Interference competition and invasion: Spatial structure, novel weapons and resistance zones

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Abstract

Certain invasive plants may rely on interference mechanisms (e.g., allelopathy) to gain competitive superiority over native species. But expending resources on interference presumably exacts a cost in another life-history trait, so that the significance of interference competition for invasion ecology remains uncertain. We model ecological invasion when combined effects of preemptive and interference competition govern interactions at the neighborhood scale. We consider three cases. Under "novel weapons," only the initially rare invader exercises interference. For "resistance zones" only the resident species interferes, and finally we take both species as interference competitors. Interference increases the other species' mortality, opening space for colonization. However, a species exercising greater interference has reduced propagation, which can hinder its colonization of open sites. Interference never enhances a rare invader's growth in the homogeneously mixing approximation to our model. But interference can significantly increase an invader's competitiveness, and its growth when rare, if interactions are structured spatially. That is, interference can increase an invader's success when colonization of open sites depends on local, rather than global, species densities. In contrast, interference enhances the common, resident species' resistance to invasion independently of spatial structure, unless the propagation-cost is too great. The particular combination of propagation and interference producing the strongest biotic resistance in a resident species depends on the shape of the tradeoff between the two traits. Increases in background mortality (i.e., mortality not due to interference) always reduce the effectiveness of interference competition.

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1. Introduction

Both lateral and vertical interactions can affect the likelihood that an invasive species advances when rare (Schwinning and Parsons, 1996; Levine et al., 2004; Going et al., 2009). But for many plants, competitive asymmetry between invader and resident species governs both the outcome and the timescale of ecological invasion (Lavergne and Molofsky, 2004; Vila and Weiner, 2004; O'Malley et al., 2006a; MacDougall et al., 2009). Given invader–resident competition, ecological superiority may depend on more than one mechanism (Case and Gilpin, 1974; Ridenour and Callaway, 2001). Our analysis addresses the combined impact of preemptive and interference competition on invasion dynamics when biotic interactions are structured spatially. We assume that interference has a cost (Adams et al., 1979; Bazzaz and Grace, 1997; Amarasekare, 2002); an increasing level of interspecific interference requires a reduction in propagation rate, diminishing that species' capacity to colonize unoccupied sites.

In many plant communities the primary mode of competition is site preemption (Schoen, 1983; Bergelson, 1990; Crawley et al., 1999; Yurkonis and Meiners, 2004); i.e., species interact through colonization of empty sites. Superior preemptive competitors have higher propagation rates or lower mortality rates (Korniss and Caraco, 2005; O'Malley et al., 2006b; Allstadt et al., 2007). The former increases colonization of open sites, and the latter decreases a competitor's opportunities for colonization. Preemptive competitors have the same niche in a spatially homogeneous environment (Amarasekare, 2003). Ordinarily this precludes coexistence (Shurin et al., 2004; Allstadt et al., 2009), since self-regulation does not exceed interspecific competition. Case and Gilpin (1974) suggest that this niche similarity might favor evolution of interference mechanisms.

An interference competitor inhibits another species' access to a critical resource, often by harming individuals of the other species. Examples include interspecific territoriality in animals and
chemical competition in plants (Case et al., 1994; Callaway and Ascheghoug, 2000; Cantor et al., 2011). Exotic invaders may suppress native species' densities through interference competition (D'Antonio and Vitousek, 1992; Callaway and Ridenour, 2004; Cappuccino and Arnason, 2006). The “novel weapons” hypothesis proposes that some invasive plants release chemicals that inhibit growth of native species (Callaway and Ascheghoug, 2000). Allelopathic interference may act directly on individuals of the resident competitor, or may act indirectly through toxic effects on native species' microbial mutualists, particularly mycorrhizal fungi (Wolfe and Klironomos, 2005; Callaway et al., 2008; Cantor et al., 2011). So, under the novel weapons hypothesis, invaders attain competitive superiority because their phytochemicals present novel challenges to native species. Reasonably, in other communities, exotic species likely encounter interference competition from natives; see comments in Von Holle et al. (2003). Our models address three general cases where interference can affect the outcome of resident–invader competition. We associate novel weapons with interference by the invader only. We refer to “resistance zones” when the resident species, but not the invader, exerts interference competition. And, of course, invaders and residents may each compete via interference (Case and Gilpin, 1974). If neither species exhibits interference, our model leaves preemption as the sole competitive mechanism. In this case the species that, when alone, maintains the greater equilibrium density via preemption as the sole competitive mechanism. In this case the species that, when alone, maintains the greater equilibrium density will always displace its competitor (Amarasekare, 2003; O’Malley et al., 2006a; Allstadt et al., 2007).

Discrete, stochastic spatial models and their deterministic analogues have been employed, commonly and successfully, to gain insight into collective behavior of multi-“species” interactions (Marro and Dickman, 1999; Murray, 2003) in physics (Korniss et al., 1995, 1997), chemistry (Antal et al., 1998; Toroczkai et al., 1997), and in the study of population dynamics (Ellner et al., 1998; McKane and Newman, 2004; O’Malley et al., 2009). We use both methods to investigate the combined effects of preemptive and interference competition on invasion. We organize our paper as follows. First, we present a discrete (individual-based), stochastic model where an invader and a resident species compete preemptively, and one or both species also employs interference. We let a species’ propagation rate depend on its level of interference; we consider convex, linear and concave tradeoffs. We analyze invasibility criteria of both a mean-field approximation (homogeneous mixing) and a pair approximation. Then we apply results of the approximations to interpret simulations of the full spatial model. The Discussion compares our results to other spatial models incorporating allelopathy. Appendices collect analytical details.

Our results show that interference by the invader increases its likelihood of successful invasion only when interactions are spatially structured. That is, the novel weapons advantage appears only when we account for local clustering of invader individuals. Interference by the resident, the initially common species, can inhibit invasion with and without spatial structure. For the unstructured case, we specify the minimal propagation-rate advantage required for an invader to overcome the resident’s biotic resistance. Increases in background mortality rate (mortality before adjustment due to interference) diminish the competitiveness of a species relying more on interference. Increased mortality reduces both local and global densities; that is, the frequency of empty sites increases. Consequently, the value of interference, relative to propagation, declines. Our model assumes that interference increases mortality of a preemptive competitor. However, the preemption–interference combination does not generate an alternative niche, and we consequently should not anticipate coexistence absent continuous introduction or strong effects of spatial clustering (Allstadt et al., 2009).

2. A discrete, stochastic spatial model

When plant species compete, interactions regulating population growth generally occur at the neighborhood scale (Goldberg, 1987; Uriarte et al., 2004). Interference competition, including allelopathy, ordinarily has a local spatial structure. And, when propagule dispersal distance (inter-ratem distance in clonal species) is limited, plants compete preemptively for space at the neighborhood scale. Consequently, our model – which integrates preemption and interference – assumes that competitive interactions among nearest neighbors drive invasion and the dynamics of species’ abundances.

2.1. Model construction

Two clonal plant species compete on an \(L_x \times L_y\) lattice with periodic boundaries. Each lattice site represents the resources required to sustain a single individual (a ramet) of either species. The local occupation number at site \(x\) is \(n_i(x) = 0,1\) with \(i = 1,2\), referring to the resident and invader species, respectively. During a single simulated time unit, one Monte Carlo step per site [MCSS], \(L_x L_y\) sites are randomly sampled (with replacement) and updated.

