and increased abundance in habitat 2 (as a result of lower gene flow from habitat 1). Beyond a threshold predation level, the generalist equilibrium disappears,

and the system abruptly shifts to specialization on habitat 2.

Transient increases in predation in one habitat can induce permanent shifts in the habitat to which a prey species is specialized. Figure 2b shows an example. The system starts without predation, with the prey specialized to habitat 1 (x in fig. 1a). At  $T = 0$ , a predator (imposing a mortality rate of 0.1) is added to habitat 1, shifting the balance of gene flow and selection in the prey. There is a small initial drop in abundance in habitat 1, reducing gene flow to habitat 2 and allowing the individuals there to slowly adapt. Eventually increasing movement from hab-



**Figure 2:** a, Gradual increases in predation can cause abrupt shifts in prey habitat use. Equilibrial densities in habitats 1 (solid line) and 2 (dashed line) as functions of predation in habitat 1 ( $D_1$ ).  $D_1 = 0$  has a symmetric generalist equilibrium. At sufficiently high predation, the generalist shifts to specialization to the predator-free habitat (habitat 2).  $H = 7$ ,  $\Gamma = 0.1$ ,  $\theta = 2$ ,  $M = 0.15$ . b, Example of transient increases in predation leading to permanent switches in prey habitat use. The system is initially at the asymmetric equilibrium of specialization to habitat 1; at  $T = 0$ , predation ( $D_1 = 0.1$ ) is added to habitat 1, leading to a permanent shift in prey use to habitat 2. At  $T = 800$ , this predation is removed, and the system remains specialized to habitat 2. Parameters as in a, except  $M = 0.5$ .

itat 2 (where numbers increase after an initial small drop, as fitness rises) degrades adaptation in habitat 1, depressing prey abundance and strengthening gene flow relative to selection. The prey shifts in its specialization to habitat 2 ( $x$  in fig. 1*b*). Predation is removed at  $T = 800$ , but the prey remains specialized to habitat 2. We show below that permanent shifts in prey habitat use can also occur with a dynamic predator population coupled to the prey. (In the appendix, we explore how prey habitat specialization vs. generalization is influenced by intraspecific density dependence.)

*Generalized Ronce-Kirkpatrick Model  
with a Specialist Predator*

The mortality imposed by specialist predators should not be fixed but instead varies with changes in predator numbers. For simplicity, we assume linear functional and numerical responses by the predator to its prey. If  $P_i$  is predator density in habitat  $i$ , the mortality imposed on the prey is  $D_i = \rho P_i$ , where  $\rho$  is the attack rate. The predator moves between habitats at a constant per capita rate of  $M_p$  and dies at a constant rate of  $\mu$ . With an appropriate scaling for predator numbers (see appendix), these assumptions lead to the following model for the predator:

$$\frac{dP_i}{dt} = (\rho N_i - \mu) P_i + M_p (P_j - P_i). \quad (3)$$

The full model consists of equations (1) and (2) with equation (3).

One equilibrium is for the prey to be a symmetrical habitat generalist:

$$Y_1^* = Y_2^* \equiv Y^* = \frac{HM}{2M + \Gamma}, \quad (4)$$

$$N_1^* = N_2^* \equiv N^* = \frac{\mu}{\rho}, \quad (5)$$

$$P_1^* = P_2^* \equiv P^* = \frac{1}{\rho} \left( 1 - N^{*\theta} - \frac{\Gamma Y^{*2}}{2} \right). \quad (6)$$

The quantity  $Y^*$  is unchanged by predation (cf. Ronce and Kirkpatrick 2001; Filin et al. 2008), so predation in this case alters prey densities without changing the evolutionary equilibrium. Solving for the maximum value of heterogeneity  $H$  allowing  $P^* > 0$  gives a condition for predator persistence:

$$H < \frac{\sqrt{2[1 - (\mu/\rho)^\theta]}(2M + \Gamma)}{M\sqrt{\Gamma}}. \quad (7)$$

(Predator persistence also requires  $\mu < \rho$ .) High  $H$  causes predator extinction. The prey alone persists if

