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Appendix from R. D. Holt et al., "Predation and the Evolutionary Dynamics of Species Ranges"

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Model Derivations and Additional Results

Here, we provide technical details that are useful for understanding the model derivation and analyses presented in the main text and expand on some additional points.

The Generalized Ronce-Kirkpatrick Model with Predation

Ronce and Kirkpatrick (2001) derived the model on which our first model (eqq. [1], [2]) is based, and our equation for evolution is identical to theirs. Our ecological model differs because we include nonlinear density dependence and mortality due to predation. It is useful to express the ecological model in terms of more familiar ecological and evolutionary parameters to point out a difference in the normalization that we employ, because we include nonlinear density dependence. Two habitats (denoted by $i = 1, 2$) have equal area, with prey at density n_i in habitat i (table A1 summarizes variables and parameters). The trait under selection has a normal distribution with mean $\bar{z}_i$ in habitat i (phenotypic variance σ_p^2 is assumed constant and equal in both habitats). An individual of phenotype z_i in habitat i has fitness (per capita growth rate) of $r_0[1 - (n_i/K)^\theta] - \gamma(z_i - z_{\text{opt},i})^2/2 - d_i$, where $z_{\text{opt},i}$ is the optimum phenotype in habitat i, K is carrying capacity, r_0 is intrinsic growth rate at low density of an individual with the optimum phenotype, γ is the strength of stabilizing selection, θ is a density dependence parameter (all assumed equal across habitats), and d_i is per capita predator-induced mortality in habitat i. (This expression with $\theta = 1$ and $d_i = 0$ matches one in Ronce and Kirkpatrick 2001.) The form of density dependence is theta logistic (Sæther and Engen 2002; Filin et al. 2008). The larger θ is, the weaker density dependence is at low density. Without predation, a population with its mean phenotype at the optimum for habitat i has a maximal growth rate of $r' = r_0 - \gamma\sigma_p^2/2$ (the second term reflects mean fitness depression due to phenotypic variation around the optimum). We scale time by maximal growth rate ($T = r't$) and normalize prey densities with $N_i = (n_i/K)(r_0/r')^{1/\theta}$. We express mean phenotypes as normalized deviations from the habitat optima ($Y_i = |\bar{z}_i - z_{\text{opt},i}|/\sigma_g$, where σ_g^2 is genetic variance, which is used to normalize phenotypes as it determines their rate of change). The per capita rate of dispersal between the two habitats is m (i.e., the probability an individual moves in a short time interval Δt is $m\Delta t$), again scaled by r'. The dimensionless parameters of the model are (1) $\Gamma = \sigma_g^2\gamma/r'$, strength of selection; (2) $H = (z_{\text{opt},2} - z_{\text{opt},1})/\sigma_g$, difference in phenotypic optima; and (3) $M = m/r'$, movement rate. Predator-induced mortality is scaled by r', so $D_i = d_i/r'$ (this can be a fixed parameter or dynamic; we consider both in the main text).

With these rescalings, the equation for prey population dynamics in habitat i is

$$\frac{dN_i}{dT} = (1 - N_i^\theta)N_i - \frac{\Gamma}{2}Y_i^2N_i + M(N_j - N_i) - D_iN_i, \tag{A1}$$

which is equation (2) in the main text.

We assume that predator numbers (p_i) follow a Lotka-Volterra model, in which a specialist predator has linear functional and numerical responses to its prey—with attack rate a', conversion of prey density to new predator density b', and predator mortality rate m'—and moves at a constant rate (m_p) between habitats. Linearity in the predator functional and numerical responses ensures a stable predator-prey interaction.

After rescaling, the model for the predator dynamics is as follows:

$$\frac{dP_i}{dT} = (\rho N_i - \mu)P_i + M_p(P_j - P_i), \tag{A2}$$

(eq. [3] in the main text), where $P_i = a'p_i/r'$ is normalized predator density in habitat i and $\rho = a'b'K(r/r')^{1/\theta}$, $\mu = m'/r'$, and $M_p = m_p/r'$ are normalized attack, mortality, and movement rates, respectively, of the predator. Mortality of the prey due to predation is $D_i = \rho P_i$.