An empty site may be occupied by species \(i\) through introduction from outside the environment, or through local propagation. Introduction of species \(i\) at an open site occurs as at constant rate \(\beta\). Each species has the same introduction rate, to avert any effect of propagule pressure on the outcome of competition; see Levine and Rees (2002). Local propagation into an open site has rate \(z_i \eta_i(x)\), where \(z_i\) is the individual-level propagation rate for species \(i\), and \(\eta_i(x) = (1/\delta) \sum_{j:m(x)} n_j(x)\) is the density of species \(i\) in the neighborhood around open site \(x\). \(m(x)\) is the set of nearest neighbors of site \(x\), and \(\delta\) is the number of sites in the neighborhood \((\delta = |m(x)|)\). We assume dispersal from an occupied site to only the four adjacent sites, so that \(\delta = 4\). Colonization can occur only at open sites. An occupied site opens through mortality. Individuals of each species suffer density-independent mortality at rate \(\mu\). An individual occupying site \(x\) experiences an increased mortality rate due to interference if \(m(x)\) includes any heterospecifics. That is, an individual of species \(i\) at site \(x\) has total mortality rate \(\mu + \theta_i \eta_j(x)\), \(i \neq j\), \(\theta_i \geq 0\) represents interference by species \(j\), and \(\eta_j(x)\) is the density of species \(j\) on the neighborhood around site \(x\).

Table 1: Definitions of model variables and parameters.

<table>
<thead>
<tr>
<th>Symbols</th>
<th>Definitions</th>
</tr>
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<tbody>
<tr>
<td>(L_x, L_y)</td>
<td>Lattice size</td>
</tr>
<tr>
<td>(x)</td>
<td>Location of lattice site</td>
</tr>
<tr>
<td>(n_i(x))</td>
<td>Occupation number for residents at site (x); (n_i(x) = 0, 1)</td>
</tr>
<tr>
<td>(m(x))</td>
<td>Occupation number for invaders at site (x); (m(x) = 0, 1)</td>
</tr>
<tr>
<td>(\delta)</td>
<td>Size of neighborhood around site (x); (\delta =</td>
</tr>
<tr>
<td>(\beta)</td>
<td>Common introduction rate at empty sites</td>
</tr>
<tr>
<td>(\eta_i(x))</td>
<td>Density of species (i) on (m(x))</td>
</tr>
<tr>
<td>(z_i)</td>
<td>Individual rate of propagule production, species (i)</td>
</tr>
<tr>
<td>(\mu)</td>
<td>Background mortality rate, both species</td>
</tr>
<tr>
<td>(\theta_i)</td>
<td>Mortality of species (i) due to interference by species (j)</td>
</tr>
<tr>
<td>(\bar{\beta})</td>
<td>Minimal propagation rate for persistence</td>
</tr>
<tr>
<td>(C)</td>
<td>Maximal propagation rate; (C = 0.8)</td>
</tr>
<tr>
<td>(R)</td>
<td>Curvature of (z_i \theta_i) tradecoff</td>
</tr>
<tr>
<td>(\rho^e)</td>
<td>Equilibrium single-species density</td>
</tr>
<tr>
<td>(\Delta)</td>
<td>Minimal propagation advantage required for invasion</td>
</tr>
<tr>
<td>(z_1, z_2)</td>
<td>Given (z_1, z_2 = z_1 + z_2)</td>
</tr>
</tbody>
</table>

![](Author's personal copy)
Summarizing transition rules for an arbitrary site \( x \), we have
\[
\begin{align*}
0^{\beta + 2z_i(\mu)} & \rightarrow 0, \\
0^{\beta + z_i(\mu)} & \rightarrow 1, \\
1^{\mu + 0z_i(\mu)} & \rightarrow 0, \\
2^{\mu + 0z_i(\mu)} & \rightarrow 0,
\end{align*}
\]
where 0, 1, 2 indicates whether a site is open, resident-occupied, or invader-occupied, respectively. Table 1 defines the symbols we use. We assume that each species can persist absent competition. Therefore, we restrict attention to the regime where \( x_i(\mu) \) is the critical propagation rate below which either species, in the other's absence, grows too slowly to avoid extinction (Oborny et al., 2005; O'Malley et al., 2006a).

2.2. Life-history constraint

An essential lesson of life-history theory is that increased allocation of resources to one trait advancing survival or reproduction comes at the expense of another trait (Bell and Koufopanou, 1986). Therefore, we assume that any increase in a species' level of interference reduces that species' clonal propagation rate. The functional dependence (tradeoff) has the form \( z_i^{\beta} + \theta_i^B = C_i^B \), where \( C_i \) is a constant, equal to the maximal feasible rate of propagation, and \( \theta_i \) defines the shape of the tradeoff (Giraldeau and Caraco, 2000). If \( 0 < R < 1 \), the cost of increasing \( x \) (i.e., the decrease in \( \theta \)) decreases as \( x \) increases. If \( R = 1 \), the cost of increasing \( x \) is constant, and if \( R > 1 \), the cost of increasing \( x \) increases at greater \( x \). Of course, any increase in \( R \) increases competitiveness of a species that employs both preemption and interference; the constraint moves farther from the origin as \( R \) increases. If a species does not exercise interference, we let its propagation rate vary on \( (x_i(\mu), 0.8) \). That is, we let \( C = 0.8 \).

2.3. Novel weapons

Under the novel weapons hypothesis, the invader exercises interference competition while the resident does not. That is, \( \theta_i = 0 \), and
\[
0 < \theta_2 < \sqrt{C_i - z_i(\mu)^B}. \]
Invasive plants may interfere with native competitors through several mechanisms. Some impact natives directly; others are mediated through a third species. Invaders can act as a disease reservoir (Eppinga et al., 2006; Borer et al., 2007), focus herbivory on native species (Prati and Bossdorf, 2004; Cipollini et al., 2008). Our analysis emphasizes the invader's tradeoff between propagation and interference across a range of native species lacking interference. Under the novel weapons hypothesis, the resident species does not compete through interference, and we let its reproductive rate vary to model variation in the strength of preemption the invader encounters.

2.4. Resistance zones

To study resistance zones, we let the resident exercise interference competition while the invader does not. That is, \( 0 < \theta_i < \sqrt{C_i - z_i(\mu)^B} \), and \( \theta_2 = 0 \). A resident species exercising interference competition may strongly resist invasion since its initial density (by definition) will be relatively high. Given biotic resistance combining preemption and interference, we vary the invader's propagation rate to consider a range of matches between the invader and the abiotic environment invaded.

2.5. Mutual interference

Under the assumption of mutual interference competition, we have \( 0 < \theta_i, \theta_2 < \sqrt{C_i - z_i(\mu)^B} \). For this case we assume that each species' resource allocation between propagation and interference follows the same tradeoff.

3. Mean-field approximation

The spatially homogeneous mean-field approximation to our model ignores effects of locally clustered growth. The mean-field (MF) model so offers comparison to results due to neighborhood-scale interactions; see Wilson (1998), Pascual and Levin (1999) or Cuddington and Yodzis (2000) for perspective. \( \rho_1 \) and \( \rho_2 \) represent the global densities of species 1 and species 2, respectively. Ignoring continuous introduction (letting \( \beta = 0 \)) we have a mean-field dynamics:
\[
\begin{align*}
\dot{\rho}_1 &= 2\rho_1(1-\rho_2-\rho_1)(\mu+\theta_2), \\
\dot{\rho}_2 &= 2\rho_2(1-\rho_1-\rho_2)(\mu+\theta_1),
\end{align*}
\]
\( \theta_i \) represents the increased mortality of species \( j \) induced by species \( i \); we scale \( \theta_i \) per unit density of species \( i \). In the absence of interspecific competition, species' persistence in the MF approximation requires only that each \( x_i > x_i(\mu) = \mu \). Letting \( \theta_i \geq 0 \), for \( i = 1, 2 \), we conduct a general stability analysis of the MF model's equilibria. Thereafter, we analyze the two-species system when the propagation–interference constraint introduced above applies.