$$H < H_v = \frac{\sqrt{2}(2M + \Gamma)}{M\sqrt{\Gamma}} \quad (8)$$

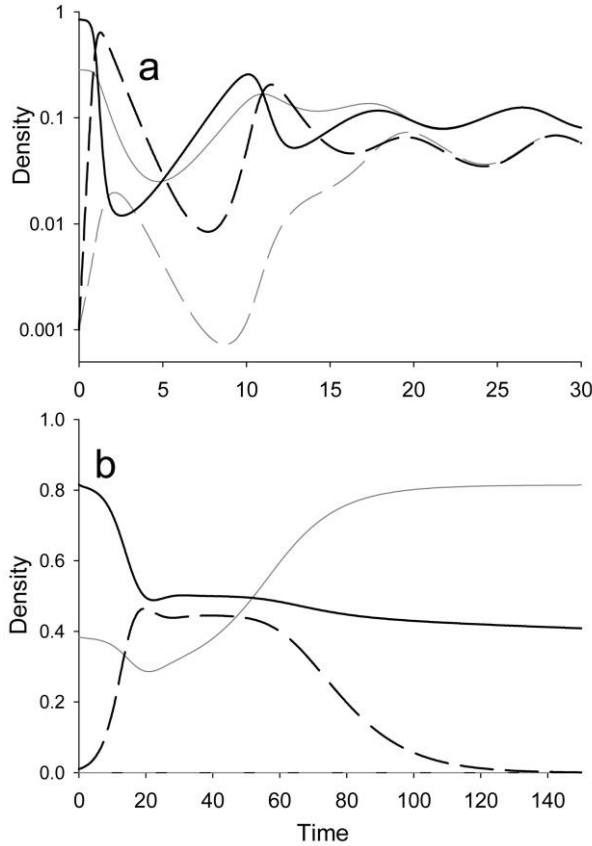
(Filin et al. 2008). Condition (7) implies condition (8). The difference between these two conditions is larger at smaller  $\theta$ . Thus, the magnitude of habitat heterogeneity tolerable by a specialist predator is less than for its prey, particularly when prey density dependence is stronger at low densities. This is because heterogeneity depresses maximal prey abundance and productivity (via maladaptive gene flow), making it harder to sustain the predator.

The appendix contains results of a stability analysis of the generalist equilibrium for stationary and moving predators and different strengths of density dependence (fig. A2). With weak density dependence (fig. A2*a*), predation often destabilizes habitat generalization, leading to specialization. With somewhat stronger density dependence (fig. A2*b*), the prey alone is less likely to be a generalist, and the predator has little impact on this. For even stronger density dependence (fig. A2*c*, A2*d*), adding a predator tends to stabilize prey habitat generalization, given that the predator can persist. The effect of a predator on the evolutionary stability of prey habitat use is thus rather complex, but in general, prey habitat generalization is more likely to be stable with a dynamic predator than without, at low prey movement, and less likely to be stable at higher prey movement or with weak density dependence.

Figure 3 demonstrates two distinct ways the introduction of a specialist, dynamic predator can alter the evolutionary habitat distribution of an initially specialized prey. In figure 3*a*, after predator colonization, prey specialization is lost and replaced by a generalist distribution. Predation can thus indirectly induce habitat generalization in its prey. By contrast, in figure 3*b*, the prey is initially specialized to habitat 1; at  $T = 0$ , a stationary predator ( $M_p = 0$ ) invades habitat 1, leading to a reversal of prey habitat specialization. This reduction in the predator's food supply leads to its own extinction, leaving behind a permanent shift in prey habitat use.

### The Evolution of a Prey Species Range along Continuous Gradients

These results demonstrate that predation can substantially alter asymmetric gene flow among habitats in a prey species and thus the evolution of habitat use. Comparable effects should occur for prey along smooth gradients. To examine this, we generalize a model of coupled demographic and evolutionary dynamics first explored by Kirkpatrick and Barton (1997) and later expanded to encompass a wider range of functional forms for density dependence (Filin et al. 2008). The strength of density dependence influences the steepness of spatial gradients



**Figure 3:** Examples of predators leading to both habitat generalization and specialization. *a*, Initially, the prey is a habitat 1 specialist. Adding a dispersing predator leads to evolution of generalization. Prey (solid lines) and predator (dashed lines) population densities in habitat 1 (black lines) and habitat 2 (gray lines) are shown.  $H = 7$ ,  $\Gamma = 0.1$ ,  $M = 0.4$ ,  $\theta = 2$ ,  $\rho = 10$ ,  $\mu = 1$ ,  $M_p = 0.01$ . *b*, A predator restricted to habitat 1 colonizes at time 0. The prey, initially a habitat 1 specialist, switches its equilibrial habitat use, remaining specialized to habitat 2 even after the predator becomes extinct.  $H = 7$ ,  $\Gamma = 0.1$ ,  $M = 0.5$ ,  $\theta = 2$ ,  $\rho = 1$ ,  $\mu = 0.5$ .

in density and thus the strength of asymmetries in gene flow.

The model tracks changes in density and the mean of a quantitative trait for a prey species along a continuous one-dimensional gradient in the trait's phenotypic optimum, assuming fixed genetic variance and spatial uniformity in maximal intrinsic growth rate, carrying capacity, and strength of stabilizing selection. We assume that the predator has linear functional and numerical responses to its prey, disperses at a constant per capita rate along the gradient, and does not evolve. These assumptions lead to the following model:

$$\frac{\partial N}{\partial T} = \frac{\partial^2 N}{\partial X^2} + \left[ U(N) - \frac{1}{2}(\bar{Z} - Z_{\text{opt}})^2 - P \right] N, \quad (9)$$

$$\frac{\partial \bar{Z}}{\partial T} = \frac{\partial^2 \bar{Z}}{\partial X^2} + 2 \frac{\partial \ln N}{\partial X} \frac{\partial \bar{Z}}{\partial X} - A(\bar{Z} - Z_{\text{opt}}), \quad (10)$$

$$\frac{\partial P}{\partial T} = \delta \frac{\partial^2 P}{\partial X^2} + (\alpha N - \mu) P \quad (11)$$

where  $T$ ,  $X$ ,  $N$ ,  $\bar{Z}$ , and  $P$  are dimensionless variables of time, space, prey population density, prey mean phenotype, and predator population density, respectively;  $\alpha$  converts consumed prey to predator reproduction, and  $\mu$  is predator mortality (for initial equations and normalization, see appendix). Both species disperse diffusively. The prey diffusion constant is absorbed into  $X$ , while  $\delta$  is the predator's diffusion rate, relative to that of its prey. Equations (9) and (10) match equations (4a) and (4b) of Filin et al. (2008), with predation added.