Generalized Ronce-Kirkpatrick Model with Weak Density Dependence at Low Prey Density

Filin et al. (2008) showed that in the generalized Ronce-Kirkpatrick model (Ronce and Kirkpatrick 2001), making density dependence weaker at low densities (i.e., increasing θ) facilitates habitat generalization. This expectation is retained with habitat-specific predation, but over some movement rates, habitat specialization more readily emerges. Figure 1*b* in the main text shows an example for $\theta = 2$. Figure A1 shows another example for $\theta = 8$ and high predation in habitat 1 ($D_1 = 0.3$). This value for θ implies that density dependence (measured as marginal changes in per capita growth rate, with small changes in prey density) is very weak at low densities but becomes progressively stronger as population numbers approach (or exceed) the carrying capacity. At low movement, alternative strategies include both specialization (to either habitat) and generalization. At high movement, only habitat generalization is evolutionarily stable. In comparison, with stronger density dependence (fig. 1*b*), at high movement only habitat specialization is evolutionarily stable. At intermediate movement, in figure A1, the only stable strategy is for the prey to specialize on habitat 2; with lower mortality (e.g., $D_1 = 0.1$; not shown), the generalist is stable for all movement rates. The higher mortality now makes habitat 1 so poor that the only stable strategy for moderate M is specialization to habitat 2. How predation influences habitat specialization and generalization in its prey—and the potential for alternative evolutionary states—thus depends in a rather complex way on both the rate of prey movement between habitats and the pattern of density dependence in the prey species itself.

Stability of Prey Habitat Generalists in the Ronce-Kirkpatrick Model with Specialist Predation

Local stability analysis can be used to gauge the evolutionary stability of the generalist equilibrium of the generalized Ronce-Kirkpatrick model with a predator. For simplicity, we present the final results of our analyses. Without predation (Filin et al. 2008), the condition for habitat generalization is

$$H < H_{\max} = \frac{H_v\sqrt{2M + \theta}}{\sqrt{4 + \theta}} = \frac{\sqrt{2(2M + \theta)}(2M + \Gamma)}{M\sqrt{\Gamma(4 + \theta)}}. \tag{A3}$$

With predation, the two conditions required for stability are

$$H < \frac{2M + \Gamma}{M}\sqrt{\frac{2\{\mu[1 - (\mu/\rho)^\theta] + 2\theta(\mu/\rho)^\theta M_p + 4MM_p\}}{\Gamma(8M_p + \mu)}} \tag{A4}$$

(if $M_p = 0$, this matches the predator persistence criterion in the main text) and

$$H < \frac{2M + \Gamma}{M}\sqrt{\frac{2[\mu(1 - N^{*\theta}) + 2\theta N^{*\theta}M_p + 4MM_p + \Gamma(\theta N^{*\theta} + \Gamma + 2M + 2M_p)]}{\Gamma[\mu + 4\Gamma(\theta N^{*\theta} + \Gamma + 2M)/(\theta N^{*\theta} + 2M_P + 2M)]}}, \tag{A5}$$

where $N^* = \mu/\rho$. The pertinent stability limit is the smaller of these expressions. Generally, the second condition holds at low M_p, while the first applies at high values; the derivative of the maximum H for stability is discontinuous at that value of M_p for which the controlling expression switches. Examples of the persistence and stability limits for different degrees of density dependence are shown in figure A2. In all cases, generalist equilibria are stable below the corresponding curve and unstable above. Broadly speaking, generalist equilibria can stably persist mainly for quite high—or rather low—rates of movement, unless habitat heterogeneity is quite modest. When the prey is alone, changes in the nonlinear character of its density dependence have an effect on the stability of habitat generalization. Adding a predator can alter the range of parameters that permit stable habitat generalization. This is particularly true for $\theta = 8$, which is quite weak density dependence at low numbers. For such prey, predation makes it much less likely that habitat generalization is evolutionarily stable.