3.1. Equilibria and stability

The dynamics has three boundary equilibria and one positive equilibrium. Mutual extinction, designated equilibrium E1, cannot be stable since each \( x_i > \mu \). At equilibrium E2, species 1 competitively excludes species 2:
\[
\begin{align*}
E2: \quad & \rho_1^* = 1 - \mu \quad \rho_2^* = 0.
\end{align*}
\]
Species 2 excludes species 1 at equilibrium E3:
\[
E3: \quad \rho_1^* = 0, \rho_2^* = 1 - \frac{\mu}{\mu_2}.
\]
Finally, a positive (internal) equilibrium, E4, can be expressed as
\[
\begin{align*}
\rho_1^* &= \frac{\theta_2(z_2 - \mu) + \mu(z_2 - \xi_1)}{\theta_1 z_1 + 2z_2}, \\
\rho_2^* &= \frac{\theta_1(z_1 - \mu) + \mu(z_1 - \xi_2)}{\theta_2 z_2 + 2z_1}.
\end{align*}
\]
As noted above, when competition is strictly preemptive (\( \theta_i = 0 \)), the species cannot coexist under homogeneous mixing.

From Appendix A, local stability of invader extinction requires
\[
\mu \left( \frac{z_2}{\xi_2} - 1 \right) + \theta_i \left( \mu - 1 \frac{\mu}{\xi_1} \right) < 0.
\]
Since \( \mu < \xi_1 \),
\[
\theta_i \left( \mu - 1 \frac{\mu}{\xi_1} \right) \leq 0.
\]
Therefore, any interference by the resident tends to stabilize invader extinction by increasing invader mortality. That is, interference by the common species can help prevent advance of the rare species, even if the rare species has the greater propagation rate. Clearly, if \( x_1 > x_2 \) and \( \theta_i \geq 0 \), the resident species repels the invader. Note that \( \theta_i \), the invader’s level of interference competition, does not appear in expression (8). Hence the invader cannot increase its growth rate when rare through interference; only increased propagation promotes mean-field invasion.

To elaborate, the invader advances from rarity only if expression (8) is reversed. From Appendix A, assuming \( \theta_1 > 0 \), the rare species
invades iff:
\[ x_2 - x_1 > \theta_1 \left( \frac{z_1}{\mu} - 1 \right) > 0. \]  \hspace{1cm} (9)

To invade successfully, the rare species must have the greater propagation rate, and must not suffer too much interference from the common species. Expression (9) specifies a minimal propagation advantage required for invasion by species 2; Section 3.4 asks how this requirement depends on the propagation–interference constraint. Appendix A shows that if species 2 invades the resident, then equilibrium \( E_3 \) is locally stable. That is, if species 2 advances when rare, it will grow to exclude species 1.

### 3.2. Bistability

Bistability requires that local-stability conditions for both single-species equilibrium nodes (\( E_2 \) and \( E_3 \)) hold simultaneously. Given bistability, initial conditions determine the outcome; at some point, the more abundant species “wins.” From Appendix A, the common species (1 or 2) repels its rare competitor if

\[ \theta_1 \left( 1 - \frac{z_1}{\mu} \right) < z_1 - z_2 \quad \text{and} \quad \theta_2 \left( \frac{z_2}{\mu} - 1 \right) > z_1 - z_2, \]  \hspace{1cm} (10)

If \( z_1 > z_2 \), the first expression must hold, since

\[ \theta_1 \left( 1 - \frac{z_1}{\mu} \right) \leq 0. \]

The second expression can hold simultaneously if \( \theta_2 \) is relatively large, and the difference between propagation rates is not too large. If \( z_2 > z_1 \), the second expression must hold, since

\[ \theta_2 \left( \frac{z_2}{\mu} - 1 \right) > 0 \]

and symmetric conditions promoting bistability are clear. Bistability can arise even if the species with the greater propagation rate does not exert interference. For example, if \( z_1 > z_2 \), the system can be bistable when \( \theta_1 = 0 \) (provided, of course, \( \theta_2 > 0 \)).

Fig. 1 shows how the mean-field dynamics can flow in the \((\rho_1, \rho_2)\) phase space. One example plots competitive asymmetry. Species 2 advances when rare, and when common, excludes species 1. The other plots show bistability, one for a symmetric (identical species), and one for an asymmetric, domain of attraction.

### 3.3. Coexistence?

Coexistence (local stability of a positive equilibrium) requires that each species invade the other. Together, the conditions for mutual invasion indicate that coexistence requires (Appendix A):

\[ \theta_1 \left( 1 - \frac{z_1}{\mu} \right) > z_1 - z_2 \quad \text{and} \quad \theta_2 \left( 1 - \frac{z_2}{\mu} \right) > z_2 - z_1. \]  \hspace{1cm} (11)

Since each \( z_i > \mu \), each

\[ \left( 1 - \frac{z_i}{\mu} \right) < 0. \]

The LHS of each of these expressions must then be non-positive. But one \((z_1 - z_2)\) must be positive, so that the two expressions cannot be true simultaneously. Hence the MF does not permit mutual invasion, and so does not admit competitive coexistence.

### 3.4. Propagation–interference constraint and biotic resistance

Invoking the tradeoff between propagation and interference, the condition for the invader’s advance when rare, expression (9),
of successful invasion. Greater mortality, with 
resident’s resistance, an invader must have a sufficiently greater advantage needed for successful invasion. For Appendix A.1: Dotted lines denote $\alpha$ for each species, and neither species is viable in the hatched region.

$\alpha_2 > \alpha_1 + \left( \frac{\alpha_1}{\mu} - 1 \right) \sqrt{C^2 - \alpha_2^2}$. \hfill (12)

Any increase in $R$ relaxes the constraint and increases the range of parameter combinations where the resident repels the invader (Fig. 2). Biotic resistance increases as $R$ increases under homogeneous mixing, since only the resident benefits from interference. Increasing background mortality ($\mu$) increases the likelihood of successful invasion. Greater mortality, with $\theta_1$ and the $\alpha_2$ fixed, increases the density of sites available for colonization, and so decreases the parameter range where the resident repels the invader; see Fig. 2.

Under homogeneous mixing, the range of invaders (range of $\alpha_2$ levels) a given resident excludes varies with the shape of the life-history constraint. We summarize results on biotic resistance versus invasion from Appendix A.1, by focusing on the values of $R$ used to construct Fig. 2.

Consider the linear tradeoff ($R = 1$) first. To overcome the resident’s resistance, an invader must have a sufficiently greater propagation rate. Let $A_s$ represent the minimal propagation advantage needed for successful invasion. For $R = 1$, we have from Appendix A.1:

$A_s = (C - \alpha_1)(\alpha_1 - \mu)/\mu$. \hfill (13)

Clearly, $A_s$ declines as $\mu$ increases; greater background mortality increases open-site density, making invasion easier. The propagation advantage required for invasion reaches a maximum at

$\alpha_1 = (C + \mu)/2$, implying that $\theta_1 = (C - \mu)/2$. Hence the resident that maximizes biotic resistance propagates at an increased rate, and exercises less interference, as $\mu$ increases.

