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E-ARTICLE

Evolutionarily Labile Species Interactions and Spatial Spread of Invasive Species

T. Alex Perkins*

Center for Population Biology, University of California, Davis, California 95616

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ABSTRACT: Both exotic and native species have been shown to evolve in response to invasions, yet the impacts of rapidly evolving interactions between novel species pairs have been largely ignored in studies of invasive species spread. Here, I use a mathematical model of an interacting invasive predator and its native prey to determine when and how evolutionary lability in one or both species might impact the dynamics of the invader's spatial advance. The model shows that evolutionarily labile invaders continually evolve better adapted phenotypes along the moving invasion front, offering an explanation for accelerating spread and spatial phenotype clines following invasion. I then analytically derive a formula to estimate the relative change in spread rate due to evolution. Using parameter estimates from the literature, this formula shows that moderate heritabilities and selection strengths are sufficient to account for changes in spread rates observed in historical and ongoing invasions. Evolutionarily labile native species can slow invader spread when genes flow from native populations with exposure to the invader into native populations ahead of the invasion front. This outcome is more likely in systems with highly diffuse native dispersal, net directional movement of natives toward the invasion front, or human inoculation of uninvaded native populations.

Keywords: adaptation, exotic species, lag phase, range expansion, victim-exploiter interactions.

Introduction

As recently as 1996, a prominent textbook in invasion biology stated, "Invasions are fast, evolution is slow" (Williamson 1996, p. 168). Evolution has since gained recognition for its significance in exotic species invasions (Cox 2004) and is most often invoked for its role in the colonization (Lee and Gelembiuk 2008), establishment (Blossey and Notzold 1995; Ellstrand and Schierenbeck 2000), eventual extent (Sakai et al. 2001; Dietz and Edwards 2006), and long-term impacts (Mooney and Cleland 2001; Strauss et al. 2006) of invasive species. The role of evolution in the spread phase of invasions has received less consideration. Spatial spread is a transient, rapidly unfolding process (Williamson 1996), and the importance of rapid evolution on timescales relevant to population dynamics has only recently gained widespread attention and acceptance (Thompson 1998; Pelletier et al. 2009). Nonetheless, recent work has begun to demonstrate the importance of evolutionary processes in the spread phase of exotic species invasions.

Rates of spatial spread are determined by a demographic and a dispersal component (Skellam 1951), so evolutionary changes in either could potentially alter the dynamics of spread (Crooks and Soule 1999). A number of theoretical and empirical studies have shown that evolution of dispersal ability can (Travis and Dytham 2002; Phillips et al. 2008) and has (Simmons and Thomas 2004; Phillips et al. 2006; Hughes et al. 2007) altered spatial spread dynamics in a few decades or less. Likewise, a few studies have acknowledged that the demographic component of spread may be sensitive to evolutionary forces, but these studies have mostly focused on the special case of spread along an environmental gradient (Garcia-Ramos and Rodriguez 2002; Butin et al. 2005; Filin et al. 2008).

At the same time, the impacts of evolutionarily labile species interactions on spatial spread dynamics are unknown. Species interactions strongly influence invader demography and spatial spread (Shigesada and Kawasaki 1997; Lockwood et al. 2007) and often evolve following exotic species invasions. Examples of invader evolution following introduction are diverse, including the expansion of host use (Cox 2004), increased disease virulence (Fenner 1959), altered defense strategies against exploiters (Rogers and Siemann 2004), and both character displacement (King 1991) and increased competitive ability (Blossey and Notzold 1995). An equally impressive bestiary of evolutionarily labile native species has been documented. Examples include insects evolving better host exploitation

* E-mail: taperkins@ucdavis.edu.

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(Carroll et al. 2005), marine invertebrates gaining bolstered defenses against predators (Freeman and Byers 2006), vertebrate predators acquiring genetically based avoidance of toxic prey (Phillips and Shine 2006), hosts acquiring heightened defenses against pathogens (Marshall and Fenner 1958), and grasses evolving greater competitive ability (Leger 2008).

These and other examples of evolution by invasive and native species engaged in novel interactions have all occurred on timescales coincident with the process of invasion itself. As such, a great potential exists for evolutionarily labile species interactions to affect spatial spread, and understanding these dynamics is crucial for effective management (Epanchin-Niell and Hastings 2010). Before real-world applications can be realized, more basic questions must be answered. These include (1) how the intensification or abatement of interactions due to evolution may affect the dynamics of an advancing wavefront and (2) what characteristics predispose a species to these effects. Additionally, it is clear from empirical work that interactions between invasive and native species evolve quickly, but the question remains (3) whether realistic levels of selection strength and genetic variance allow for impacts on timescales relevant to spatial spread.

To answer these questions, I followed a long tradition of theoretical work on spread (Hastings et al. 2005) by analyzing a standard reaction-diffusion model with the added complexities of both demographic and phenotypic dynamics of an invasive and a native species. Similar models have been applied to study range limits on environmental gradients (Case and Taper 2000; Case et al. 2005; Goldberg and Lande 2007), but they have focused on equilibrium dynamics and are seldom applied to the transient dynamics of spread (but see Garcia-Ramos and Rodriguez 2002; Filin et al. 2008). The model here is simple in the sense that it applies to a single, environmentally homogeneous spatial dimension, but importantly, it incorporates the interaction between local adaptation and gene flow, which is often antagonistic and relevant to ecology (Kirkpatrick and Barton 1997; Garcia-Ramos and Rodriguez 2002; Forde et al. 2007). Using numerical solutions of these models, I show that rates of spatial spread may be sensitive to the evolution of invaders, natives, or both. I then provide an analytical approximation for the spread rate of an evolving invader and qualitative requirements necessary for native evolution to impact invader spread. In addition to detailing the effects of evolutionary lability on spread in this model, I analytically derive a more general formula for the relative change in spread rate due to invader evolution. I then assess the potential magnitude of invader evolution for spread in natural systems by applying observed estimates of linear selection gradients (Kingsolver et al. 2001) and demographic parameters from well-known invasions to the newly derived formula for relative change in spread.

Model

Verbal Description

The model features two species: a native prey and an invasive predator that specializes on it. The model also includes a quantitative trait in each species that is relevant to their interaction, such as bill depth and seed size in crossbills and pines (Benkman 1999). Before the invader's introduction, the native occupies a one-dimensional landscape (e.g., a coastline) at its carrying capacity everywhere along the landscape. Individuals continually follow random walks about the landscape such that dispersal is characterized by a diffusion process. Births and deaths also occur continually; both species therefore experience overlapping generations.

Births and deaths enter the model as contributions to or detractions from per-capita, per-generation population growth rates, or Malthusian fitnesses, for each species and depend on three independent fitness components. First, a stabilizing selection fitness component penalizes fitness if an individual's phenotype is so extreme as to interfere with aspects of its lifestyle external to the interaction (e.g., excessively large cane toads are plagued by arthritis; Brown et al. 2007). Second, a within-species fitness component accounts for births and deaths that occur independent from the invader-native interaction. I assume a Lotka-Volterra predator-prey relationship such that the predator's constant death rate and the prey's logistic birth rate represent the within-species fitness components. Third, a between-species fitness component accounts for births and deaths due to the interaction. Predators with more suitable phenotypes for catching prey contribute more to the population's birth rate, and prey with phenotypes better suited for predator evasion contribute less to their population's death rate. The suitability of a predator with a given phenotype for capturing a prey with a given phenotype is assessed using a function for interaction strength. I assume that predators are best able to capture and consume prey when their phenotypes match, as in the case of crossbills and pine seeds (Benkman 1999). Other interaction functions exist (e.g., Nuismer et al. 2007), but their distinction is more relevant to long-term equilibrium dynamics than it is to the transient dynamics of spread.

Mathematical Description of the Modeling Framework

The scenario described heuristically above of two interacting species inhabiting a one-dimensional landscape is commonly modeled using a system of reaction-diffusion

equations. A careful derivation of these equations in an ecological setting is provided by Okubo and Levin (2001), and their application to invasive species is detailed by Shigesada and Kawasaki (1997). Here, such a model accounts for invader and native population sizes $I(t, x)$ and $N(t, x)$ specified continuously for all points in time t and space x (the notation for which is now omitted for brevity). The equations governing each species' population dynamics are

$$\frac{\partial I}{\partial t} = \frac{\sigma_I^2}{2}\frac{\partial^2 I}{\partial x^2} + \bar{m}_I I, \tag{1a}$$

$$\frac{\partial N}{\partial t} = \frac{\sigma_N^2}{2}\frac{\partial^2 N}{\partial x^2} + \bar{m}_N N, \tag{1b}$$

where σ_i^2 is the per-generation mean squared displacement of species i (hereafter dispersal coefficient) and $\bar{m}_i(t, x)$ is its mean Malthusian fitness averaged across all phenotypes $z_i(t, x)$. Mean Malthusian fitness represents the average per-capita, per-generation population growth rate of individuals at a given time t in a given location x. It is therefore dynamic, and its dependencies on other variables and parameters are described in more detail below.