Normalization of the Generalized Kirkpatrick-Barton Model with Predation

In the Kirkpatrick-Barton model (Kirkpatrick and Barton 1997), the original dynamical equations are as follows:

$$\frac{\partial n}{\partial t} = D\frac{\partial^2 n}{\partial x^2} + n\left[r_0\left(1 - \frac{n}{K}\right) - \frac{\gamma\sigma_p^2}{2} - \frac{\gamma}{2}(\bar{z} - z_{opt})^2\right], \tag{A6}$$

$$\frac{\partial \bar{z}}{\partial t} = D\frac{\partial^2 \bar{z}}{\partial x^2} + 2D\frac{\partial \ln(n)}{\partial x}\frac{\partial \bar{z}}{\partial x} - \gamma\sigma_g^2(\bar{z} - z_{opt}). \tag{A7}$$

With a specialist predator and theta-logistic density dependence, these become

$$\frac{\partial n}{\partial t} = D\frac{\partial^2 n}{\partial x^2} + n\left\{r_0[1 - \left(\frac{n}{K}\right)^{\theta}] - \frac{\gamma\sigma_p^2}{2} - \frac{\gamma}{2}(\bar{z} - z_{opt})^2 - a'p\right\}, \tag{A8}$$

$$\frac{\partial \bar{z}}{\partial t} = D\frac{\partial^2 \bar{z}}{\partial x^2} + 2D\frac{\partial \ln(n)}{\partial x}\frac{\partial \bar{z}}{\partial x} - \gamma\sigma_g^2(\bar{z} - z_{opt}), \tag{A9}$$

$$\frac{\partial p}{\partial t} = D_p\frac{\partial^2 p}{\partial x^2} + p(a'b'n - m'), \tag{A10}$$

where D and D_p are diffusion coefficients for the prey and predator, respectively (i.e., both species disperse at random but possibly at different rates); z_{opt} (a function of x) is the optimum phenotype, which varies along the gradient; K is the carrying capacity; r_0 is the intrinsic growth rate (fitness at low density) of the optimum phenotype; γ is the strength of stabilizing selection; σ_g^2 is the genetic variance; and σ_p^2 is the phenotypic variance. The attack rate of the predator on the prey is a', its growth per unit prey consumption is b', and its mortality rate is m. A prey population with its mean phenotype at the local optimum has a maximal realized growth rate of $r' = r_0 - \gamma\sigma_p^2/2$; the actual growth rate is lower than this value if the local population is maladapted, experiences density dependence, or is attacked by the predator. Equations (9)–(11) in the main text are obtained by rescaling the variables as follows: $T = r't$, $N = (n/K)(r_0/r')^{1/\theta}$, $Z = \bar{z}(\gamma/r')^{1/2}$, $X = x(r'/D)^{1/2}$, and $P = a'p/r'$. Finally, the dimensionless parameter A, the genetic potential for adaptation, is $A = \gamma\sigma_g^2/r'$; the scaled prey-to-predator conversion coefficient is $\alpha = a'b'K(r'/r_0)^{1/\theta}/r'$; $\mu = m'/r'$; and $\delta = D_p/D$.

Comment on Steady-State Exponential Growth in the Density-Independent Case

For $B > B_{L(DINP)} = A/(2^{1/2})$, the Gaussian geographic distribution of the prey is given by

$$N(X, T) = N_0\exp\left(-\frac{1}{2}CX^2 + RT\right), \tag{A11}$$

where $C = (B - B_{L(DINP)})/(2^{1/2})$ is inversely related to the width of the Gaussian and $R = (B_U - B)/(2^{1/2})$ is the steady-state exponential growth rate. The growth rate is less than in a homogeneous environment (where $R = 1$), because there is a permanent degree of maladaptation due to gene flow. For $B < B_{L(DINP)}$—that is, the uniform distribution steady state—the steady-state exponential growth rate is $R = 1$.