For some values of $\alpha_1$, the required propagation advantage implies a level of $\alpha_2$ exceeding $C$, i.e., exceeding the maximal feasible rate of propagation. For $R = 1$, all feasible invaders are so resisted if $\alpha_1 > 2\mu$; see Appendix A.1 and Fig. 2. When $\alpha_1$ exceeds $2\mu$, the resident at equilibrium occupies more than 1/2 the sites. Then, given the linear propagation–interference tradeoff, no feasible invader ($\alpha_2 < C$) can advance when rare. An increased allocation to interference by the resident reduces its equilibrium density through the propagation–interference tradeoff. Consequent thinning of the resident’s density below a critical level, although each individual exerts stronger interference, can leave enough unoccupied sites that an invader with a sufficient propagation rate can invade successfully. The quantitative result is not so simple when the two species have different background mortality rates, but qualitatively similar pictures emerge.

Now let $R = 2$, relaxing the propagation–interference tradeoff, and so increasing the resident’s biotic resistance. Under homogeneous mixing, successful invasion requires a minimal propagation advantage of

$A_s = (C^2 - \alpha_1^2)^{1/2} / (\alpha_1 - \mu)/\mu$. \hfill (14)

Increasing $R$ increases the minimally required propagation advantage for invasion, since $(C^2 - \alpha_1^2)^{1/2} > C - \alpha_1$. $A_s$ reaches a maximum at a mix of propagation and interference. From Appendix A.1, the

![Fig. 2. Success or failure of invasion under the mean-field approximation. Each subplot’s abscissa is the resident’s propagation rate, $\alpha_1$. The resident’s level of interference is given by the life-history constraint, $\alpha_2^2 + \mu^2 = C^2$. The MF invasion criterion is insensitive to interference by the invader. Black: parameter combinations where invader’s growth is positive when rare; invasion succeeds. White: resident repels invader; invasion fails. Separation of these equilibrium phases follows from expression (12). Note that the diagonal (solid line) separates invader success from failure when each species competes through site preemption only. Rows, top to bottom, show that invasion increases as background mortality increases. Columns, left to right, show that invader’s success declines as resident propagation increases for given level of interference. Dotted lines denote $\alpha_2(\mu)$ for each species, and neither species is viable in the hatched region.](image-url)
largest propagation advantage required for invasion occurs at
\[ z_1 = \frac{\mu}{4} + \sqrt{\frac{\mu^2 + 8C^2}{4}}. \]  
(15)

Again, once \( z_1 \) is sufficiently large, no feasible \( z_2 \) can overcome biotic resistance. For \( R=2 \), all feasible invaders \( (z_2 \leq C) \) are resisted if
\[ z_1 > \mu \left[ 1 + \frac{(C-z_1)^{1/2}}{(C+z_1)^{1/2}} \right]. \]  
(16)

where both square roots are positive. As \( z_1 \) approaches its lower bound, \( z_1 \rightarrow \mu \), expression (16) cannot hold, and feasible invaders with sufficiently large propagation rates advance. But as \( z_1 \) approaches its upper bound, \( z_1 \rightarrow C \), expression (16) must hold, since \( C > \mu \). The pattern in Fig. 2, for subplots with \( R=2 \) follows; see Appendix A.1. The resident species resists feasible invaders as long as it maintains a large enough equilibrium density.

Finally, let \( R=0.5 \), so that the tradeoff strongly limits the resident’s competitiveness. The minimal propagation advantage for successful invasion is
\[ A_2 = \frac{C^{1/2} - z_1^{1/2}}{z_1^{1/2}}. \]  
(17)

Decreasing \( R \) reduces the propagation advantage required for invasion. As in the previous cases, an intermediate propagation–interference combination maximizes the advantage an invader must have to grow when rare. \( A_2 \) reaches a maximum, and the resident’s biotic resistance is strongest, at
\[ z_1 = \frac{1}{2m} (C+2\mu)^{1/2}. \]  
(18)

Eq. (18) matches the values for \( \mu = 0.1 \) and 0.2 in the first column of Fig. 2.

Resident propagation level \( z_1 \) resists invasion by all feasible \( z_2 \) if
\[ z_1 > \mu \left[ 1 + \frac{C^{1/2} + z_2^{1/2}}{C^{1/2} - z_1^{1/2}} \right]. \]  
(19)

Since the RHS of expression (19) exceeds \( 2\mu \), the range of \( z_1 \) levels excluding all feasible \( z_2 \) is smaller than cases where \( R \geq 1 \). In Fig. 2, for \( \mu = 0.1 \) and 0.2, no value of \( z_1 \) can exclude all feasible invaders. As \( z_1 \) approaches its lower bound, \( \mu \), and as \( z_1 \) approaches its upper bound, \( C \), expression (19) cannot hold. Too much propagation reduces interference so low that the dynamics approach that of preemptive competition only (Korniss and Caraco, 2005). Too much interference reduces the resident’s density so low that invaders readily find unoccupied sites for colonization.

If expression (19) holds on some intermediate interval of \( z_1 \), invaders with a sufficiently large propagation rate will invade both very low and very high levels of \( z_1 \), but will fail to overcome biotic resistance at intermediate \( z_1 \). We observe this pattern in the upper left subplot of Fig. 2. Residents mixing propagation and interference repel all invaders, but residents with quite small, and those with quite large, propagation rates can be invaded.

Under homogeneous mixing, the outcome of competitive invasion depends directly on the resident’s level of interference, but not on the invader’s level of interference. Analysis of the propagation advantage an invader needs to grow when rare finds that residents with an intermediate mix of propagation and interference generate the strongest biotic resistance, though quantitative detail varies with the shape of the propagation–interference interference constraint. If the life-history tradeoff is linear or concave \( (R \geq 1) \) there will be a density beyond which the resident can resist any invader, although the timescale of competitive exclusion will be shortest at an intermediate mix of propagation and interference. If the life-history constraint is convex \( (R < 1) \) residents with both low and high propagation may be more prone to invasion than a resident with an intermediate mix of traits.

4. Pair approximation

Pair approximation (PA) incorporates correlations of the occupation status of nearest neighboring sites into a dynamics (Dickman, 1986; Bauch and Rand, 2000; van Baalen, 2000). By assuming a minimal spatial structure, PA predicts equilibrium densities of individual-based models more accurately than do MF models (Ellner et al., 1998; Caraco et al., 2001). Our PA addresses the impact of local clustering, generated by dispersal limitation, for invasion. Appendix B presents details.

The PA tracks global densities \( \rho_i(t) \), and conditional densities \( q_{ij}(t) \) representing the likelihood that a site is in state \( j \), given that a neighboring site is in state \( i \) (Sato and Iwasa, 2000; O’Malley et al., 2006a). The three global densities \( (\rho_0(t), \rho_1(t), \rho_2(t)) \) imply nine local densities \( (q_{ij}) \). However, the PA’s dimension is limited by simple constraints:
\[ p_0 + \rho_1 + \rho_2 = 1, \]
\[ q_{0j} + q_{1j} + q_{2j} = 1, \]
(20)

where \( i,j = 0,1,2; i \neq j \); we suppress time dependence for simplicity. These constraints leave only 5 of the 12 total variables independent. Since species 2 is the invader, we track \( \rho_1, \rho_2, q_{2j}, q_{1j} \). Note that the dynamics of \( q_{1j} \), the conditional density of a resident given an invader at a neighboring site, can depend on a novel-weapons effect when \( q_2 > 0 \).

The dynamics of the global densities, like the mean-field approximation, accounts introduction to empty sites, local propagation, background mortality, and death due to interference competition. For the resident,
\[ \dot{\rho}_1 = \beta \rho_0 + \rho_1 q_{01} - \rho_1 \mu q_{12} - \rho_1 \rho_2 t. \]  
(21)

Compared to the MF model, both local propagation and mortality due to interference depend on local, rather than global, densities. Hence the PA models both site preemption and interference as effects of clustered growth.