The interaction traits z_i of each species are quantitative characters that impact births and deaths but do not affect movement. Biologically, this means that this model is concerned with traits such as bill depth and seed size (Benkman 1999) or claw size and shell thickness (Freeman and Byers 2006) as opposed to searching or avoidance behavior. Mathematically, this means that the dispersal coefficient of each species remains unchanged by the interaction. A reaction-diffusion model for traits such as these was presented by Pease et al. (1989), developed further by Kirkpatrick and Barton (1997), and has since been applied to interacting species (Case and Taper 2000) and spatial spread of a single species (Garcia-Ramos and Rodriguez 2002). This model makes the same assumptions about life history and dispersal as ecological reaction-diffusion equations but goes further and acknowledges a trait z_i with normal distribution $\phi_i(z_i)$, dynamic mean $\bar{z}_i$, and fixed phenotypic variance $V_{P,i}$. Such a formulation is consistent with the infinitesimal model of quantitative genetics, which assumes that phenotypes z_i are determined by identically small additive effects from a large number of unlinked loci and a normally distributed source of environmental variation (Bulmer 1980). According to this description, the system of equations for interaction trait dynamics in time and space is

$$\frac{\partial \bar{z}_I}{\partial t} = \frac{\sigma_I^2}{2}\frac{\partial^2 \bar{z}_I}{\partial x^2} + \sigma_I^2\frac{\partial \ln I}{\partial x}\frac{\partial \bar{z}_I}{\partial x} + V_{A,I}\frac{\partial \bar{m}_I}{\partial \bar{z}_I}, \tag{2a}$$

$$\frac{\partial \bar{z}_N}{\partial t} = \frac{\sigma_N^2}{2}\frac{\partial^2 \bar{z}_N}{\partial x^2} + \sigma_N^2\frac{\partial \ln N}{\partial x}\frac{\partial \bar{z}_N}{\partial x} + V_{A,N}\frac{\partial \bar{m}_N}{\partial \bar{z}_N}, \tag{2b}$$

where $V_{A,i}$ is additive genetic variance (derived in the appendix of Pease et al. 1989). In words, the phenotypic composition of a population inhabiting point x on the landscape at time t changes in response to two forces: (1) gene flow from nearby populations (first and second terms of the right-hand side) and (2) selection that occurs locally within that population (third term of the right-hand side). The first gene flow term accounts for diffusive rearrangement of mean phenotypes, and the second weighs that rearrangement according to uneven population sizes across space. Based on the breeder's equation (Lande 1976), the local selection term is an approximation of the evolutionary dynamics of a quantitative trait rather than an exact model. Even so, it accurately captures evolutionary change in trait means in the face of strong selection (Turelli and Barton 1994).

Tailoring the Model to Invasive Predators and Native Prey

As described thus far, the model in equations (1) and (2) could apply to any type of interaction. From here on, however, I restrict my analysis to the case of an invasive predator and a native prey species with Lotka-Volterra dynamics. Invasive predators are of particular interest because they tend to be some of the most disruptive invaders (Schoener and Spiller 1999; Blackburn et al. 2004; Greenlees et al. 2006; Pangle et al. 2007) and because victim-exploiter interactions are especially prone to selection and coevolution (Abrams 2000).

Mathematically, species interactions enter the model in equations (1) and (2) through the specification of mean Malthusian fitness $\bar{m}_i$. I assume that stabilizing selection and selection from within- and between-species interactions act on fitness independently. It follows that mean Malthusian fitness is equal to the sum

$$\bar{m}_i = \bar{m}_{i,\text{stab}} + \bar{m}_{i,\text{within}} + \bar{m}_{i,\text{between}} \tag{3}$$

of the stabilizing, within-species, and between-species fitness component means (Gavrilets 1997).

The model for stabilizing selection assumes that there is an intermediate, optimal phenotype θ_i conferring maximal fitness and that values too inadequate or too far in excess of that optimum suffer a fitness cost. Selection for these intermediate, optimal phenotypes has nothing to do with interaction between the model's focal species. Instead, it acts to constrain the range of phenotypes accessible to evolution and accounts for fitness trade-offs that may select against extreme phenotypes. These trade-offs could be related to energetic demands, the ability to find or attract mates, or vulnerability in interactions with other species. Accordingly, the stabilizing selection fitness component mean for each species is defined mathematically as

$$\bar{m}_{i,\text{stab}} = \int \{\exp[-k_i(z_i - \theta_i)^2] - 1\}\phi(z_i)dz_i, \quad (4)$$

where $\phi(z_i)$ is a normally distributed phenotype distribution with mean $\bar{z}_i$ and variance $V_{\text{P},i}$. Henceforth, $\theta_I = \theta$ and $\theta_N = 0$ for simplicity.

Within-species interactions in the model are consistent with the basic Lotka-Volterra predator-prey model. Invasive predators die at a density-independent rate d, and native prey display logistic growth at rate r up to a local carrying capacity K. Hence, the within-species fitness component means are $\bar{m}_{I,\text{within}} = -d$ and $\bar{m}_{N,\text{within}} = r(1 - N/K)$.

The between-species fitness component is also consistent with a Lotka-Volterra predator-prey formulation but includes an additional coefficient S that weakens the interaction if predators of phenotype z_I are ill-suited for capturing prey of phenotype z_N. Modifying species interactions in this way for quantitative interaction traits such as z_I and z_N has its roots in models of competitive interactions (MacArthur and Levins 1967; Bulmer 1974; Slatkin 1980; Taper and Case 1985), but the same idea can be applied to predator-prey interactions (Abrams 2001). I define S consistent with the phenotype matching model of species interaction strength, wherein the interaction is intensified by closer correspondence between trait values. Examples in nature include crossbills with appropriately sized beaks being the most effective foragers on locally plentiful seed sizes (Benkman 1999) and cuckoos' success as nest parasites being optimized when egg coloration patterns match those of their hosts (Davies and Brooke 1989). Mathematically, the interaction strength coefficient $S(\phi_I, \phi_N)$ depends on the phenotype distribution ϕ_i of each species and takes the mathematical form

$$S = \int\int \{\exp[-\alpha(z_I - z_N)^2]\phi_I(z_I)dz_I\}\phi_N(z_N)dz_N, \quad (5)$$

where α is the species interaction selection strength. Large values of α correspond to a highly specialized interaction where predators feed successfully only on like prey, whereas small values of α result in a diffuse interaction where phenotype values are largely irrelevant to predation success. With the interaction strength coefficient in hand, the between-species fitness component means are then

$$\bar{m}_{I,\text{between}} = abNS, \quad (6a)$$

$$\bar{m}_{N,\text{between}} = -aIS, \quad (6b)$$

with a the attack rate and b the efficiency with which predators convert prey deaths into predator births.

Parameters and Scaling

Numerical solutions calculated equations (4) and (5) directly. However, making approximations of $\bar{m}_{i,\text{stab}}$ and S was necessary to simplify the range of parameter combinations for numerical evaluation and to proceed with analytical work. Motivated by this, I simplified the exponential kernel in each expression by performing a Taylor approximation about $z_i = \theta_i$ in equation (4) and about $z_I - z_N = 0$ in equation (5), resulting in quadratic expressions. Because the model assumes that the phenotype distribution ϕ_i of each species is normal with mean $\bar{z}_i$ and variance $V_{\text{P},i}$, I was able to calculate the integrals in equations (4) and (5) exactly to obtain the approximations

$$\bar{m}_{i,\text{stab}} \approx -k_i[V_{\text{P},i} + (\bar{z}_i - \theta_i)^2], \quad (7a)$$

$$S \approx 1 - \alpha[V_{\text{P},I} + V_{\text{P},N} + (\bar{z}_N - \bar{z}_I)^2]. \quad (7b)$$

Inserting these approximations into equations (3) and (6) yields a simplified version of the model suitable for mathematical analysis.

One use of the simplified model is reducing the number of parameters. Predator-prey dynamics are complicated enough without the addition of evolutionary dynamics, so it is desirable to simplify the relationships among parameters that account for the model's range of dynamic behaviors. In the context of spatial spread, it is well known that behavior at the invasion front, where invaders are at low densities and natives in greater abundance, is key. It follows that one of the most important characteristics of predator-prey dynamics for spatial spread is how rapidly invasive predators at low density deplete native prey. Given values of all the other parameters, there is an attack rate

$$\hat{a} = \frac{2(d + k_I V_{\text{P},I})}{bS\hat{N}_{\text{IF}}} \quad (8)$$

that optimizes equilibrium predator densities along a spectrum of more or less voracious attack rates that lead to lower or higher equilibrium prey densities (Roughgarden 1979; $\hat{N}_{\text{IF}} = K(1 - k_N V_{\text{P},N}/r)$ is the equilibrium prey population size in the absence of predation). Predator-prey dynamics can then be characterized simply by the degree to which a exceeds or falls short of $\hat{a}$, providing a benchmark for the model's ecological dynamics regardless of the values of other parameters on the right-hand side of equation (8).