Dynamical Features of Invasion with Predation and an Evolving Prey

For a nonspatial, nonevolving system (the classical Lotka-Volterra predator-prey model with a linear functional response), the stationary point $N^* = \mu/\alpha$, $P^* = 1$ is neutrally stable, with angular frequency of oscillations of $\mu^{1/2}$. Even with prey evolution, there can be oscillations after predator colonization (in the full system of eqq. [9]–[11]); the angular frequency of these oscillations is approximately $(R\mu)^{1/2}$. A steeper gradient that lowers prey growth rates due to maladaptive gene flow should have a lower frequency of oscillations during the initial phases of invasion. Numerical solutions of the model show this behavior (fig. A3, which shows dynamics at the range center following predator introduction). These numerical studies also show that the oscillations dampen, but often very slowly.

Transients, Quasi-Stable Range Limits, and Predator-Induced Shifts in Prey Range/Niche

Here we elaborate on some points touched on in the main text. We have previously shown that quasi-stable range limits can arise during the initial phase of a species invasion (Filin et al. 2008), because of the initially density-independent population dynamics expected when species density is low. At later stages of invasion, populations near the site of origin should approach carrying capacity, and density dependence can then weaken the impact of gene flow, permitting the range to begin to expand. Similar phenomena arise with predation. For density-independent prey dynamics (fig. 6*a*) and low-θ density-dependent dynamics (fig. 6*c*), there exists a region in *A*-*B* parameter space in which with no predator or at low predator densities the prey is initially range limited (between the solid line and bottom dotted line in fig. 6*a* and between the gray solid line and gray dashed line in fig. 6*c*). However, once a predator population is introduced and grows significantly, it releases the prey from maladaptive asymmetric gene flow by eliminating or flattening prey density gradients. Consequently, the prey dynamics change from exhibiting a (quasi-stable) limited range to steady-state invasion and range expansion.

However, for high θ, one can observe an opposite dynamical behavior (e.g., see fig. 6*b*). The introduction of specialist predation now slows—and may even reverse—prey invasion. When the predator invasion speed v exceeds the steady-state prey invasion speed expected without predation, the predator invasion front can catch up with the prey front, even if initially the prey front lies far ahead. This transient catching-up phase of the predator-prey system can then give way to a limited-range steady state (of both prey and predator) for a sufficiently steep environmental gradient. In effect, the predator catches up with its prey and causes the whole system to collapse into a confined range, much smaller than the range of both prey and predator achieved during the transient phase (e.g., see fig. A4).

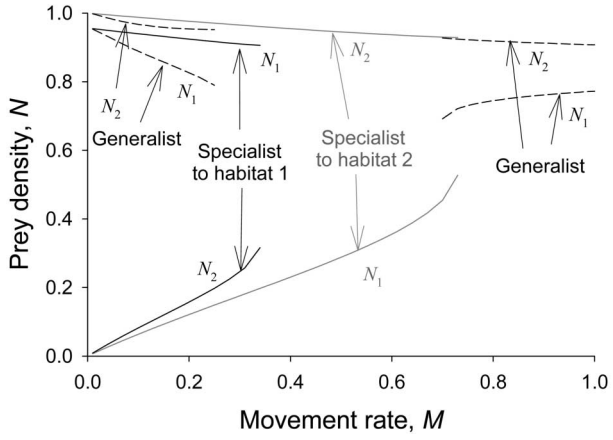

Figure A1: Evolutionary equilibria in prey habitat use as a function of prey movement rate, with a predator-induced mortality of 0.3 in habitat 1. Parameters are $H = 7$, $\Gamma = 0.1$, $\theta = 8$, that is, weak density dependence at low prey density (cf. fig. 1*b*).