To model the dynamics of a local density, we first write the dynamics of a doublet \( \rho_{ij} \), a global density, where \( \rho_{ij} = \rho_i q_{ij} \). The PA’s doublet dynamics \( (d\rho_{ij}/dt) \); see Appendix B) introduces the triplets \( q_{2ij} \) and \( q_{12j} \). Ordinary PA assumes that neighbors of neighboring sites are weakly correlated, and lets \( q_{2i} = q_{2c} \) and \( q_{122} = q_{12} \). The approximation closes the system of equations, permitting us to write an invasion criterion (Iwasa et al., 1998).

For the invasion analysis, assume that an introduction of species 2 has occurred. Invasion either succeeds or fails before the next introduction event occurs (since \( \beta < \mu < z_1 \)). Successful invasion requires that the invader have a positive growth rate when rare; \( \rho_2 > 0 \) as \( \rho_2 \rightarrow 0 \). From Appendix B, this condition yields the invasion criterion:
\[ 1 - q_{2j} - q_{1j} > \frac{\mu + \theta_1 q_{1j}}{2z_2}. \]  
(22)

The left side represents the density of open sites neighboring an invader, \( q_{0j} \). The right side is the ratio of death rate to birth rate for the invader. Successful invasion, then, requires that the invader colonize a neighboring, empty site before it dies. The death rate sums background mortality and averaged mortality from the resistance zone about a resident neighboring the invader. The simplicity of the invasion criterion masks an important biological difference between the MF criterion and the neighborhood-scale
condition for invasion. In the PA condition, the local density $q_1$, hence the local density of open sites $q_{1|2}$, depends on $\theta_1$, invader interference (see Appendix B). That is, the neighborhood-scale invasion criterion reveals a role for novel weapons, contrasting to the MF result. Consequently, an interfering invader might invade a resident species despite the resident having the greater propagation rate. The right side of the criterion, of course, includes the effect of interference by the resident.

To compare the PA invasion criterion to both the MF and simulation of the full spatial model (see below), we evaluated expression (22) numerically. We plotted invasion results for our three cases: only the invader interferes (novel weapons), only the resident interferes (resistance zones) and both species exercise interference competition. Whenever a species was an interference competitor, we invoked the propagation–interference constraint.

Fig. 3 shows the invasion results when the invader, and not the resident, is the interference competitor. That is, the invader obeys the propagation–interference constraint, and $\theta_1 = 0$ for any $\alpha$, isolating the resistance zone effect. A resident can repel an invader with a much higher propagation rate than its own through local interference. Increasing $R$ now reduces the range of parameter combinations permitting invasion, and increasing $\mu$ promotes invader success—results symmetrically opposite to those for novel weapons. When only the resident can interfere, a greater value for $R$ reduces the pleiotropic cost of interference. Increasing $\mu$ opens more space for the invader; greater background mortality renders a given level of interference less effective as a competitive mechanism.

Fig. 5 shows invasion results when both species exercise interference, and so both are constrained by the propagation–interference tradeoff. The resident’s initial density is high locally and globally. Therefore, it can employ interference to repel the invader over a much greater range of parameters than when both species lack interference. The contribution of interference to biotic resistance declines at small $R$, and at greater $\mu$; the results for $(R = 0.5, \mu = 0.2)$ match the case where competition is strictly preemptive. In most of the plots substantial regions of bistability appear. A species with intermediate levels of both propagation and interference can repel a broad range of propagation–interference combinations. And, when rare, the intermediate combination is repelled by species within that broad range, as long as $R \geq 1$. Bistability implies that the “common” species has an advantage. We obtained these results by evaluating expression (22), which invokes local densities; hence common, here, means common at a local scale.

Mutual interference does not permit coexistence under PA (Fig. 5); the invasion results resemble the case where only the resident can interfere. But for sufficiently large $R$ and small enough $\mu$, a resident that invests solely in propagation, and not in interference,
**Fig. 4.** Resistance zones under pair approximation. Success or failure of invasion according to the PA invasion criterion; only the resident exercises interference. Black: parameter combinations where invader’s growth is positive when rare; invasion succeeds. White: resident repels invader; invasion fails.

**Fig. 5.** Success or failure of invasion generated by the PA invasion criterion; both species exercise interference. Black: parameter combinations where invader’s growth is positive when rare; invasion succeeds. White: resident repels invader; invasion fails.
can be invaded by a species that mixes propagation and interference (rightmost columns of invasion plots). The resident, with an initially high density and no interference capacity, is vulnerable to a clustered invader that can open sites via interference competition. Once an invader cluster becomes established, its growth requires interference, since the resident leaves only a small density of open sites at its equilibrium abundance.

5. Simulation of individual-based model

We simulated the individual-based spatial model with \( L_x = L_y = L = 256 \), and \( \beta = 0.001 \). We tracked the global densities of each species \( \rho_i(t) \), and recorded a successful invasion when the invader reduced the global density of the resident species to \( \rho_1(t)/2 \) within 20,000 MCSS time steps.

We attribute some differences between results of the discrete, stochastic model and its pair approximation to the different treatments of spatial clustering. PA, by construction, averages over nearest-neighbor length correlations and assumes that spatial correlations drive the dynamics of global densities. In simulation of the full model, invader clusters will form even while the invader remains globally rare. Clustering impacts the relative frequency at which individuals experience local self-regulation versus interspecific competition (O’Malley et al., 2010). These frequencies, in turn, affect cluster persistence and invasion success. PA can underestimate the consequences of local variation in spatial correlations, and so may not faithfully reproduce the stochastic model’s behavior.

We simulated invasion for novel weapons, resistance zones, and the case where both species interfere competitively. Fig. 6 shows invasion results when the invader, and not the resident, interferes competitively in the individual-based spatial model; the invader obeys the propagation–interference constraint, and \( \theta_1 = 0 \). Invader interference promotes successful invasion more frequently in simulation than under pair approximation. That is, we note a stronger novel-weapons effect. Invader clustering increases the advantage of local interference competition against a resident lacking interference. Even when the propagation-cost of interference is steep \((R < 1)\) an invader may advance and exclude a resident with a greater propagation rate. When interference is less costly \((R > 1)\) most invaders succeed, except those with a very low propagation rate.

Fig. 6 shows invasion results when the resident, and not the invader, interferes competitively in the individual-based model. The resident obeys the propagation–interference constraint, and \( \theta_2 = 0 \). Interference allows a resident to repel invaders with much greater rates of local propagation. For the resistance-zone effect, PA predicts the results of the individual-based model very accurately across all parameter combinations we simulated.

The simulation model’s results for the case where both species exercise interference competition appear in Fig. 8. When the cost of interference is high \((R > 0.5)\) the results are very close to those for preemptive competition only. That is, the species with the greater rate of propagation wins in most cases. Compared to PA, invader clustering in the simulation model produces more cases of successful invasion at low background mortality. At lesser costs of interference \((R = 1, 2)\) residents mixing intermediate levels of propagation and interference repel most invading species, particularly when background mortality is not too large. Residents that invest too much in interference \((\text{low } z_i)\) or too little are
Fig. 7. Resistance zones in the simulation model. Each point represents combined results of 20 simulations. Black: parameter combinations where invader’s growth is positive when rare; invasion succeeds. White: resident repels invader; invasion fails. Gray: as described above. Increased background mortality reduces the difference between the resident’s initial density and the invasion threshold, and we record more invasions.

Fig. 8. Simulation model when both species interfere. Each point represents combined results of 20 simulations. Black: parameter combinations where invader’s growth is positive when rare; invasion succeeds. White: resident repels invader; invasion fails. Gray: as described above.
susceptible to invasion by species with a more balanced mix of propagation and interference.