Another use of the simplified model is establishing biologically informative scales for the model's variables. Equilibrium population sizes are a natural scale on which to interpret the values of I and N. It is less clear what t constitutes a short amount of time, what x is a far distance, or what difference $|z_N - z_I|$ between native and invader phenotype means is of any consequence. Recalling that

disparity between invader and native phenotypes leads to invader failure in this model, a quantity of special interest in phenotypic units is the threshold difference D between invader and native phenotypes beyond which the invader's growth rate falls below 0. Plugging equations (6) and (7) into equation (3), equating $\bar{m}_I$ with 0, and solving for $|\bar{z}_I - \bar{z}_N|$ reveal that

$$D \approx \sqrt{\frac{1}{\alpha} - \frac{d + k_I V_{\mathrm{P},I}}{ab\hat{N}_{\mathrm{IF}}\alpha} - V_{\mathrm{P},I} - V_{\mathrm{P},N}}. \tag{9}$$

Although it is not strictly true given the complications of spatial mixing, when $|\bar{z}_I - \bar{z}_N| > D$ is true at a given location, the invasion generally will not proceed past that point, and otherwise it will. This quantity naturally gives rise to a quantity in time units of special interest: the amount of time T it takes for a native to evolve to $\bar{z}_N$ such that $|\bar{z}_I - \bar{z}_N| > D$. In the absence of gene flow and invader evolution, native evolution is governed by the reduced version of equation (2b), $\partial\bar{z}_N/\partial t = V_{\mathrm{A},N}(\partial\bar{m}_N/\partial\bar{z}_N)$. This equation can then be solved to find that

$$T \approx -\frac{1}{P_N}\ln\left(1 + \frac{\theta - D}{\bar{z}_N - Q_N/P_N}\right), \tag{10}$$

where the composite parameters are defined as $Q_N = -2V_{\mathrm{A},N}a\hat{I}_{\mathrm{CE}}\alpha\bar{z}_I$ and $P_N = 2V_{\mathrm{A},N}k_N - 2V_{\mathrm{A},N}a\hat{I}_{\mathrm{CE}}\alpha$. Finally, a related spatial quantity X can be defined as

$$X \approx cT, \tag{11}$$

where c is some estimate of the invader's spread rate. The quantity X corresponds to the distance spread by the invader in the time it takes the native to mount an adaptive response. This is useful to keep in mind because if X far exceeds the potential range size of the invader, then native evolution is unlikely to have much of an impact on spread dynamics. However, if X is small compared with the invader's potential range size, then at least some natives will adapt to combat invaders and potentially slow spread.

Model Analysis

I made analytical approximations wherever possible. However, exact solutions of the model are analytically intractable. I therefore verified the analytical results with numerical solutions of the full model and also used those solutions to address topics that were otherwise inaccessible. Details of the numerical implementation of the model are discussed in appendix A.

Results

Here I present the results of examining four general cases of the model where neither, either, or both the invader and native are evolutionarily labile. In the two cases where the native is not evolutionarily labile (hereafter static), I derived an analytical approximation for the invader's asymptotic spread rate that accurately represents the transient spread rate of the evolving invader when viewed as a function of a dynamically evolving phenotype under this model. Given this result, I then derived a more general estimate of transient changes in spread due to invader evolution and quantified that estimate with empirically estimated parameter values from the literature. Results about evolutionary consequences of ecologically distinct invaders are also shown. The derivation of biologically informative variable scales is instructive about native properties that increase their potential for slowing spread, and numerical solutions of the model illustrate qualitative results about the dispersal characteristics of native species required for slowing invader spread.

Case 1: Static Invader, Static Native

A well-known property of diffusion-based spatial spread models is that the asymptotic spread rate c equals $(2\bar{m}(0)\sigma^2)^{1/2}$, where $\bar{m}(0)$ is population growth rate at low abundance (Skellam 1951). Using the approximations in equation (7), the implication for this model is that in the absence of evolutionary change, invader spread should proceed at a constant rate

$$c \approx \sqrt{2\{-k_I[V_{\mathrm{P},I} + (\bar{z}_I - \theta)^2] - d + ab\hat{SN}_{\mathrm{IF}}\}\sigma_I^2}. \tag{12}$$

Even though phenotypes do not evolve in this case, the degree of phenotype matching between invaders and natives at the onset of invasion has a perceptible impact on spread through its effect on S (see eq. [7b]). If the invader phenotype is within D phenotypic units of the native phenotype, spread proceeds; otherwise, the invasion fails. A good match between invader and native phenotypes results in fast asymptotic spread of the invader.

Case 2: Labile Invader, Static Native

In the case where an invasive species has the capacity for evolutionary change and a native species does not, the invader's phenotype dynamics at low density on the invasion front are approximated by the spatially implicit model $(\partial/\partial t)\bar{z}_I(t, x - ct) \approx V_{\mathrm{A},I}(\partial\bar{m}_I/\partial\bar{z}_I)$ (app. B). Invader phenotypes at the invasion front thus follow the trajectory

$$\bar{z}_I(t, x - ct) \approx \left(\theta - \frac{Q_I}{P_I}\right)\exp(-P_I t) + \frac{Q_I}{P_I}, \qquad (13)$$

where $Q_I = 2V_{A,I}k_I\theta + 2V_{A,I}ab\hat{N}_{IF}\alpha\bar{z}_N$ and $P_I = 2V_{A,I}k_I + 2V_{A,I}ab\hat{N}_{IF}\alpha$. This approximation shows that mean invader phenotype evolves continually at rate P_I at the invasion front until a fitness-maximizing phenotype of Q_I/P_I is attained (black line in fig. 1*b*). Numerical results validate this approximation (correspondence between lines and circles in left column of fig. 2).

A result of invader evolution at the invasion front is an increase in spread rate over time (front progresses farther in fig. 1*c* than in fig. 1*a* in the same amount of time). An approximate solution of spread rate as a function of time during the transient, evolutionarily sensitive phase of spread can be attained by plugging the solution for invader phenotype at the invasion front from equation (13) into the asymptotic spread rate approximation in equation (12) via equation (7b). Numerical solutions of the full model show that this approximation provides a useful caricature of the transient spread dynamics of evolutionarily labile invaders (correspondence between lines and circles in right column of fig. 2). Whether calculated numerically or approximated analytically, the dynamic spread rate $c_{evol}(t)$ of an evolutionarily labile invader can be compared with the fixed asymptotic spread rate c parameterized with phenotypes from the onset of invasion ($\bar{z}_I = \theta$, $\bar{z}_N = 0$) to study the effects of evolutionary lability on spread dynamics.

The response of $c_{evol}(t)/c$ to the manipulation of key parameter values provides insight into the effects of invader evolution on transient spread dynamics. Greater values of genetic variance $V_{A,I}$ and interaction selection strength α accelerate both evolution toward the optimal phenotype at the invasion front (fig. 2*a*, 2*b*) and the advance of the front itself (fig. 2*e*, 2*f*). High genetic variation and a highly specialized interaction are thus two factors that predispose invaders to evolutionary impacts on spread. On the other hand, stabilizing selection k_I has little effect on accelerating either phenotype evolution or spread but does influence the phenotype and asymptotic spread rate that can ultimately be attained (fig. 2*c*, 2*g*). In fact, if stabilizing selection is strong enough, invader evolution and spread acceleration are stifled altogether. Greater attack rates a also accelerate spread more rapidly when evolution acts, but this enhanced transient acceleration comes at the cost of a reduced asymptotic spread rate (fig. 2*d*, 2*h*). This reduction in asymptotic spread results from stabilizing selection acting on the more extreme phenotypes produced when $a \gg \hat{a}$.

Invader evolution impacts phenotype dynamics not only at the invasion front but also in its wake. One novel pattern

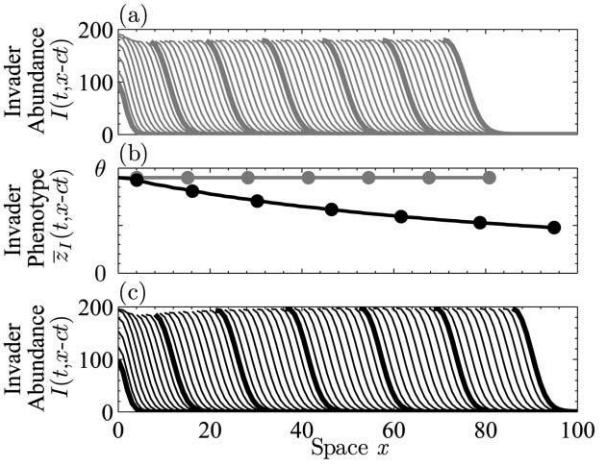

Figure 1: Progression of invasion front when invader is evolutionarily static (*a*; $V_{A,I} = 0$) or labile (*c*; $V_{A,I} = (1/4)V_{P,I}$). Invader evolution at the invasion front (*b*; black vs. gray) accounts for the acceleration in spread (*c* vs. *a*). For ease of visual interpretation, each line from left to right in *a* and *c* shows the invasion profile at successive points in time and is drawn from the peak of the invasion profile on the left to $I = 0$ in the uninvaded region on the right. Wide (narrow) lines occur in increments of 10 (1) time units between 3 and 63 from left to right in *a* and *c*.

emerging here is the formation of invader phenotype clines behind the invasion front (fig. 3). The general requirement for this result is that selection pressures differ at and behind the invasion front. At the invasion front, native prey populations are consistently at their carrying capacity, whereas populations behind the front become increasingly depressed from invader exploitation over time. This difference in native population sizes at and behind the invasion front results in differing selection pressures on invader phenotypes there, because interaction strength depends on native population size in equation (5). As a result, higher predator attack rates a depress native populations behind the invasion front more quickly and severely, leading to steeper invader phenotype clines in the wake of spread (cf. fig. 3*a*, 3*b*).