The first term of equation (9) describes changes in prey density due to dispersal. The second term (in square brackets) is density-dependent local growth (through the function  $U(N)$ ), reduced by maladaptation (dependent on how much the local mean phenotype differs from the local optimum  $Z_{\text{opt}}(X)$ ) as well as predation. We consider only negative density dependence, that is,  $U(N)$  monotonically decreases with  $N$ . Prey evolution (eq. [10]) combines remixing of phenotypes along the gradient by dispersal (the first two terms on the right-hand side) and stabilizing selection toward a local optimum (scaled by  $A$ , the genetic potential for adaptation, a parameter combining genetic variance and intensity of selection; Kirkpatrick and Barton 1997; Case and Taper 2000). We assume genetic variance is constant and that the selective optimum changes linearly along the gradient; that is,  $Z_{\text{opt}}(X) = BX$ .

A useful limiting case for understanding the full system is for the prey to experience no direct density dependence (e.g., early in invasion or at range margins where density is low). We briefly review what happens with the prey alone (see Filin et al. 2008) and then examine how predation alters prey invasion dynamics. Without the predator, on a shallow gradient ( $B < B_{L(\text{DINP})} = A/(2^{1/2})$ , where DINP denotes the density-independent, no-predator case), range evolution in the prey permits an unlimited, constantly growing uniform distribution at steady state, with exponential growth rate everywhere of  $R = 1$  (for details, see Kirkpatrick and Barton 1997; Filin et al. 2008). Expansion occurs at a constant velocity (similar to Skellam's [1951] classic result for homogenous environments), spatial density gradients flatten through time, and levels of maladaptation continually decrease. If the gradient is very steep ( $B > B_U = (A + 2)/(2^{1/2})$ ), the species becomes extinct. For intermediate steepness ( $B_{L(\text{DINP})} < B < B_U$ ), the steady

state prey density has a Gaussian shape, the height of which grows exponentially. Maladaptation increases toward the expanding range margin. After a phase of initial transients, the range edge (defined as density below some fixed low level) is found at position  $X(t) \propto (RT)^{1/2}$ , where  $R$  is depressed because of maladaptation. With a steeper gradient, invasion is slower as a result of maladaptive gene flow. Before this pattern emerges, one can see quasi-stable range limits (i.e., invasion lags) in the initial density-independent growth phase, even if the steady state is unlimited expansion (Filin et al. 2008).

How does a specialist predator alter prey invasion? The direct effect of predation is reduced prey growth rates, which should always reduce invasion speeds. Without prey evolution, for a reaction-diffusion predator-prey system in a homogeneous environment, Dunbar (1983; see also Owen and Lewis 2001; Murray 2003) showed that the Lotka-Volterra predator-prey system has traveling wave solutions. At a fixed location, initially a prey front is chased by a predator front, after which local densities settle down to periodic oscillations (Okubo and Levin 2001, pp. 326–328). Predation reduces the velocity of this prey wavefront.

Specialist predators also indirectly alter prey growth rates by shifting the relative strengths of gene flow and local selection (and hence the degree of local maladaptation) via impacts on prey abundance (e.g., see figs. 4, 5). Predator-prey oscillations emerge when a mobile predator is introduced at the range center of an exponentially growing invasive species moving along a gradient (fig. 4). The predator reduces prey density as it invades (fig. 4a) and in so doing creates reverse gradients in prey density behind the wavefront. The result is a traveling wave where a predator front chases a prey front (fig. 4b). Gene flow occurs toward the prey's range center, not its edge, over the zone where prey numbers decline because of increasing predation pressure. This boosts prey growth rates by facilitating local adaptive evolution. These adaptive increases in growth rates near the invasion front in turn should increase the speed of the prey invasion.