In a separate exercise, we released both species from the propagation–interference constraint and searched the parameter space for coexistence. We set \( \mu = 0.1, \beta = 0.001 \), and let \( L = 64 \). The simulations ran for 100,000 MCSS, and the threshold for coexistence was \( p_1 > 0.05; i = 1.2 \), well above the expected density due to introduction alone. The search yielded no evidence of competitive coexistence, as our analytical models predict.

6. Discussion

If species mix homogeneously, we find that a rare invader gains no advantage through interference competition (Case and Gilpin, 1974). But if competitive interactions occur at the local scale, an invader can employ interference to advance against a resident species with a greater rate of local propagation (Figs. 3 and 6). Invaders are rare globally but locally clustered in our spatial models. Invaders can use interference to reduce resident density at the perimeter of these clusters, where local invader density is sufficiently high to compete effectively for open sites. That is, some investment in interference by the clustered invader leaves enough propagule production to occupy the sites opened.

Interference by the resident has a strong effect under homogenous mixing; few invaders advance successfully unless interference is costly and background mortality is high (Fig. 2). Under pair approximation, interference by the resident restricts invasion both when the resident, but not the invader, interferes, and when both species exercise interference. The resistance zone effect also appears in simulation of the individual-based model. When both species can interfere, successful invasion is far more common in the simulation model. The difference between the full spatial model and its pair approximation lies in the impact of invader clusters larger than neighborhood size (hence, longer correlation distances) generated by the individual-based model.

A similar distinction between spatially implicit and explicit competition has been identified in allelopathic bacteria. Chao and Levin (1981) studied competition between colicin producing (allelopathic) and colicin sensitive strains of Escherichia coli. The competitive outcome in a well mixed system (liquid culture) depended on initial concentrations of each strain; i.e., the fully mixed system showed bistability. However, in a spatially structured environment (agar plate) the colicin producing strain displaced the colicin sensitive strain, even when initially rare. Durrett and Levin (1997) simulated a spatially explicit model and demonstrated that for some combinations of propagation rates and interference, the allelopathic strain advances even from a low initial density. Together, these results indicate that bacterial allelopathy fails to promote a rare invader’s growth under homogenous mixing, but may advance the same invader’s growth when interactions are locally structured.

When both species exhibit interference in our model, the plotted invasion results reveal regions of bistability (if only one species interferes, one cannot meaningfully reflect invasion plots about the diagonal). Relatively extensive regions of parameter values produce bistability under homogenous mixing; bistability declines as spatial structure increases, and nearly disappears in the simulation model (Fig. 8). Given a sufficiently long time, we would anticipate that the spatial process on a large, but finite, lattice would result in competitive exclusion (Liggett, 1980).

6.1. Novel weapons: role of invader clustering

We found that interference competition by a rare invader promotes its growth when interactions are structured locally, but not under homogeneous mixing. To draw a comparison, we recently modeled preemptive spatial competition under tradeoffs between propagation and mortality rates (Allstadt et al., 2009). That study examined an often overlooked difference between “environments” in models for spatial invasion: an open environment versus an environment occupied by a resident competitor. An unoccupied environment assumes invasion into an unstable equilibrium phase; a resident-occupied environment rests at metastable equilibrium (Korniss and Caraco, 2005). Invasive growth in an open environment favored increased propagation rate, despite the increase in density-independent mortality rate. However, invasion of an occupied environment always favored decreased mortality rate, despite the consequent reduction in propagation. During invasive expansion in an open environment, individuals at the perimeter of invader clusters readily find open sites for colonization, despite nearest-neighbor propagule dispersion. Relatively high mortality can be tolerated, since the fraction of propagules finding open sites is so large. In an occupied environment, opportunities for colonization decline; the density of open sites at a cluster’s perimeter decays to the within-cluster level. Consequently, low propagation can be tolerated – since most propagules fall on occupied sites – and low mortality implies that individuals can wait until an opportunity for reproduction occurs (Allstadt et al., 2009).

Turning to tradeoffs between propagation and interference, some parallels apply. When dispersal and interference have a nearest-neighbor scale, invaders grow as spatial clusters. Hence propagules will be wasted on occupied sites. Interference can promote invasion by decreasing competitor density at a cluster’s perimeter, even if the invader’s within-cluster density declines due to the propagation-rate reduction. Clustered invaders may focus interference across the same sites, increasing the cluster’s persistence and, perhaps, its expansion. As neighborhood size increases, dispersal radius increases. Under homogeneous mixing, an invader always has sites available for colonization, but its global rarity means that any interference will not impact the resident’s density, and so will not promote invasion.

Nakamura et al. (1997) model nearest neighbors playing an iterated prisoner’s dilemma. Each individual has either a conditionally cooperative (tit-for-tat) or an always-defect phenotype. The cumulative payoff from repeated play governs an individual’s mortality probability. Mutual cooperators enjoy greater survival than do mutual defectors, but defecting against cooperation may open a site for the defector’s replication. At the scale of phenotypic evolution, Nakamura et al. (1997) effectively allow an individual to reduce a neighbor’s survival, at a cost to its own survival. Our model assumes that interference reduces a heterospecific neighbor’s survival, and the cost of interference is reduced reproduction.

Nakamura et al. (1997) find the minimal probability of repeated play required to fix the cooperative strategy. The critical values vary with the degree of spatial structure. Under the model’s homogenous-mixing approximation, a rare cooperative strategy cannot invade a resident defector population. However, spatially structured interactions allow clusters of mutual cooperators to form, so that tit-for-tat can successfully invade an all-defect population. Given spatially structured play, evolution of cooperation depends on the interaction of increased survival between neighboring cooperators and the advantage of reducing a neighbor’s survival by defecting. To offer a parallel, interference in our model enhances a competitor’s advance from rarity only when interactions are structured spatially. Within a cluster of conspecifics, there is no interference-induced mortality. But at the cluster’s perimeter, where the invader and resident interfere, individuals of each species suffer increased mortality due to interference. To a limited extent, this mortality difference resembles an invading tit-for-tat strategy. Within-cluster “cooperators”
live longer; individuals at the cluster’s boundary “defect” and reduce a competitor’s survival.

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Appendix A. Mean-field stability

The Jacobian for the spatially homogeneous MF dynamics is

\[
J = \begin{bmatrix}
    x_1 - \mu - 2x_1 \rho^0_1 - \rho^0_2 (x_1 + \theta_2) & -\rho^0_1 (x_1 + \theta_2) \\
    -\rho^0_2 (x_2 + \theta_1) & x_2 - \mu - 2x_2 \rho^0_2 - \rho^0_1 (x_2 + \theta_1)
\end{bmatrix}
\]

(A.1)

At equilibrium $E_1$, mutual extinction, the two eigenvalues are $x_i - \mu$, for $i = 1, 2$. The quantities are positive, by assumption, so that mutual extinction is unstable.

At $E_2$ the resident excludes the invader. The associated Jacobian yields

\[
J(E_2) = \begin{bmatrix}
    x_1 - \mu - 2x_1 (1 - \frac{\rho}{C_1}) & -(1 - \frac{\rho}{C_1})(x_1 + \theta_2) \\
    0 & x_2 - \mu - (1 - \frac{\rho}{C_1})(x_2 + \theta_1)
\end{bmatrix}
\]

(A.2)

The two eigenvalues are

\[
\lambda_1(E_2) = \mu \left( \frac{2}{x_1} - 1 \right) + \theta_1 \left( \frac{\mu}{x_1} - 1 \right) \tag{A.3}
\]

\[
\lambda_2(E_2) = \mu - \frac{x_2}{x_1} < 0 \tag{A.4}
\]

Local stability requires $\lambda_1(E_2) < 0$.