Case 3: Static Invader, Labile Native

The temporal and spatial scales T and X defined in equations (10) and (11) provide insight into when it is possible for native evolution to impact spread. As one example, small θ means that the invader and native are phenotypically very similar at the onset of invasion, so according to equation (10), it will take a relatively long time for the native to evolve a phenotype capable of reducing invader population growth below 0. As another example, equation (11) shows that if the invader spreads quickly (large c),

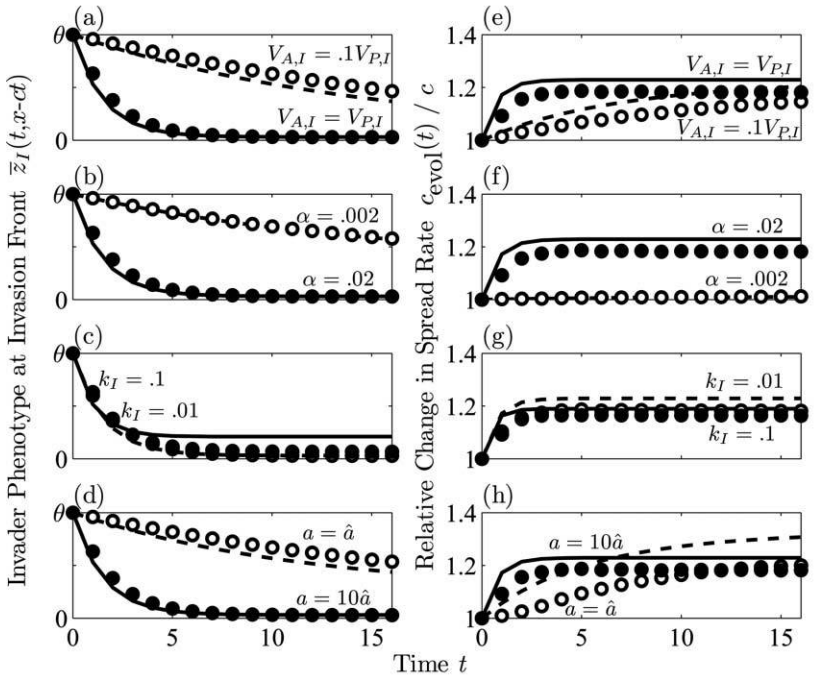

Figure 2: Invader phenotype evolution at the invasion front (*a–d*) and relative change in spread rate over time (*e–h*) in response to changes in invader additive genetic variance $V_{A,I}$ (*a*, *e*), interaction selection strength α (*b*, *f*), invader stabilizing selection strength k_I (*c*, *g*), and attack rate a (*d*, *h*). Dashed lines and open circles are used for smaller parameter values, while solid lines and filled circles are used for larger values. Lines come from the approximation in equations (12) and (13), and circles show the results of numerical solutions. Equation (15) does not apply directly because its parameters do not vary with time; otherwise, it also agrees with the lines because it is a mathematically equivalent restatement of equations (12) and (13). The spatial domain was of length $|x_r - x_l| = 120$, and all other parameters are as in table 1.

the potential range size of the invader must be very large for there to be enough time for native evolution to be relevant; otherwise, spread will be complete before natives can make an adaptive response.

The ability of a native species to impact spread depends not only on the quickness of its adaptive response and on the eventual size of the invader's range but also on the native's ability to relay genes from adapted populations toward the invasion front. In the absence of human assistance, the means by which invader-adapted native genes could catch up to the invasion front is either by the native dispersing more widely than the invader ($\sigma_N^2 \gg \sigma_I^2$; fig. 4) or by the native having a directional bias, or advection, in dispersal relative to the invader (fig. 5). Even if advection carries invader-adapted genes toward the front, native dispersal must still be orders of magnitude greater than the invader's to have an impact on spread (dashed line in fig. 6). If advection moves against the front, impacts on spread are less likely and require that natives possess an even greater dispersal advantage (solid line in fig. 6). Such a directional bias was introduced into the model by adding advective terms $\delta(\partial N/\partial x)$ and $\delta(\partial \bar{z}_N/\partial x)$ to the right-hand sides of equations (1b) and (2b), respectively.

Case 4: Labile Invader, Labile Native

When both an invader and a native are evolutionarily labile, invader impacts on spread dominate native impacts. In case 3, where the native was evolutionarily labile and the invader was not, the conditions under which the native could relay invader-adapted genes to the invasion front fast enough to impact spread were quite stringent. If the invader were to spread even faster, it would be more difficult for the native to impact spread (i.e., the lines in fig. 6 would shift to the right). Numerical results show that this is precisely what happens when an invader is evolutionarily labile (fig. 7); it accelerates spread (fig. 7*b*), making it even more difficult for invader-adapted native genes to catch up to the front (fig. 7*a*). Thus, given similar capacities for evolutionary lability (i.e., $V_{A,I} \approx V_{A,N}$), the impacts of invader evolution on spread are likely to dominate impacts of native evolution.

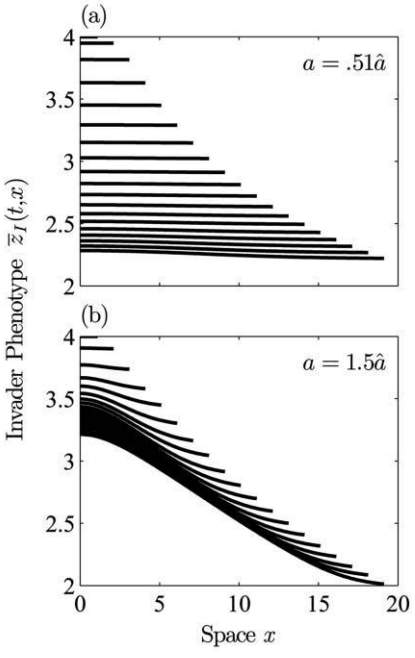

Figure 3: Spatial patterns in invader phenotype during spread. Lines show the pattern of invader phenotypes across its range at successive points in time (from $t = 0.1$ to 19.1 in increments of 1) from numerical solutions. *a* and *b* show the different spatial patterns that result from spread of invaders with different attack rates. These solutions were obtained numerically on a spatial domain of length 100 over time 100, and all other parameters are as in table 1.

Beyond Interspecific Interactions

In case 2, in which the invader is evolutionarily labile but the native is not, the result of invader evolution at the invasion front and subsequent spread acceleration applies more broadly than just to invader-native interactions. As the invasion front moves, native populations just ahead of the front present invaders with consistent native abundances and phenotype means and thereby with consistent invader population growth rates $\bar{m}_I$ at the invasion front. For the purpose of modeling spread, natives are therefore static and factor into the invader's spread rate via $\bar{m}_I$ just like anything else (Shigesada and Kawasaki 1997). Consequently, the relative increase in spread rate $c_{\text{evol}}(t)/c$ for an invasive predator discussed in case 2 and shown in figure 2 can be generalized to other evolving invaders with the approximation

$$\frac{c_{\text{evol}}(t)}{c} \approx \sqrt{1 + \frac{1}{\bar{m}_I(0)}\left(\frac{\partial \bar{m}_I}{\partial \bar{z}_I}\right)^2 V_{\text{A},I}\, t} \qquad (14)$$

(app. C). To enhance its applicability to real invasions, the approximation in equation (14) can be rewritten in terms of more readily estimable parameters as

$$\frac{c_{\text{evol}}(t)}{c} \approx \sqrt{1 + \frac{h^2\beta^2}{\bar{m}_I(0)G^2}\, t}, \qquad (15)$$

where narrow-sense heritability $h^2 = V_{\text{A},I}/V_{\text{P},I}$, β is the standardized selection gradient, and G is generation time (app. C). In general, this formula shows that greater acceleration of spread due to invader evolution is expected when baseline population growth $\bar{m}_I(0)$ is low, generation time G is short, heritability h^2 is high, or selection β is strong. Of course, some of the parameters in equations (14) and (15) may change as an invasive species spreads, in which case they can be made functions of time. For instance, in the full invader-native model, β decreases over time as stabilizing selection reduces the strength of selection on the increasingly extreme phenotypes favored by selection on predation success. Nonetheless, rather than making accurate predictions, the purpose of this approximation is to act as a tool for those who wish to make a rough guess about the potential impact of invader evolution on spread.