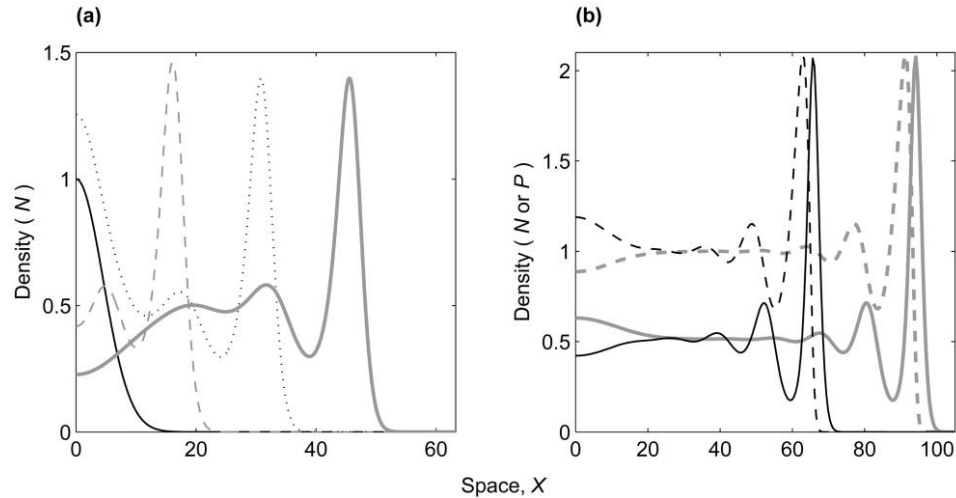
Without predation, density-independent prey growth occurs only during transient low-density phases (e.g., early in invasion). Eventually, density dependence checks growth. A prey species growing alone along a mild gradient still has a traveling wave solution (see Filin et al. 2008), where an advancing wavefront connects a region of high population density (near carrying capacity) and near complete local adaptation ( $\bar{Z} \approx Z_{\text{opt}}$ ) with a region of low population density and higher maladaptation. On steeper gradients, the range is limited. Density dependence increases the magnitude of gradient steepness needed to limit the range (i.e.,  $B_{L(\text{DDNP})}$  [where DDNP denotes density-dependent prey growth, no predator] is higher than

$B_{L(\text{DINP})} = A/(2^{1/2})$ , particularly at high  $\theta$ ; see Filin et al. 2008).

Imagine the prey has settled into a traveling wave solution ( $B < B_{L(\text{DDNP})}$ ), that is, expansion and adaptation at a constant speed, where well within the range  $N = 1$  and the phenotype is at its optimum, everywhere. Now add a predator in the range interior. The predator sees a homogeneous environment, since prey density is (locally) uniformly at  $N = 1$ . Given  $\alpha > \mu$  (required for predator persistence), a traveling wave of predators forms. After the wavefront passes, prey density is depressed from  $N = 1$  to  $N^* = \mu/\alpha < 1$ . Prey density is still  $N = 1$  at locations invaded by prey but not yet by the predator. Over a transition zone, prey density increases from  $\mu/\alpha$  to 1, and predator density drops from  $P^*$  ( $= U(\mu/\alpha)$ ; eq. [9]) to 0. Within this zone, prey maladaptation emerges because the prey density gradient generates asymmetric gene flow back toward the range center. Prey individuals in the transition zone should be better adapted to locations farther from the range center, as a result of the density gradient set up by the predator. Overall, we obtain an expanding range of the prey as it invades and adapts, with a lagged expansion of the predator range and a transient, moving zone of prey maladaptation.

For a more detailed analysis, it is useful to consider the predator invasion speed (i.e., how fast the predator expands within the occupied prey range) given by  $v = 2[\delta(\alpha - \mu)]^{1/2}$  and the length scale of the predator spillover effect (i.e., how far a predator penetrates areas where prey are absent or scarce), which is scaled by  $\xi = (\delta/\mu)^{1/2}$ . Another key compound variable is equilibrium prey density,  $N^* = \mu/\alpha$ , which measures the efficacy of the predator in limiting prey. Figure 5 shows examples (with logistic growth) of a predator tracking a prey adapting to a gradient. If steady state invasion speed of the prey exceeds  $v$ , the predator front never reaches the prey front, and the distance between the two increases indefinitely. This can occur along a shallow gradient where prey adaptation is not markedly impeded by gene flow (e.g., fig. 5a). On a steeper gradient (see fig. 5b), prey invasion slows and the predator front now catches up to the prey, with a constant spatial lag. The range is still unlimited, but the rate of prey expansion is greatly slowed. Even steeper gradients lead to evolutionarily stable range limitation of the prey (and the predator) caused by maladaptive gene flow in the prey (see fig. 5c). Hence, the ecological effect of predator "spillover" causing stable prey range limits shown by Hochberg and Ives (1999) is magnified by the indirect effect of predation on prey adaptive evolution.

By depressing prey numbers, predation can alter the strength of prey density dependence (eq. [9]). The effects of predation on invasion dynamics and conditions for prey range limitation depend on the form of density depen-



**Figure 4:** Interplay of predation and local adaptation in prey can create reversals in prey abundance along a gradient, facilitating invasion. *a*, Prey density across space for density-independent predator-prey dynamics at four time points in an invasion by both species:  $T = 0$  (solid black line), 14 (dashed gray line), 28 (dotted line), and 43 (solid gray line). Parameters:  $\alpha = 0.7$ ,  $\delta = 0.7$ ,  $\mu = 0.35$ ,  $A = 0.4$ ,  $B = 0.34$ . Initial conditions at  $T = 0$  for both prey and predator are narrow Gaussian distributions centered at  $X = 0$ , with maximum prey density of 1 and maximum predator density of 0.01. As the prey expands, its abundance is maximal near the range margin, where it escapes predation; predation can reverse density gradients, altering gene flow away from the invasion front. *b*, Heterogeneity in dynamics along a gradient. Prey (solid lines) and predator (dashed lines) densities across space at  $T = 62$  (black lines) and  $T = 89$  (gray lines). Parameters and initial conditions as in *a*, except  $\delta = 0.35$  and  $\mu = 0.36$ . Note that densities are scaled separately for each species (for details, see appendix in the online edition of the *American Naturalist*). The predator lags the prey; the magnitude of population oscillations varies greatly across the range, with instability pronounced near the range edge but with stabilized dynamics toward the interior.