Species 2 invades (i.e., extinction of species 2 is unstable) if $\lambda_1(E_2) > 0$. If $x_2 > x_1$, then

\[
\left( \frac{2}{x_1} - 1 \right) > 0,
\]

promoting invasion by species 2 when species 1 rests at single-species equilibrium $E_2$. When $\theta_1 > 0$, species 2 invades iff:

\[
x_2 - x_1 > \theta_1 \left( \frac{x_1}{\mu} - 1 \right) > 0. \tag{A.5}
\]

The middle term of expression (A.5) indicates the propagation advantage the invading species requires to overcome the resident’s biotic resistance.

Next, consider $E_3$, where species 2 competitively excludes species 1. Evaluating the Jacobian, we have

\[
J(E_3) = \begin{bmatrix}
    x_1 - \mu - (1 - \frac{\rho}{C_2})(x_1 + \theta_2) & 0 \\
    -\left(1 - \frac{\rho}{C_2}\right)(x_2 + \theta_1) & x_2 - \mu - 2x_2 (1 - \frac{\rho}{C_2})
\end{bmatrix}
\]

(A.6)

The symmetry between $J(E_2)$ and $J(E_3)$ extends to the eigenvalues. From Eq. (A.6), we obtain

\[
\lambda_1(E_3) = \mu \left( \frac{2}{x_2} - 1 \right) + \theta_2 \left( \frac{\mu}{x_2} - 1 \right) \tag{A.7}
\]

\[
\lambda_2(E_3) = \mu - x_2 < 0 \tag{A.8}
\]

Species 2 cannot exclude species 1 iff

\[
x_1 - x_2 > \theta_2 \left( \frac{x_2}{\mu} - 1 \right) > 0. \tag{A.9}
\]

Clearly, reversing subscripts in expression (A.5) yields expression (A.9). More importantly, we see that if expression (A.5) holds, then $\lambda_1(E_3) < 0$. Hence, if species 2 can invade species 1, $E_3$ is the only stable equilibrium, and species 2 advances to exclude species 1 competitively.

Now consider bistability. When common, species 1 repels invasion by species 2 if $\lambda_1(E_2) < 0$. When species 2 is common, it repels invasion by species 1 if $\lambda_1(E_3) < 0$. Then bistability requires that expressions (A.3) and (A.7) both hold. The combined result appears as expression (10) in the text.

From our analysis of the single-species equilibria, the two species can coexist iff $\lambda_1(E_2) > 0$ and $\lambda_1(E_3) > 0$. The first expression implies that when species 1 rests at its single-species equilibrium, species 2 advances from rarity. The second expression reverses roles of common and rare. In the text we show that the mean-field dynamics does not admit competitive coexistence.

A.1. Invoking the life-history constraint

Using the mean-field stability analysis, we ask how the propagation–interference tradeoff affects biotic resistance. At the homogeneous-mixing model’s equilibrium $E_2$, we have $(\rho^0_1 = 1 - \mu/x_1 > 0, \rho^0_2 = 0)$. The resident resists invasion if both eigenvalues $\lambda_i(E_2) < 0$; $i = 1, 2$, $\lambda_2(E_2) = \mu - x_2 < 0$, by the requirement that $\rho^0_2 > 0$. Hence, success or failure of biotic resistance depends on the sign of $\lambda_1(E_2)$, given by Eq. (A.3). Since $\theta_1 > 0$, $\lambda_1(E_2) < 0$ whenever $x_2 < x_1$, and the resident resists invasion. However, the invader may overcome biotic resistance if $x_2 > x_1$; if the invader’s growth advantage is large enough, $\lambda_1(E_2) > 0$ and invasion succeeds.

$\lambda_1(E_2) = 0$ locates the equilibrium phase boundary, separating $(x_1, x_2)$ combinations where the resident resists invasion from those combinations where the invader advances. From above, $\lambda_1(E_2) = 0$ requires $x_2 > x_1$.

Fixing $x_1$, fixes $\theta_1$ through the constraint. Then we find $\tilde{x}_2$, the invader propagation rate where $\lambda_1(E_2) = 0$. From the text, each $x_1 \in (\mu/C)$. So, if $C < \tilde{x}_2$, no feasible $x_2$ can overcome biotic resistance at the given level of $x_1$. But if $C > \tilde{x}_2$, any $x_2$, such that $\tilde{x}_2 < x_2 < C$, invades and excludes the resident. Letting $\lambda_1(E_2) = 0$, and recalling that $\theta_1 = (C^2 - \tilde{x}_1^2)/C$, yields

\[
\tilde{x}_2 = x_1 + [\left( C^2 - x_1^2 \right)^{1/2}/R (x_1 - \mu)/\mu]. \tag{A.10}
\]

The second term on the RHS of expression (A.10) specifies the minimal propagation advantage ($\tilde{x}_2 - x_1$) required for successful invasion.

We analyze biotic resistance with respect to values of $R$ used in Fig. 2; generalization is straightforward. Let $R = 1$. For given $x_1$: $\tilde{x}_2 = x_1 + [(C - x_1)/(x_1 - \mu)/\mu]$. \tag{A.11}

(C – $x_2$) is the resident’s level of interference. Setting $\delta_3(\tilde{x}_2 - x_1) = 0$, we see that the minimal propagation advantage required for invasion attains a maximum at $x_1 = (C + \mu)/2$. Then $\theta_1 = (C - \mu)/2$.

A more ecologically useful application of the formula for $\tilde{x}_2$ follows. The resident resists all feasible $x_2$ if $C < \tilde{x}_2$, which reduces to $x_1 > 2\mu$. That is, $C < \tilde{x}_2$, and biotic resistance cannot be overcome, if $\mu/x_1 < 1/2$, implying that $\rho^0_2 > 1/2$. Alternatively, invasion succeeds if both $x_2 > 2\mu$ and $\tilde{x}_2 < x_2 < C$. Subplots for $R = 1$ in Fig. 2 demonstrate these results.

If $R = 2$, the tradeoff relaxes. Given $x_1$, $\lambda_1(E_2) = 0$ if $\tilde{x}_2 = x_1 + [(C^2 - x_1^2)^{1/2}/(x_1 - \mu)/\mu]$. \tag{A.12}

Comparison with the case where $R = 1$ shows that the minimal propagation advantage for successful invasion increases as $R$ increases, since $(C^2 - x_1^2)^{1/2} > C - x_1$.

A mix of the resident’s propagation and interference maximizes the propagation advantage an invader must achieve to succeed.
We have
\[
\hat{\sigma}_1, \hat{\sigma}_2 = \mu^{-1} \left[ \left( C - 2x_1^* \right)^{1/2} - \frac{2x_1(x_1 - \mu)}{\left( C - 2x_1^* \right)^{1/2}} \right].
\] (A.13)

The value of \( \sigma_1 \) where \( \hat{\sigma}_1 \) reaches a maximum is given by Eq. (15).