Quantitative Significance in Natural Populations

To get an idea of how great heritability and selection must be to exert a meaningful impact on exotic species invasions, I applied estimates of $c_{\text{evol}}(t)/c$, $\bar{m}_I(0)$, and G from six invasions (app. D) to equation (15) and solved for the values of h^2 and β that would be required to account for observed levels of spread acceleration (fig. 8). This exercise was not meant to attribute the dynamics of these invasions to evolution but rather to determine whether invader evolution could ever possibly generate increases in spread rate similar to what has been observed in real invasions. To provide a context for how strong selection gradients typically are in natural populations, in each panel of figure 8, I plotted the fiftieth, seventy-fifth, and ninety-fifth percentiles of the distribution of published selection gradients compiled by Kingsolver et al. (2001). Using parameters from these six invasions and equation (15), moderate heritabilities and selection gradients appear sufficient to produce spread acceleration comparable to that observed in these invasions. Even though the year-to-year increases observed in these invasions varied dramatically, from 2% to 500%, variation among the heritabilities and selection gradients necessary to account for these differences is relatively small because of concomitant variation in generation times (fig. 8).

Although the analysis in figure 8 does not ascribe spread dynamics of its six example invasions to evolution, it may be possible to do so for invasions with both a record of

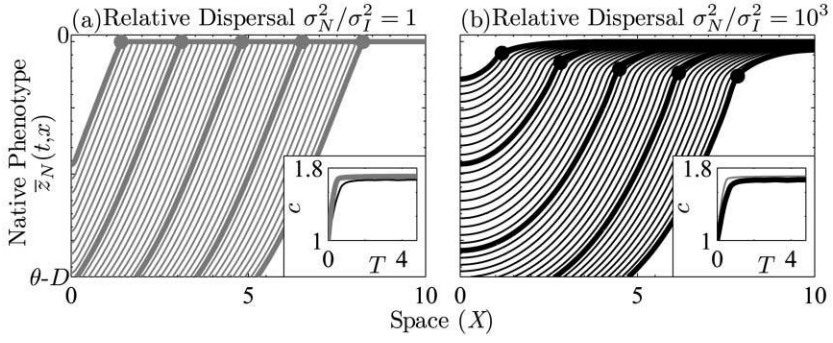

Figure 4: Effect of relative dispersal ability σ_N^2/σ_I^2 on mean native phenotype $\bar{z}_N(t, x)$ as an invader spreads from left to right across an area inhabited by an evolutionarily labile native ($V_{A,N} = (1/2)V_{P,N}$). Insets show corresponding invader spread rates over time, with line thickness and color indicating which spread rate trajectory corresponds to which panel (gray for *a*, black for *b*). Circles show mean phenotype at the invasion front at different points in time, and wide (narrow) lines occur in increments of 100 (10) time units between 81 and 481 from left to right in each panel. For ease of visual interpretation, each line shows mean phenotype from its minimum value on the left toward the uninvaded side of the native's range on the right. These solutions were obtained numerically on a spatial domain of length $10X$ over time $10T$ to allow ample time for natives to mount an evolutionary response, and all other parameters are as in table 1.

range expansion over time and data on the spatial distribution of phenotypes. Two invasions that may meet such minimum data requirements are the myxoma virus and cane toad invasions of Australia. In addition to the potential for invader evolution to affect spread in these cases (Fenner 1959; Phillips et al. 2010), they also display evidence of native evolution in response to the invaders (Marshall and Fenner 1958; Phillips and Shine 2006). Comparison of models with evolution and other plausible mechanisms for observed spread dynamics in systems such as these could be a fruitful line of future investigation.

Discussion

In this study, I used a mathematical model of interacting invasive and native species to determine when and how evolutionary lability in one or both species might impact the dynamics of the invader's spatial advance. Given sufficient genetic variation and selection pressure, evolution of traits relevant to the interaction can take place in either species at the invasion front, impacting spread by way of the invader's reproductive success there. Enabled by gene flow from past occupants of the front and spurred by consistent selection pressures from uninvaded areas, invaders may evolve faster at the invasion front than behind it. In contrast, native evolution occurs in the wake of the advancing front. Genetic material from these populations must somehow catch up to the front to impact spread. The range of possible impacts in these scenarios is summarized in table 1.

Evolutionarily Labile Invader

One of the primary findings of this study is that when conditions in uninvaded regions incite directional selection in invader phenotypes, invader populations at the edge of a moving invasion front are exposed to consistently strong selection pressures and show continual evolutionary responses there. Behind the front, invaders evolve more slowly if selection pressures change over time. Selection on interaction traits may often change over time if an invader effects changes in native abundance or the distributions of native interaction traits, size, or age. In this model, selection on the invasive predator's interaction trait weakens behind the invasion front because of prey depletion, leading to the formation of a spatial cline (fig. 3). Even so, this result about cline formation is more general and applies to any trait for which selection is density dependent or whose selection depends on time since invasion (e.g., male aggression in Western bluebirds is most adaptive in newly colonized populations; Duckworth and Badyaev 2007). Another case where this pattern may occur is virulence evolution of emerging pathogens. Virulence is often observed to peak soon after the onset of pathogen emergence and then wane over time as susceptible hosts are depleted and selection pressures change (Bolker et al. 2010). As an emerging pathogen spreads over space, which may take place over hundreds of generations (app. F, available online), my results suggest that virulence would continually increase at the invasion front, where pathogens encounter a never-ending supply of susceptible hosts, and attenuate behind the front as susceptibles become exhausted.

Another important finding is that the evolution of in-

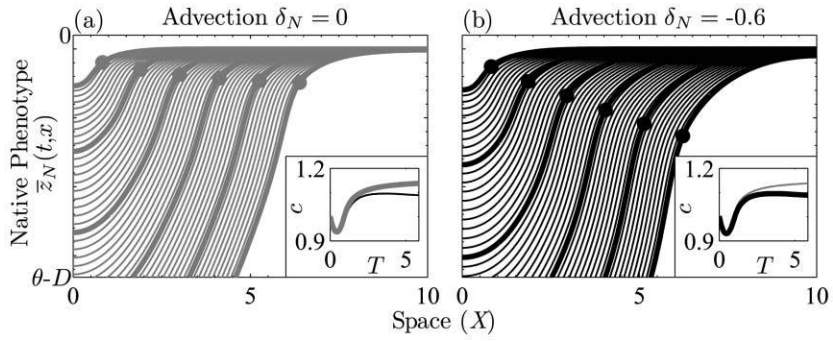

Figure 5: Effect of advective dispersal bias δ on mean native phenotype $\bar{z}_N(t, x)$ as an invader spreads from left to right across an area inhabited by an evolutionarily labile native ($V_{A,N} = (1/2)V_{P,N}$). Insets show corresponding invader spread rates over time, with line thickness and color indicating which spread rate trajectory corresponds to which panel (gray for *a*, black for *b*). Circles show mean phenotype at the invasion front at different points in time, and wide (narrow) lines occur in increments of 100 (10) time units between 85 and 485 from left to right in each panel. For ease of visual interpretation, each line shows mean phenotype from its minimum value on the left toward the uninvaded side of the native's range on the right. These solutions were obtained numerically on a spatial domain of length $10X$ over time $10T$ to allow ample time for natives to mount an evolutionary response, and all other parameters are as in table 1, except $\alpha = 0.01$ and $\sigma_N^2 = 100$.

vader interaction traits can accelerate spread by increasing invader population growth at the front over time. Consistent with data from real invasions, accelerating spread is ultimately a transient phenomenon (e.g., the house finch invasion of North America; Veit and Lewis 1996) but may persist for much of the duration of spread (e.g., geographic spread of several pathogens; Mundt et al. 2009). In the model, invader phenotypes eventually settled on an equilibrium (fig. 2*a*–2*d*) that was determined by a balance of maximizing predation and mitigating stabilizing selection. Once invaders at the front attained that phenotype, spread leveled off at a maximum speed (fig. 2*e*–2*h*). In nature, evolution at an invasion front may cease before the conclusion of spread as a result of any number of factors, including fitness trade-offs, exhaustion of genetic variation, or constraints due to genetic or phenotypic correlations. These results about invader evolution and increasing spread rates also apply to other traits affecting population growth (Holt et al. 2005; Phillips et al. 2010), such as fecundity or juvenile survival (Siemann and Rogers 2001; Duckworth and Badyaev 2007; Phillips 2009).