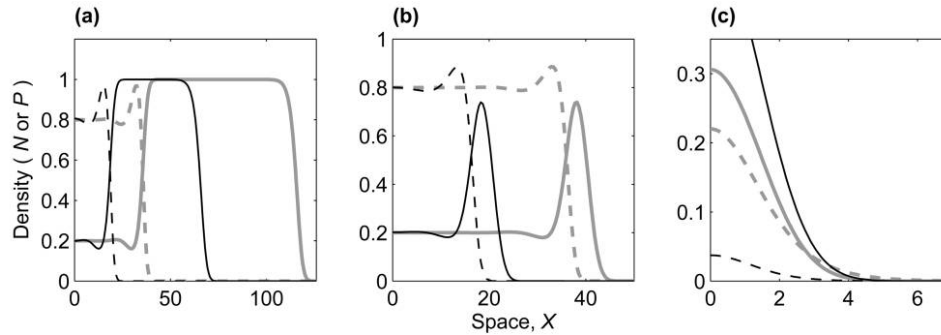
dence. Whether predation slows the asymptotic rate of prey invasion depends on gradient steepness, the evolutionary potential  $A$ , and predator dispersal. Using the theta-logistic model ( $U(N) = 1 - N^\theta$ ) in equation (9), we carried out numerical studies to assess the effect of different forms of density dependence. We start with the limiting case of no prey density dependence. Figure 6*a* shows for a slice of parameter space that predation can enhance prey invasion (in this example, predator spillover is rather weak). Between the solid and lower dashed lines, predation permits the growth rate to reach its maximal value ( $R = 1$ ), when without predation growth rate would be constrained below this by gene flow. Thus, predators sometimes enhance the rate of invasion by prey.

Density dependence permits the prey to settle into a spatially bounded range at evolutionary and ecological equilibrium. Figure 6*b* and 6*c* shows examples of how predation alters the transition between range-limited evolution, and an unlimited range, with prey density dependence. The solid gray line in figure 6*b* describes range limitation for the prey alone. When predation has only a minor effect on prey abundance (dotted line), there is little effect on the transition line. When predation has a more substantial impact on prey numbers, increases in spillover lead to prey range limitation in a swath of parameter space

where otherwise the range would be unlimited (dashed and dotted-dashed lines). In such cases, if a prey species is expanding so that on its own it would occupy the entirety of a gradient, predator invasion can first halt and then reverse this invasion, so that the range collapses to a much narrower slice of the gradient (fig. A4 provides a numerical example).

Conversely, when predation has a strong impact on prey numbers but weak spillover (solid black line), prey ranges may expand that otherwise would be constrained by gene flow. Finally, figure 6*c* assumes strong density dependence at low prey numbers and moderate reduction of prey numbers. In this case, predation has a relatively minor effect of increasing the range of parameter space where prey can expand along all of a gradient. However, even in these cases, a predator may strongly influence the realized equilibrium range of its prey. If a prey species has settled into a limited-range equilibrium, introducing a specialist predator in just a portion of that range may induce transient dynamics, after which the prey settles into a new limited-range equilibrium, with a different limit along the gradient. If the predator is constrained in the part of the gradient it can occupy, for reasons other than prey availability, it may be vulnerable to driving itself to extinction as its prey shifts largely out of reach (a gradient analogue of the sce-





**Figure 5:** Predator-prey invasion along gradients of increasing steepness. Prey densities are solid lines, predator densities are dashed lines. The gradient becomes steeper (i.e., higher  $B$ ) from *a* to *c*. Initial conditions are as in figure 4*a*. Prey density dependence is logistic ( $\theta = 1$ ). *a*, Both prey and predator expand along a gentle gradient, with the prey expanding faster than the predator (densities shown for two times,  $T = 58$  [black lines] and  $T = 110$  [gray lines]).  $\alpha = 0.5$ ,  $\delta = 0.1$ ,  $\mu = 0.1$  (giving  $\nu = 0.4$ ,  $\xi = 1$ ,  $N^* = 0.2$ ),  $A = 0.5$ ,  $B = 0.35$ . *b*, For a steeper gradient ( $B = 0.7$ ), prey and predator fronts move at the same rate (shown for  $T = 67$  [black lines] and  $T = 134$  [gray lines]). *c*, Along an even steeper gradient ( $B = 1.2$ ), both species settle into a limited range (shown for  $T = 15$  [black lines] and  $T = 68$  [gray lines]), with evident predator spillover into the prey range margin.

nario of fig. 3*b*). Such examples suggest that even if predation does not alter the fact that gene flow constrains the range of the prey, predation can strongly affect where, exactly, range limits occur.