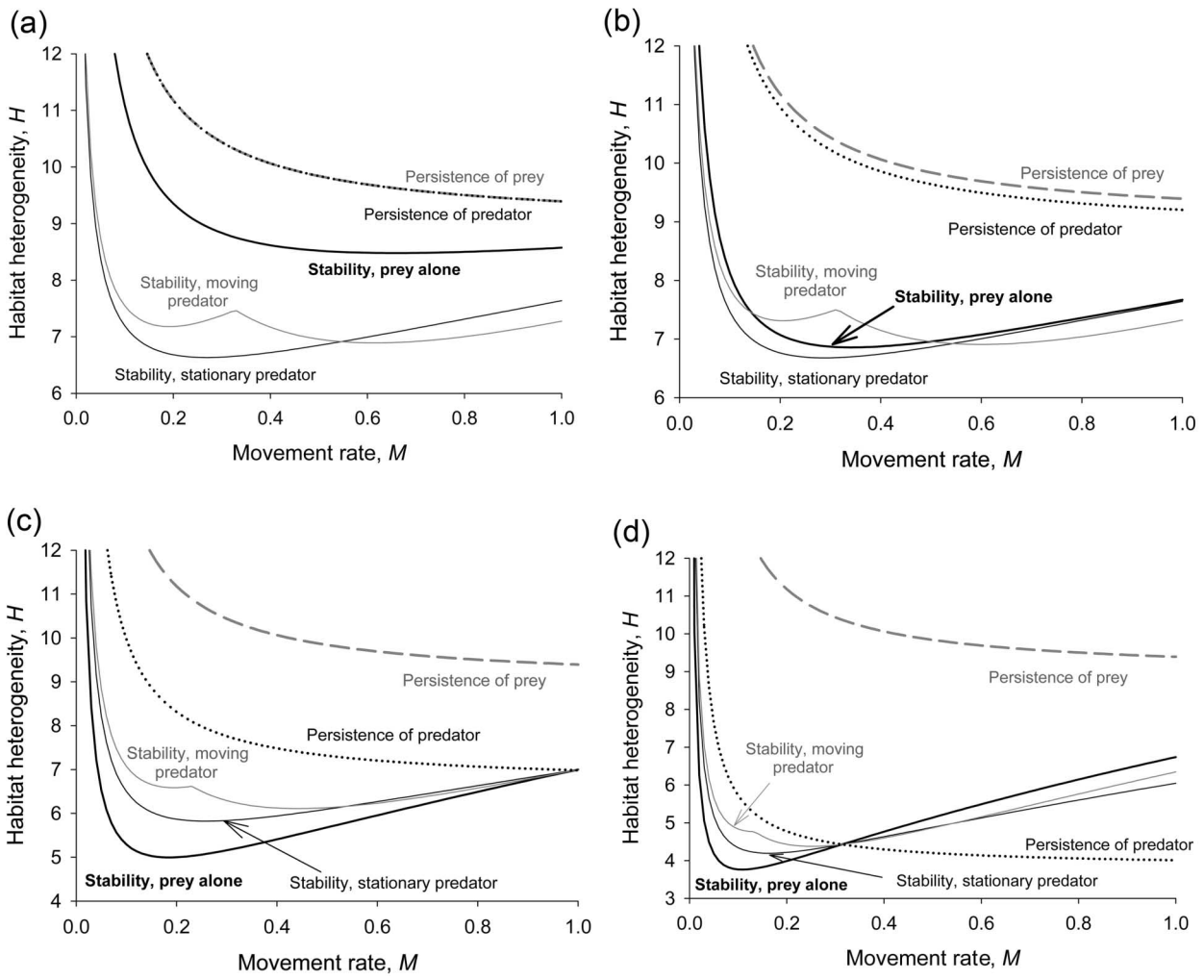

Figure A2: Maximum H for stability or persistence of the symmetric (habitat generalist) equilibrium with and without a predator. Other parameters are $\Gamma = 0.1$, and when the predator is present, $\mu = 0.2$ and $\rho = 1$. For a moving predator, its movement rate is one-tenth that of the prey. A stationary predator is present in both patches but does not move between patches. In all cases, the region of stability (or persistence) is below the corresponding curve, so a lower curve represents a more stringent requirement. If an equilibrium is unstable, the system will go to a different (specialist) equilibrium. Persistence limit of the prey is the dashed gray line, and that of the predator is the dotted line (virtually the same in *a*). The stability limit of the prey alone is the thick solid black line; thin solid black line $=$ with the stationary predator; solid gray line $=$ with the moving predator. The figures are ordered in terms of the strength of density dependence at low densities, from low to high: $\theta = 8$ (*a*), 2 (*b*), 0.5 (*c*), and 0.125 (*d*).