Invader evolution and transient spread acceleration were synonymous in my model, but in general that need not be true. Using a similar model, Garcia-Ramos and Rodriguez (2002) also found that an invader's phenotype mean formed a traveling wave spurred by evolution at the invasion front, yet spread proceeded at a constant speed and in some cases decelerated. The reason for the difference between our models is that Garcia-Ramos and Rodriguez (2002) defined the trait to be a spatially varying stabilizing selection optimum; thus, adaptation was a prerequisite for spread rather than an accelerant. This may often be the case for invasions along spatial gradients or during the expansion of established ranges (e.g., Butin et al. 2005).

Of course, invader evolution is not the only phenomenon that can accelerate spread. Other factors such as Allee effects (Lewis and Kareiva 1993) and long-distance dispersal (Shigesada et al. 1995; Kot et al. 1996) are usually thought of as the most parsimonious explanations for increasing spread rates (Hastings et al. 2005), including for two of the species in figure 8 (Veit and Lewis 1996; Gammon and Maurer 2002). Rather than competing with these alternative explanations by attempting to attribute accelerating spread in the examples from figure 8 to invader evolution, the point of this exercise was to determine whether spread acceleration to the extent observed in nature could ever possibly be accounted for by invader evolution. Indeed, figure 8 shows that moderate heritabilities and realistic selection strengths are capable of accelerating spread to the extent observed for species with the population growth rates and generation times of those species, suggesting that invader evolution should be taken as seriously as classical explanations for accelerating spread in analyses of empirical spread data.

Evolutionarily Labile Native

One of the most important factors that limits the impact of native evolution on invader spread is time. Most essentially, there must be enough time for native evolution to occur before spread has concluded (i.e., $T < t_{\text{spread}}$). In a compilation of published results, Strauss et al. (2006) showed that it can take anywhere from a few years to a

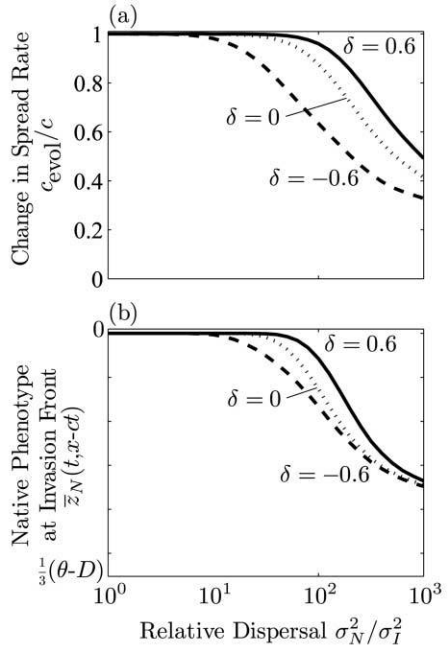

Figure 6: Requirements for native dispersal relative to invader dispersal (σ_N^2/σ_I^2) for slowing spread (*a*) and changing native phenotypes at the invasion front (*b*). Different lines correspond to different advective dispersal biases (solid line, $\delta = 0.6$; dotted line, $\delta = 0$; dashed line, $\delta = -0.6$). The native species is evolutionarily labile ($V_{A,N} = (1/2)V_{P,N}$), whereas the invader is not ($V_{A,I} = 0$). Solutions were obtained numerically on a spatial domain of length $4X$ over time $4T$ to allow ample time for natives to mount an evolutionary response, and all other parameter values are as in table 1, except $\alpha = 0.01$.

century or more for natives to display adaptive, genetically based responses to invaders. In my own compilation of 30 invasions, I gathered published data to determine how long it typically takes for spatial spread to run its course (app. F). Spread can proceed for anywhere from a few months to centuries, meaning that the duration of spread is as long and as variable as the elapsed time before natives evolve (i.e., $T \sim t_{spread}$). Thus, in some cases natives probably mount evolutionary responses before spread has concluded, and in other cases they probably do not. For native species who do respond before the conclusion of spread, there must also be enough time for adaptive genetic material to reach the invasion front if impacts on spread are to occur. What will determine whether the amount of time $T - t_{spread}$ between native response and the conclusion of spread is sufficient for impacts on spread is the time it takes for invader-adapted native genes to spread toward the invasion front.

The most general way that invader-adapted native genes can catch up to the invasion front is if native dispersal is more diffuse than invader dispersal. One place where this is possible is the marine realm, where mean dispersal distances varied by several orders of magnitude in a survey of 90 species distributed across different taxonomic groups (Kinlan and Gaines 2003). Another generalization that stands out in that study is that in many cases primary consumers have mean dispersal distances that are two to four orders of magnitude greater than those of the producers on which they feed (Kinlan and Gaines 2003). Native primary consumers who display an evolutionary response to invasive producers might therefore be strong candidates to impact spread through phenotypic evolution (per fig. 6).

Another factor that affects whether invader-adapted native genes have enough time to catch up to the invasion front is net advective dispersal bias of the native relative to the invader (figs. 5, 6). Such a bias can occur if one species advects and the other does not (e.g., wind pollination) or if both advect but at different rates (e.g., pelagic larvae spending different amounts of time in the planktonic stage). One relevant invasion in an advection-dominated system is that of the Asian shore crab *Hemigrapsus*

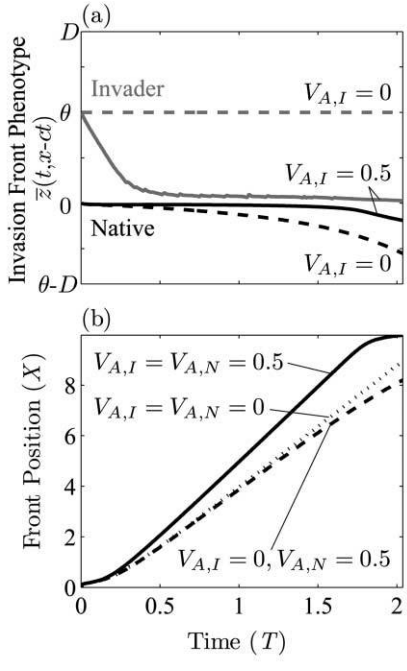

Figure 7: Different effects of native evolution ($V_{A,N} = (1/2)V_{P,N}$) on phenotypes at the invasion front (*a*) and invasion front position (*b*) when invaders are either evolutionarily labile (solid lines; $V_{A,I} = (1/2)V_{P,I}$) or static (dashed lines; $V_{A,I} = 0$). In *a*, invader phenotype trajectories appear in gray and native phenotype trajectories in black. The dotted line in *b* shows the front position when there is no evolution in either species. All parameters are as in table 1, except $\alpha = 0.01$ and $\sigma_N^2 = 10^3$.

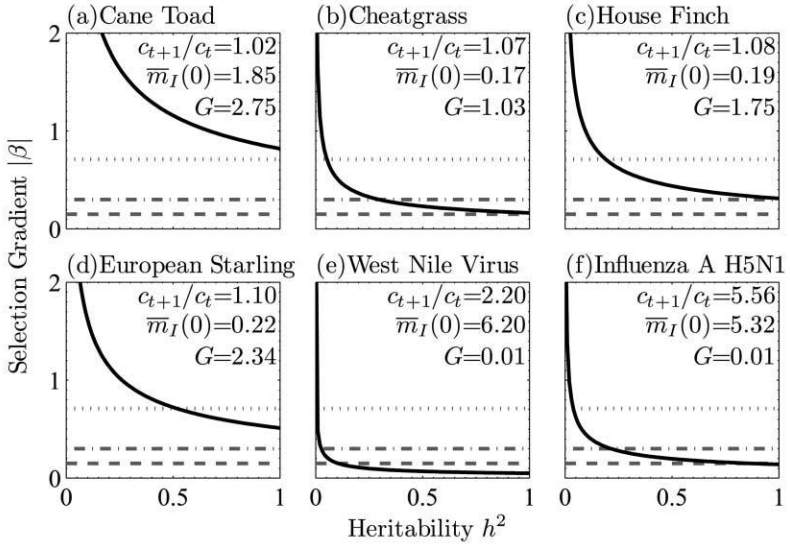

Figure 8: Selection gradient and heritability requirements to achieve relative changes in spread rate from invader evolution consistent with average year-to-year increases c_{t+1}/c_t observed in six invasions. The solid line in each panel shows the combination of $|\beta|$ and h^2 that satisfies equation (15), given parameter values displayed in the panel (for explanation of parameter values, see app. D). In the region above and to the right of this line, invader evolution would produce even greater spread acceleration. In the region below and to the left of these lines, invader evolution would lead to less of an increase in spread rate. The dashed, dash-dotted, and dotted lines in each panel indicate the fiftieth, seventy-fifth, and ninety-fifth percentiles of the absolute values of selection gradients ($|\beta| = 0.15, 0.30, 0.71$) from Kingsolver et al. (2001).

sanguineus off the coast of New England, where the native mussel *Mytillus edulis* evolved an increased capacity for shell thickening (Freeman and Byers 2006). Because the invader spread north against the dominant advective current, crab-adapted mussels from the south would have been unlikely to influence the genetics of unexposed mussel populations at the invasion front (Byers and Pringle 2006; Freeman and Byers 2006), consistent with results in figure 6. Impacts of native evolution on spread would seem more likely were *H. sanguineus* spreading in the direction of the dominant advective current. Even this would depend on the duration of the planktonic stage in each species though, because δ is a relative quantity.