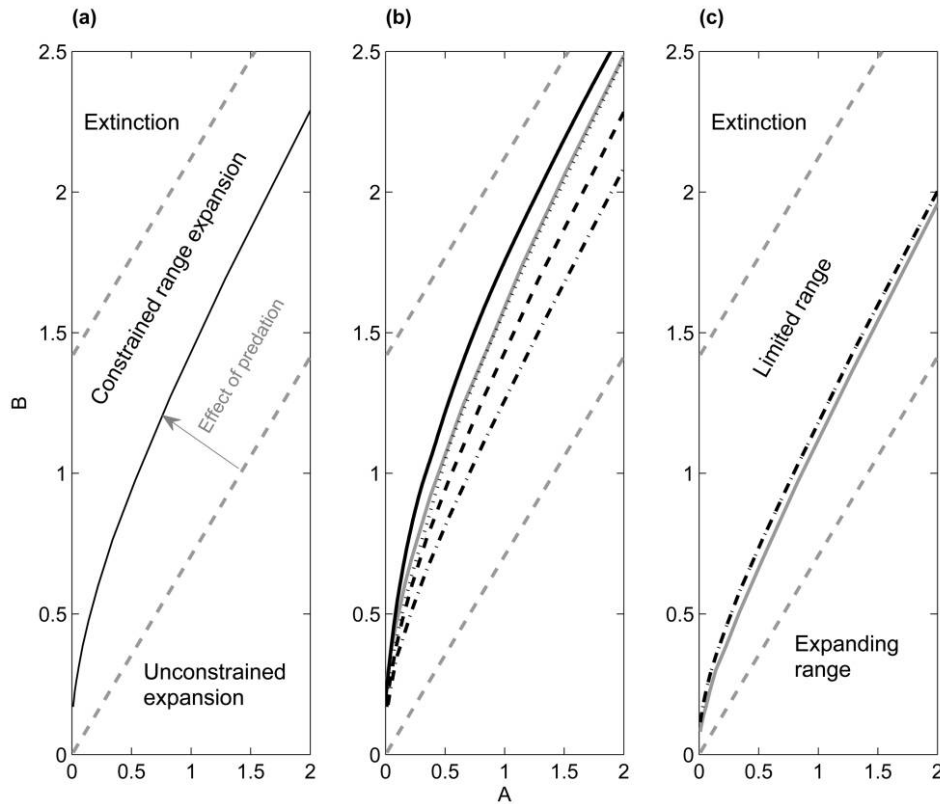
### Discussion

Interspecific interactions can influence species' ranges and habitat specialization both directly and indirectly via evolutionary mechanisms such as gene flow and selection. The ecological impact of predation is usually to constrain the habitat distribution and geographical ranges of prey. Our models show that evolutionary responses of prey to spatial variation in selection, coupled with gene flow, can imply a rich array of alternative outcomes, including dramatic shifts in habitat use and expansion of prey species along gradients where otherwise ranges would be limited (as conjectured by Holt and Barfield [2009]). Facilitative effects of predation on prey ranges are particularly likely early in invasions, at least if predator dispersal is rather modest and prey growth is largely density independent. Alternatively, sometimes predators that are effective dispersers sharpen the impact of gene flow on prey range limitation. Such predators can even induce prey range collapse to a much greater extent than expected on purely ecological grounds.

We have deliberately considered two idealized extremes of landscape structure: a pair of discrete habitats and smoothly varying, continuous gradients. There are many commonalities in the evolutionary patterns emerging in both landscapes. For instance, in general, an increase in habitat heterogeneity in both landscapes makes the restriction of range and habitat breadth by gene flow more

likely, with and without predation. Introducing predators in both cases can lead to dramatic shifts in habitat use or the position of a range edge. But there are differences in these scenarios as well. The continuous gradient case can, for example, exhibit dynamics with no obvious parallel for the discrete habitat case, such as predator and prey wavefronts either moving at the same rate or with prey outracing the predator across a landscape. Another difference is that qualitative changes in the range seem to be more likely in the two-patch system because of the frequent presence of multiple stable equilibria. In this case, adding a predator can dramatically shift the system from one stable state to another. However, it is difficult to quantify this qualitative impression. The discrete habitat case has only a small number of possible qualitative equilibria (a prey may be specialized to one habitat or the other, or a complete generalist, or in between). Along a continuous gradient, there are an infinite number of alternative stable range limits, depending on initial conditions.