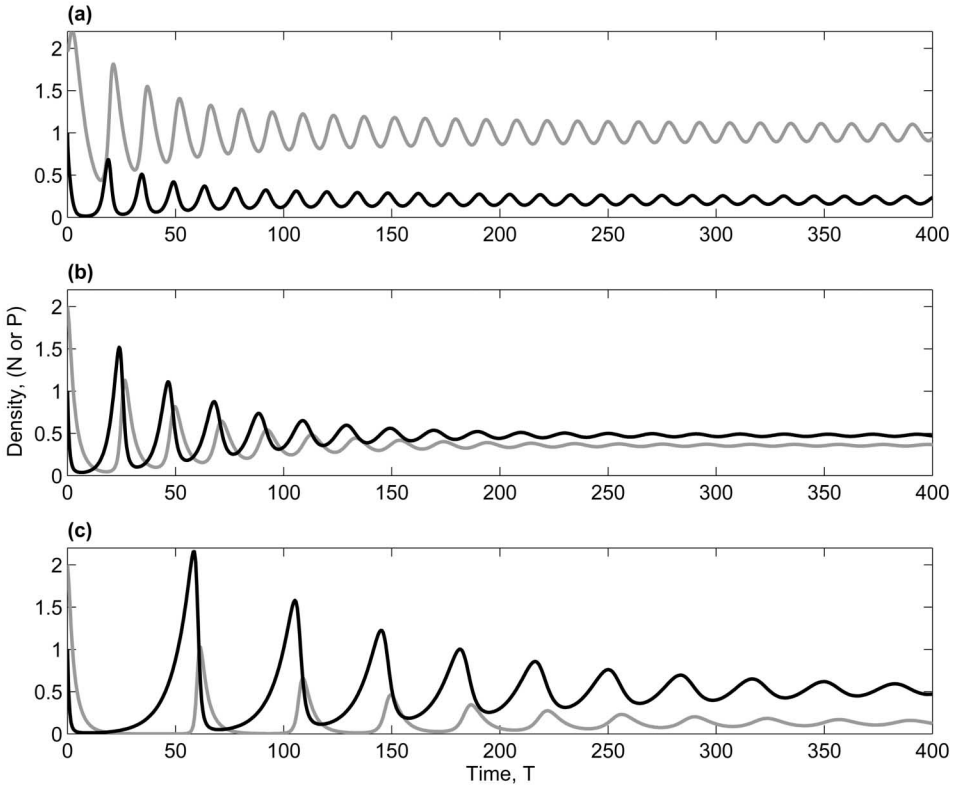

Figure A3: Modulation of the frequency of predator-prey oscillations by the steepness of the environmental gradient B for the density-independent predator-prey model. Prey densities at the range center ($X = 0$) are shown as black lines, predator densities are shown as gray lines. Parameters: $\alpha = 1$; $\delta = 1$; $\mu = 0.1$; $A = 0.4$; $B = 0.25$ (*a*), 1.24 (*b*), 1.53 (*c*).