Management Implications

Although most evolutionarily labile native species probably do not satisfy the requirements for impeding invader spread, natural resource managers may sometimes have the ability to facilitate gene flow from adapted core populations to naive edge populations. For example, seed from native plant populations displaying adaptation to competition from invasive plants or herbivory by invasive insects could be introduced to uninvaded populations (Leger 2008). Managers can also slow down spread by influencing invader evolution at the front. One option for achieving this is targeted culling of the best-adapted invaders at the front (Sih et al. 2010) with the intention of influencing the direction or strength of selection in a way that prevents spread acceleration. Even if targeting certain individuals is not feasible, culling could also retard invader evolution at the invasion front by reducing adaptive genetic variation there.

Model Limitations

One of the most tenuous assumptions in the model is that genetic variation is fixed and unchanging. It is unclear how relaxing this assumption might affect the results, because some factors tend to decrease additive genetic variation and retard adaptation (inbreeding and genetic drift; Bulmer 1980) whereas others could supplement genetic variation and drive adaptation (multiple introductions: Kolbe et al. 2004; gene flow: Alleaume-Benharira et al. 2005; mutation accumulation: Butin et al. 2005). Another simplification is that the ecological model features a single specialist predator and its lone prey. Even so, in some instances, strong evolutionary forces can also result between nonspecialist species (e.g., Motychak et al. 1999). Finally, only the simple case of a predator-prey interaction with Lotka-Volterra dynamics is considered. A saturating functional response would generate a lesser gradient in

Table 1: Summary of possible impacts of evolution on spread, depending on which species is evolutionarily labile

Invader	Native	Impact of evolution on spread
Static	Static	Spread at a constant rate
Labile	Static	Accelerating spread due to evolution of invader phenotype at the invasion front
Static	Labile	Decelerating spread if exposed and adapted natives contribute genetically to populations at the invasion front
Labile	Labile	Invader evolution accelerates spread and attenuates effects of native evolution at the invasion front

prey density immediately behind the invasion front, which could lead to nominally different results, such as shallower clines or slightly stronger native gene flow toward the invasion front. Nonetheless, the present formulation is generally applicable to exploiter-victim interactions, and key results should apply to all interaction types.

Acknowledgments

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APPENDIX A

Numerical Implementation of the Model

To obtain numerical solutions of equations (1)–(6), I used the function pdepe in Matlab (ver. 7.8; MathWorks, Natick, MA), which involves forward integration of the ordinary differential equations that result from discretizing the spatial dimension. Concurrent calculations of equations (4) and (5) were performed by standard numerical integration (trapz function in Matlab), and the selection gradient $\partial\bar{m}_i/\partial\bar{z}_i$ was calculated using a finite difference approximation (Press et al. 2002). The model's initial conditions were a uniform distribution of natives at their stable, invader-free equilibrium $\hat{N}_{\mathrm{IF}} = K(1 - k_N V_{\mathrm{P},N}/r)$ across 1,000 discrete points in the spatial dimension, whereas invaders were uniformly distributed at their coexistence equilibrium $\hat{I}_{\mathrm{CE}}$ across 10 points at one end of the landscape, where $\hat{I}_{\mathrm{CE}} = [-k_N V_{\mathrm{P},N} + r(1 - \hat{N}_{\mathrm{CE}}/K)]/aS$ and $\hat{N}_{\mathrm{CE}} = (k_I V_{\mathrm{P},I} + d)/abS$. I chose to initialize invader populations at a somewhat high abundance because my focus here is on spread, which is a process that is defined to occur after establishment (Williamson 1996). Initializing invaders to be scarce at time $t = 0$ leads to similar results using this model. I imposed reflecting boundary conditions at the boundary points x_l, x_r such that the partial derivatives $(\partial \cdot /\partial x)$ with respect to space equaled 0 for native and invader abundance (N, I) and mean phenotypes ($\bar{z}_N$, $\bar{z}_I$).

The choice of boundary conditions, length of the spatial dimension, and length of the time span could all have impacts on spread, primarily near the far end of the invaded region. To minimize any such impacts, I chose a sufficiently long spatial dimension and time span and assessed spread rate in the interior of the landscape. Specifically, I assessed instantaneous spread rate $c_{\mathrm{evol}}(t)$ by recording the farthest position at which the invader population exceeded $\hat{I}_{\mathrm{CE}}/10$ at each time step, taking the differences between those positions per time step length and smoothing them with a low-pass filter (rectangular window of width $|x_r - x_l|/10$, using the filtfilt function in Matlab). Numerical estimation of the wave speed occurs at the very front of the wave (location $x - ct$) where invader density is low, so dynamics behind the crest of the wave should have virtually no impact on behavior at the front or on numerical estimation of the wave speed.

APPENDIX B

Approximation of Evolutionary Dynamics at the Invasion Front

Rather than thinking about phenotype dynamics in terms of mean phenotype $\bar{z}$, for the purpose of calculating asymptotic spread rate, it is more convenient mathematically to think in terms of the sum of phenotype values p across all n members of a population. For example, if $\bar{z}$ were mean body mass in a population, then p would be the sum of body masses of all members of the population; that is, $p = \sum_{i=1}^{n} z_i$. The relationship of these variables,

$$\bar{z} = \frac{p}{n}, \tag{B1}$$

can be used with equation (1) and the equation governing the dynamics of p in time and space (shown below) to derive equation (2) (Pease et al. 1989).

Given mean Malthusian fitness $\bar{m}$ and additive genetic variance V_{A}, the local dynamics of p are embodied mathematically by

$$\frac{\partial p}{\partial t} = \bar{m}p + V_A \frac{\partial \bar{m}}{\partial \bar{z}} n, \tag{B2}$$

which is consistent with equation (B1) and the last terms on the right-hand sides of equations (1) and (2). Biologically, the first term on the right-hand side of equation (B2) accounts for changes in population size n that increase or decrease p without changing the mean phenotype $\bar{z}$, and the second term represents evolution by natural selection that increases or decreases the mean phenotype $\bar{z}$.

In the model for p, the sum of phenotypes p should redistribute itself according to a diffusion process with mean squared displacement per time σ^2, irrespective of individuals' phenotypes, as is the case in equations (1) and (2). Adding a diffusion term to equation (B2) then results in

$$\frac{\partial p}{\partial t} = \frac{\sigma^2}{2}\frac{\partial^2 p}{\partial x^2} + \bar{m}p + V_A \frac{\partial \bar{m}}{\partial \bar{z}} n, \tag{B3}$$

which, combined with

$$\frac{\partial n}{\partial t} = \frac{\sigma^2}{2}\frac{\partial^2 n}{\partial x^2} + \bar{m}n, \tag{B4}$$

describes the dynamics of p and n in time and space (Nagylaki 1975).

If there is little genetic variance or selection pressure (i.e., small V_A and $\partial \bar{m}/\partial \bar{z}$) at the invasion front of a species spreading according to equations (B3) and (B4), then calculation of the spread rate of p and n is straightforward (Murray 2002): $c = (2\bar{m}\sigma^2)^{1/2}$. In this scenario with no evolution, the sum of phenotype values p simply increases at the invasion front as the number of individuals there increases. However, if there is appreciable genetic variance and selection pressure, the term $V_A(\partial \bar{m}/\partial \bar{z})n$ ensures that some additional p—beyond increases in p owing to population growth—will accrue at the front as a result of evolution. These changes in p due to evolution in turn change $\bar{z}$ at the invasion front at rate $V_A(\partial \bar{m}/\partial \bar{z})$, as in equation (13). The accuracy of this approximation and its impact on spatial spread are corroborated by numerical solutions of the model (fig. 2).