The impact of the rate of movement on the evolution of habitat specialization and range dynamics is complex (Holt et al. 2005; Garant et al. 2007; Kawecki 2008) and is made more so by predation. For instance, for the discrete landscape with a generalist predator in one habitat, for a prey species with a low  $\theta$  to be either a specialist to the habitat with predation or a generalist, its movement rate must be quite low (fig. 1*b*, left side). This result sharply contrasts with what is expected if the prey is alone, where at high movement only habitat generalization is stable (Filin et al. 2008). This effect of habitat-specific predation is altered at high  $\theta$ . Now generalization can again be evolutionarily stable at high but not intermediate movement rates (see fig. A1).



**Figure 6:** Predation and density dependence influence the potential for prey range limitation by gene flow along a gradient. Equilibrial states are depicted in terms of  $B_L$  curves, the gradient steepness  $B$  needed for range constraint as a function of the evolutionary potential for adaptation,  $A$ . In *a–c*, the bottom gray dashed line is the  $B_L$  curve for density-independent, single-species dynamics. The top gray dashed line is an extinction threshold; above it, the gradient is too steep for prey persistence. Between those two lines, either range expansion (below a line) or a limited range (above) may occur. *a–c* all have predation but differ in the presence and functional form of prey density dependence. *a*, Density independence ( $\alpha = 1.25, \delta = 0.25, \mu = 0.25; \nu = 0.5, \xi = 5, N^* = 1$ ). The solid line shows how predation expands the potential for prey range expansion at its maximal rate,  $R = 1$ , unconstrained by gene flow. Predation increases the  $A$ - $B$  combinations where invasion occurs at maximal speed. *b*, Density dependence with  $\theta = 8$  (weak at low  $N$ ) for several cases (lines in *b* and *c* indicate range limitation): prey alone (solid gray line); predator-prey dynamics (dashed line;  $\nu = 1, \xi = 1, N^* = 0.2$ ); weak predation:  $N^* = 0.9$  (dotted line;  $\nu = 1, \xi = 1$ , hardly distinguishable from the single-species case); the length scale of predator spillover  $\xi$  is increased (dotted-dashed line;  $\nu = 1, \xi = (10)^{1/2}, N^* = 0.2$ ); highly effective predation:  $N^* = 0.01, \xi = 0.1$ , while keeping  $\nu = 1$  (solid line). *c*, Density dependence with  $\theta = 0.25$  (strong at low  $N$ ): prey alone (solid gray line); with predator (dotted-dashed line;  $\nu = 1, \xi = 1, N^* = 0.2$ ).

We have made simplifying ecological assumptions that should be relaxed in future studies, such as linear functional responses. Predators with saturating functional responses can generate unstable dynamics (Case and Taper 2000). The consequences of unstable dynamics for the interplay of gene flow and selection in defining range limitation have not been addressed in the literature. Movement in our models is symmetric and random. Yet physical transport processes can be asymmetric, and many organisms are not at the mercy of air currents and water flows but directionally disperse. Asymmetric movement, including habitat selection, can strongly alter the direction of evolution in heterogeneous landscapes (Kawecki and Holt 2002; Holt 2003) and would doubtless influence our conclusions.

Interspecific interactions can create historical contingencies in determining species ranges and do so in a way difficult or impossible to discern by examining current distributions. For instance, in the discrete habitat model, predation can induce permanent switches in habitat specialization of a prey species, even if the predator then disappears (fig. 3*b*), and similar effects arise in the gradient model. In a spatially closed community, evolutionary responses by a victim to a specialist natural enemy seem rather unlikely a priori to lead to the enemy's extinction. As a prey species evolves to withstand predation, the predator declines, and selection on the prey to avoid predation weakens. But in a heterogeneous landscape, prey evolution can lead to predator extinction if the prey utilizes habitats

inaccessible to a predator. Nuismer and Kirkpatrick (2003) explored a model in which a host and a specialist parasite coevolve in a landscape of two discrete habitats. They assumed that the parasite depends demographically on the host but that the host, conversely, is not regulated by parasitism (and has a fixed distribution). They showed that host gene flow significantly affects the parasite range and, in some circumstances, even leads to parasite extinction. In our model, by contrast, only one species (the prey) evolves, and its density is strongly impacted by predation. As in the model explored by Nuismer and Kirkpatrick (2003), prey habitat evolution in response to specialist predation can lead to predator extinction. Such evolutionary mechanisms for the “ghost of predation past” (Brown and Vincent 1992) could provide a substantial source of historical contingency in community assembly and the realized ranges of prey or host species.

We made a number of simplifying assumptions about evolution, such as the assumption that genetic variation is fixed. If genetic variation freely evolves, negative effects of gene flow on local adaptation can be counteracted by dispersers providing genetic variation (Gomulkiewicz et al. 1999; Barton 2001). Range limits in this case are more likely to reflect purely organismal and ecological factors, rather than gene flow. Conversely, our models ignore effects of drift and demographic stochasticity, which deplete genetic variation. When prey are productive and predators have saturating functional and numerical responses, large-amplitude limit cycles arise. This could force recurrent bottlenecks, amplifying the impact of genetic drift even if average abundance is high. In general, the implications of unstable population dynamics for range limits are poorly understood. Moreover, we have ignored evolution in the prey to reduce predation itself (via evolution of the attack parameter) and coevolutionary responses by the predator. Coevolutionary dynamics can lead to complex mosaics (Thompson 2005), and the processes we have explored could contribute to this complexity. Prey with a long evolutionary history of exposure to particular predators may evolve successful adaptations to escape high rates of predation. Effects of predation on limiting a prey's range should be unimportant if the prey has evolved effective counter defenses so that prey densities are not significantly depressed by predation (until the predator then evolves in response). A more detailed account of generalist predator impacts on the evolution of prey ranges will also need to consider evolution and coevolution across multiple prey species, which can experience apparent competition via shared predation with the focal prey species (Case et al. 2005).