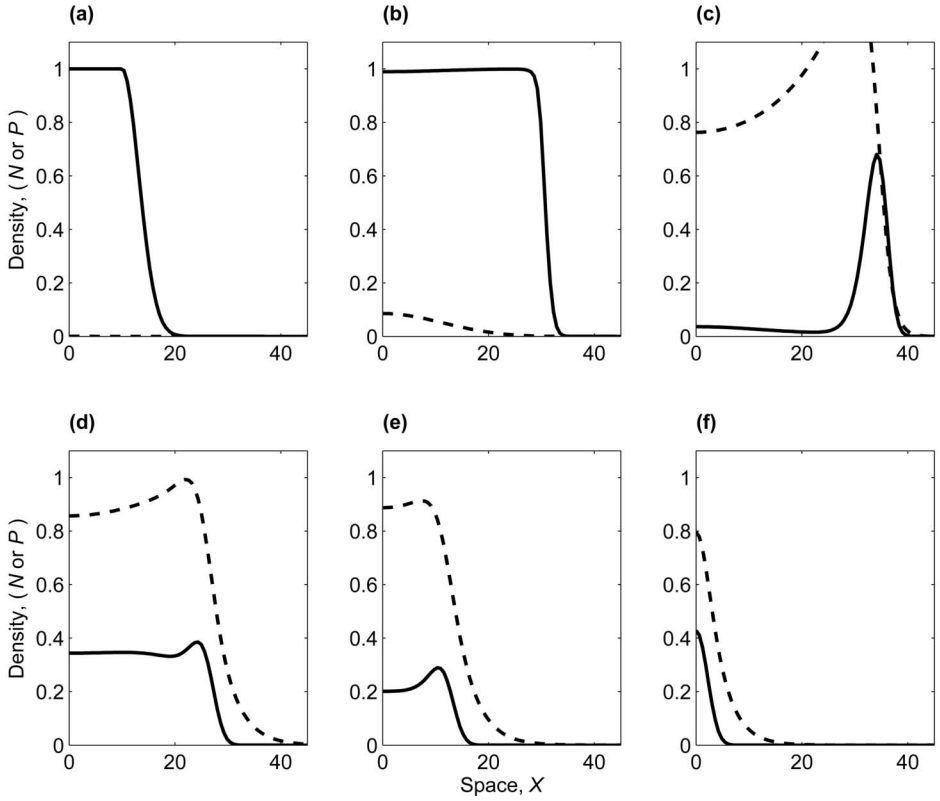

Figure A4: Predator-induced reversal of prey invasion and range collapse. Prey (solid lines) and predator (dashed lines) densities are shown at time 0 (*a*), 80 (*b*), 104 (*c*), 536 (*d*), 804 (*e*), 1098 (*f*). Parameters: $v = 1$, $\xi = (10)^{1/2}$, $N^* = 0.2$, $A = 0.5$, $B = 0.83$, $\theta = 8$. The predator spillover is clearly visible, causing the predator to occupy locations with negligible prey density.

Table A1. Variables and parameters used in the discrete space (two-habitat) models

Symbol	Definition	Normalized version
t	Time	$T = \frac{t}{r'}$
n_i	Prey density	$N_i = \left(\frac{n_i}{K}\right)\left(\frac{r_0}{r'}\right)^{1/\theta}$
$\bar{z}_i$	Mean phenotype	$Y_i = \frac{\lvert \bar{z}_i - z_{\text{opt},i} \rvert}{\sigma_g}$
p_i	Predator density	$P_i = \frac{a' p_i}{r'}$
r_0	Prey intrinsic growth rate	
K	Prey carrying capacity	
σ_p^2	Prey phenotypic variance	
σ_g^2	Prey genetic variance	
γ	Strength of selection	$\Gamma = \frac{\sigma_g^2 \gamma}{r'}$
r'	Realized maximum growth rate	$\frac{r_0 - \gamma \sigma_p^2}{2}$
θ	Density dependence parameter	
$z_{\text{opt},i}$	Optimum phenotype	$H = \frac{z_{\text{opt},2} - z_{\text{opt},1}}{\sigma_g}$
m	Prey movement rate	$M = \frac{m}{r'}$
d_i	Predator-induced mortality	$D_i = \frac{d_i}{r'}$
a'	Predator attack rate	$\rho = a' b' K \left(\frac{r}{r'}\right)^{1/\theta}$
b'	Conversion of prey to predator density	
m'	Predator mortality	$\mu = \frac{m'}{r'}$
m_p	Predator movement rate	$M_p = \frac{m_p}{r'}$

Note: Symbols with i subscripts are habitat specific.