APPENDIX C

Derivation of the Formula for Change in Spread Rate

Taking the result that mean phenotype dynamics at the invasion front are described by $(\partial/\partial t)\bar{z}_I(t, x - ct) \approx V_{A,I}(\partial \bar{m}_I/\partial \bar{z}_I)$ from appendix B and making the simplifying assumption that additive genetic variance $V_{A,I}$ and the selection gradient $(\partial \bar{m}_I/\partial \bar{z}_I)$ are constants, we obtain a related solution to equation (13) of

$$\bar{z}_I(t, x - ct) = \bar{z}_I(0) + V_{A,I}\frac{\partial \bar{m}_I}{\partial \bar{z}_I} t. \tag{C1}$$

Because linear selection gradients encapsulate how phenotype changes affect fitness over time, it follows from linearizing fitness about the mean phenotype that

$$\bar{m}_I(t) = \bar{m}_I(0) + \frac{\partial \bar{m}_I}{\partial \bar{z}_I}[\bar{z}_I(t, x - ct) - \bar{z}_I(0)]. \tag{C2}$$

With this formula describing how Malthusian fitness changes at the invasion front over time, it follows that the relative change in spread rate over time due to invader evolution is

$$\frac{c_{evol}(t)}{c} = \frac{\sqrt{2\bar{m}_I(t)\sigma_I^2}}{\sqrt{2\bar{m}_I(0)\sigma_I^2}} = \sqrt{1 + \frac{1}{\bar{m}_I(0)}\left(\frac{\partial \bar{m}_I}{\partial \bar{z}_I}\right)^2 V_{A,I} t}. \tag{C3}$$

More readily estimable parameters can be applied to equation (C3) by substituting the strength of selection $\partial \bar{m}_I/\partial \bar{z}_I$ with the standardized selection gradient $\beta = (\partial \bar{m}_I/\partial \bar{z}_I)\sqrt{V_{P,I}}G$, where G is generation time. The change in spread rate over t years due to invader evolution is then simplified to

$$\frac{c_{evol}(t)}{c} = \sqrt{1 + \frac{h^2\beta^2}{\bar{m}_I(0)G^2} t}, \tag{C4}$$

where narrow-sense heritability $h^2 = V_{A,I}/V_{P,I}$.

Assuming that evolutionary change in the native species is negligible at the invasion front, $\bar{m}_I(0)$ will depend only on properties of the invader. In that case, the derivation in this appendix is generally applicable to any spreading invader with a quantitative trait that affects Malthusian fitness and is under selection at the invasion front.

APPENDIX D

Calculation of Observed Year-to-Year Increases in Spread Rate

Calculating year-to-year increases in spread rate for the North American cheatgrass, house finch, and starling invasions assumed that spread accelerated at a constant rate. Data for these invasions were time series of range radii over the course of each invasion (Wing 1943; Mack 1981; Veit and Lewis 1996). First, I performed a linear regression of the log of each time series. Where m is the slope and b the intercept of this regression, the instantaneous spread rate is $me^b e^{mt}$. The change in spread rate from one year to the next is then e^m, which is 1.0713 for cheatgrass,

1.0788 for house finches, and 1.1030 for starlings. Mundt et al. (2009) performed the same type of regression on spatial spread data for West Nile virus and influenza A (H5N1), which suggested respective yearly changes in spread rate of 2.2034 and 5.5622 for those invasions. Calculating year-to-year increase in spread rate for the cane toad invasion of Australia made use of the initial spread rate (10 km year^{-1}), a more recent spread rate (55 km year^{-1}), and the time since introduction to Australia (73 years; Urban et al. 2008). Assuming that spread rate increases by the same percent each year, the year-to-year increase in cane toad spread rate was $(55/10)^{(1/73)} = 1.0236$.

Estimates of demographic parameters displayed in figure 8 also come from diverse sources. Population growth rate $\bar{m}(0)$ was calculated explicitly for starlings by van den Bosch et al. (1992). For cane toads, cheatgrass, influenza, and West Nile virus, one or more estimates of R_0 (Lampo and De Leo 1998; Cruz-Pacheco et al. 2005; Ward et al. 2009; Griffith 2010) were used to calculate population growth rate as $\bar{m}(0) = \ln\{[(1/N)\sum_{i=1}^{N} R_{0,i}]/G\}$. Mean annual population increase in house finches was reported by Robbins et al. (1986), the log of which is equal to $\bar{m}(0)$. The procedures I used to obtain values of generation time G are discussed in appendix F, available online.

APPENDIX E

Parameter and Variable Definitions

Table E1: Base parameter and variable definitions

Symbol	Definition	Units	Baseline value
a	Attack rate	t^{-1}	$\hat{a}$
b	Conversion efficiency	N^{-1}	.5
d	Predator death rate	t^{-1}	.5
$I(t, x)$	Invasive predator abundance	I	$\hat{I}_{\mathrm{CE}}$
K	Prey carrying capacity	N	1,000
k_i	Stabilizing selection strength	$z^{-2}\ t^{-1}$	.001
$N(t, x)$	Native prey abundance	N	$\hat{N}_{\mathrm{IF}}$
r	Prey growth rate	t^{-1}	.5
t	Time	t	...
$V_{\mathrm{A},i}$	Additive genetic variance	z^2	0–1
$V_{\mathrm{P},i}$	Phenotypic variance	z^2	1
x	Space	x	...
$z_{I,N}(t, x)$	Mean phenotype	z	θ, 0
α	Species interaction selection strength	z^{-2}	.02
δ	Advective dispersal bias	xt^{-1}	0
σ_i^2	Mean squared displacement per time	x^2t^{-1}	1
$\theta_{I,N}$	Stabilizing selection optimum phenotype	z	4, 0

Table E2: Composite parameter definitions

Symbol	Definition
m	Malthusian fitness
S	Effect of phenotype evolution on attack rate
$\hat{a}$	Attack rate that maximizes equilibrium predator density
$\hat{N}_{\mathrm{IF}}$	Invader-free native prey equilibrium
$\hat{I}_{\mathrm{CE}}$, $\hat{N}_{\mathrm{CE}}$	Coexistence equilibria
c, c_{evol}	Spread rate, with and without evolution
Q_i, P_i	Parameters for trajectory of phenotype evolution
D	Maximum phenotype distance between invader and native for invader establishment to be possible
T	Time required for native to evolve phenotype capable of repelling invader establishment
X	How far invasion spreads in time T
h^2	Narrow-sense heritability
β	Standardized selection gradient

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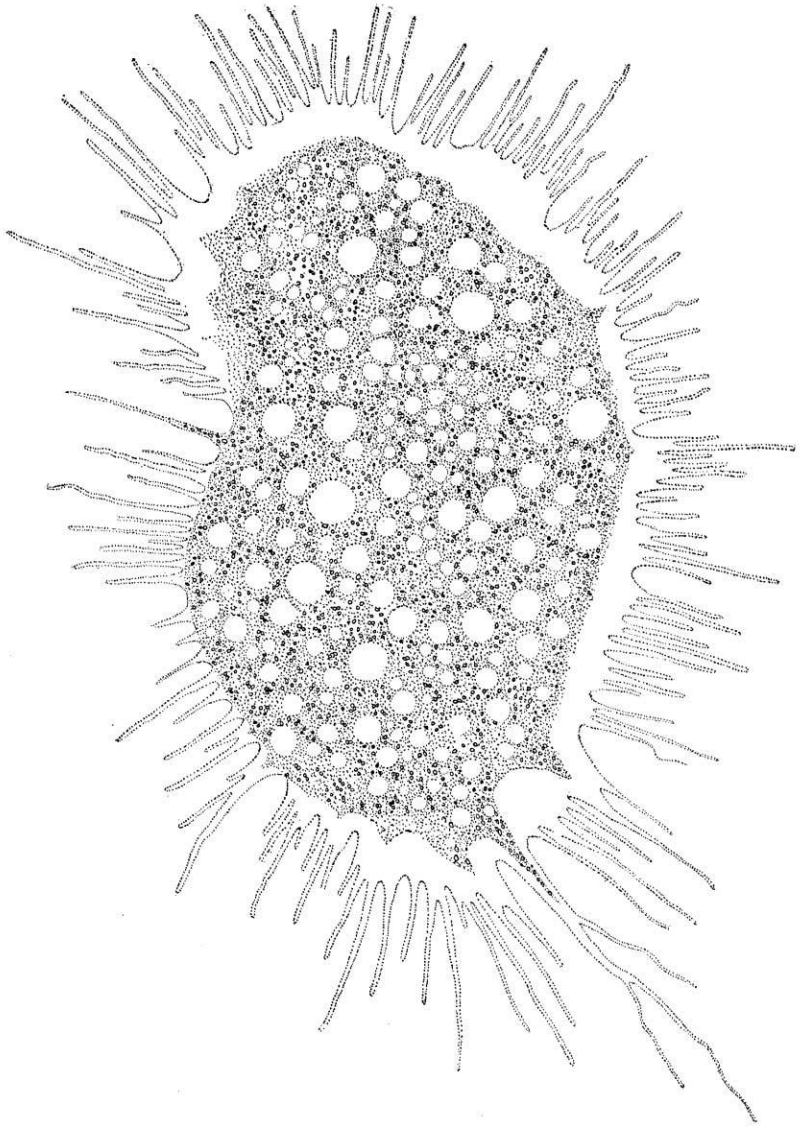

"In December, 1904, I discovered in some water taken by Mr. William G. Lapham from an oozy bank near Afton, Virginia, a large Vampyrella-like specimen, which except for the absence of nuclei and the variable size of the vacuoles answered in detail to *Leptophrys elegans*." From "Notes on the Genus *Leptophrys*," by William A. Kepner (*American Naturalist*, 1906, 40:335–342).