Possibly the most important evolutionary assumption in our models is that gene flow hampers local adaptation. Though long assumed (e.g., Mayr 1963), this may not

always hold (Gomulkiewicz et al. 1999; Barton 2001). Interspecific interactions in isolation can lead to range limits with a strong evolutionary component. Price and Kirkpatrick (2009) have recently shown that at times range limits generated by competition can be evolutionarily stable, even if gene flow does not constrain local adaptation, because of how interspecific competition in effect maintains stabilizing selection on resource use. A competitor can constrain the shape of the adaptive landscape, so the optimal phenotype is spatially uniform—so gene flow cannot perturb local adaptation. Holt (1997*b*) sketched a resource-consumer model that implied that spatial variation in resource supply rates (including effects of resource competition) can lead to range limits without altering the optimal trade-off between consumption rates and mortality rates. In this model, stabilizing selection is spatially uniform, so again gene flow should not be a significant determinant of range limits. It would be illuminating to craft comparable models including predation.

Despite these cautionary remarks, this study strongly suggests that predators—even specialist predators—can profoundly affect habitat use and geographical distributions in their prey via a modulation of the effect of gene flow on local adaptation. Several empirical arenas we suggest could provide testing grounds and applications for our theories.

First, our extension of the Ronce-Kirkpatrick model (Ronce and Kirkpatrick 2001) to include predation could be used to analyze patterns of host plant use in herbivorous insects, particularly when host plants are spatially segregated in different habitats. Many insect ecologists have argued that natural enemy impacts have a strong influence on insect community structure (Jeffries and Lawton 1984). Our models suggest that predation could have a particularly strong impact on host plant specialization if their prey have weak density dependence at low densities.

Second, an intriguing finding in island biogeography is a pattern of evolutionary habitat shifts called the taxon cycle. Wilson (1959) suggested that southwest Pacific ants colonized coastal habitats and then shifted to interior habitats such as montane forests. Such evolutionary shifts can be driven by asymmetric gene flow and local adaptation in a single species (Filin et al. 2008) or competitive displacement by later colonists (the explanation favored by Wilson [1965]; see also MacLean and Holt [1979]), but predation and parasitism could also play a role. The dynamics of figure 3*b* can occur during island community assembly and evolution. The solid lines are prey density in two island habitats, one near the sea (habitat 1) and the other interior (habitat 2). The prey, initially better adapted to habitat 1, occurs in habitat 2 but remains maladapted there as a result of gene flow from habitat 1. A specialist predator invades but is constrained to habitat 1

(e.g., by thermal adaptation). Reducing prey numbers in habitat 1 shrinks gene flow into habitat 2, facilitating adaptation and increased abundance there. A backflow of dispersers to habitat 1 grows; gene flow degrades adaptation there, reducing local growth. The predator in habitat 1 experiences an inexorable decay in its environment, with declining prey abundance and productivity eventually leading to predator extinction. The net effect is that the taxon cycle has turned a notch, with the island colonizer having shifted to the interior habitat. Ricklefs and Bermingham (2007) suggest that pathogens introduced with sister taxa may drive island extinctions, preventing secondary sympatry. Our results suggest that habitat shifts may provide an alternative outcome to pathogen and predator introductions, at least if natural enemies are constrained to the initial habitat range of the victim.

Third, one potential source of application of our results is invasion biology. Sometimes species show puzzling lags after establishment, remaining in a small area for a while before range expansion. There are a number of potential explanations for this (Crooks 2005), and our theoretical results suggest yet another.

Finally, if gene flow constrains a prey species to remain specialized to a particular habitat, the introduction of a biological control agent may have at times the perverse consequence of unleashing the target species to expand over a much larger landscape. We do not argue that this is the norm but, rather, that it is one possibility that should be kept in mind in biological control. Our models suggest that this outcome is most likely if the natural enemy is an effective local control agent but a rather poor disperser, and if the target species has weak density dependence at low numbers. Failed biological control attempts could possibly occur for this reason (though of course there are many other reasons for such failure).

Our results show that the interplay of predation and evolutionary dynamics in prey species in theory can lead to a rich range of potential effects, such as shifts in habitat use and expansions in prey range size, that go well beyond considering the purely ecological effects of predation. Beyond further theory development, we suggest that empiricists interested either in the basic biology of range limits or in applied questions based on such understanding—such as controlling invasions, crafting biological control programs, or projecting impacts of climate change—should consider the intertwined roles of trophic interactions and evolution in determining range limits.

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