If we suppose that the resident, with propagation rate \( \sigma_1 \), excludes all feasible invaders, then \( C < \hat{\sigma}_2 \), which reduces to
\[
\sigma_1 > \mu \left[ 1 + \frac{C - 2x_1^* \left( C + 2x_1^* \right)^{1/2}}{C - \mu} \right].
\] (A.14)

Negative square roots in the last expression violate definitions of biological terms. Note that the RHS of expression (A.14) does not exceed 2\( \mu \). The increase in \( R \) has decreased the minimal \( \sigma_1 \) resisting invasion by all feasible \( \sigma_2 \); see Fig. 2. Invasion succeeds if the given \( \sigma_1 \) is too small to satisfy expression (A.14), and \( \hat{\sigma}_2 < 2 \mu \). Put another way, as \( \sigma_1 \) approaches its lower bound, \( \sigma_1 \rightarrow \mu \), expression (A.14) cannot hold. But as \( \sigma_1 \) approaches its upper bound, \( \sigma_1 \rightarrow C \), expression (A.14) must hold, since \( C > \mu \). Hence the phase boundary will be crossed once, at most, as \( \sigma_1 \) increases.

When \( R = 0.5 \), \( \lambda_1(E2) = 0 \) at
\[
\hat{\sigma}_2 = \sigma_1 + \frac{1}{2} \left( C - 2x_1^* \right)^{1/2} \left( x_1 - \mu \right)/\mu.
\] (A.15)

For \( R > 0.5 \), the value of \( \sigma_1 \) maximizing \( \lambda_2 \) is given in the text by Eq. (18). For \( C < \hat{\sigma}_2 \), the resident, with propagation level \( \sigma_1 \), resists invasion by all feasible \( \sigma_2 \). Substitution yields
\[
\sigma_1 > \mu \left[ 1 + \frac{C - 2x_1^* \left( C + 2x_1^* \right)^{1/2}}{C - \mu} \right].
\] (A.16)

As \( \sigma_1 \) approaches either its lower bound, \( \sigma_1 \rightarrow \mu \), or its upper bound, \( \sigma_1 \rightarrow C \), expression (A.16) cannot hold. Hence, as \( \sigma_1 \) increases, the phase boundary may be crossed twice; note the nonmonotonic phase boundary in the upper left subplot of Fig. 2.

Appendix B. Pair-correlation dynamics

Our pair approximation (PA) modifies methods described by Iwasa et al. (1998). We write a dynamics for five state variables: \( \rho_1, \rho_2, q_{2|1}, q_{1|2}, q_{1|1} \). The first two are global densities; the other three are local densities. Invoking constraints listed in the text, expression (20), we express the remaining PA variables in terms of the five state variables:
\[
\rho_0 = 1 - \rho_1 - \rho_2, \quad q_{1|0} = \frac{\rho_1}{\rho_1 - \rho_2} \left( 1 - q_{1|1} - q_{1|2} - q_{2|1} - q_{2|2} \right), \quad q_{2|0} = \left( \frac{\rho_2}{\rho_1 - \rho_2} \right) \left( 1 - q_{2|2} - q_{1|2} \right), \quad q_{0|1} = 1 - q_{1|1} - q_{1|2} - q_{2|1}, \quad q_{0|2} = 1 - q_{2|2} - q_{1|2}.
\] (B.1)

We omit \( q_{0|0} \), since it does not appear in the analysis.

We rewrite the dynamics of the resident’s global density, Eq. (21), in terms of our five state variables. Doing the same yields the invader’s global density’s yields
\[
\rho_1 = \beta (1 - \rho_1 - \rho_2) + \rho_1 \left[ x_1 \left( 1 - q_{1|1} - q_{1|2} - q_{2|1} - q_{2|2} \right) \right], \quad \rho_2 = \beta (1 - \rho_1 - \rho_2) + \rho_2 \left[ x_2 \left( 1 - q_{2|2} - q_{1|2} \right) - \mu - \theta q_{1|1} q_{1|2} \right].
\] (B.2)

The dynamics of the three conditional densities first requires the dynamics of corresponding doublets. By definition, \( q_{1|1} = \rho_0 / \rho_1 \), where \( \rho_0 \) is the unordered doublet density. We differentiate the conditional density, obtaining
\[
\bar{q}_{1|1} = \frac{1}{\rho_1} \frac{\partial q_{1|1}}{\partial \rho_1} = \frac{\rho_0}{\rho_1}, \quad q_{1|1} = \frac{\partial q_{1|1}}{\partial \rho_1} = \frac{\rho_0}{\rho_1}
\] (B.4)

and then separate the unordered doublet’s dynamics. Applying the recipe to \( q_{2|2} \) yields
\[
\bar{q}_{2|2} = 2 \rho_{2|2} \bar{q}_{2|2} \left[ \frac{1}{\gamma} + \frac{\delta - 1}{\delta} q_{2|2} \right] - 2 \rho_{2|2} \left[ \mu + \frac{\delta - 1}{\delta} \theta q_{1|2} \right].
\] (B.5)

The first two terms represent generation of new invader pairs through introduction and birth, and the third term represents the loss of invader pairs due to background mortality and interference competition. This equation introduces triplets \( q_{2|0} \) and \( q_{1|2} \) into the dynamics. As mentioned in the text, ordinary PA assumes that \( q_{2|2} = 0 \) and \( q_{1|2} = 0 \). The resulting closure of the equations allows the analysis without including triplets, or any higher order spatial correlations.

Using the closure assumption and converting terms with Eq. (B.1), Eq. (B.3) becomes
\[
\bar{q}_{2|2} = 2 \rho_{2|2} \left( 1 - q_{2|2} - q_{1|2} \right) \left[ \beta + \frac{\beta}{\mu} \left( \frac{1}{\gamma} + \frac{\delta - 1}{\delta} \left( \frac{1}{\gamma} - q_{2|2} - q_{1|2} \right) \right) \right] - 2 \rho_2 q_{2|2} \left[ \mu + \frac{\delta - 1}{\delta} \theta q_{1|2} \right].
\] (B.6)

Substituting Eqs. (B.2) and (B.3) into Eq. (B.4) yields
\[
\bar{q}_{1|1} = 2 \left( 1 - q_{1|1} - q_{1|2} - q_{2|1} - q_{2|2} \right) \left[ \beta + \frac{\beta}{\mu} \left( \frac{1}{\gamma} + \frac{\delta - 1}{\delta} \left( \frac{1}{\gamma} - q_{2|2} - q_{1|2} \right) \right) \right] - \rho_1 \left[ 1 - q_{1|1} - q_{1|2} - q_{2|1} - q_{2|2} \right] \left[ \mu + \frac{\delta - 1}{\delta} \theta q_{1|2} \right].
\] (B.7)

Similarly,
\[
\bar{q}_{1|1} = \left[ \frac{\beta}{\mu} \left( \frac{1}{\gamma} + \frac{\delta - 1}{\delta} \left( \frac{1}{\gamma} - q_{2|2} - q_{1|2} \right) \right) \right] - \rho_1 \left[ 1 - q_{1|1} - q_{1|2} - q_{2|1} - q_{2|2} \right] \left[ \mu + \frac{\delta - 1}{\delta} \theta q_{1|2} \right].
\] (B.8)

\( \bar{q}_{1|2} \) is slightly more complicated. There are two ways to make a (1, 2) pair, and each member of a (1, 2) pair interferes with the other. Proceeding:
\[
\bar{q}_{1|2} = 2 \rho_2 (1 - q_{2|2} - q_{1|2}) \left[ \beta + \frac{\beta}{\mu} \left( \frac{1}{\gamma} + \frac{\delta - 1}{\delta} \left( \frac{1}{\gamma} - q_{2|2} - q_{1|2} \right) \right) \right] - \rho_2 \left[ 1 - q_{1|1} - q_{1|2} - q_{2|1} - q_{2|2} \right] \left[ \mu + \frac{\delta - 1}{\delta} \theta q_{1|2} \right].
\] (B.9)

Then, following substitution:
\[
\bar{q}_{1|2} = \left[ \frac{\beta}{\mu} \left( \frac{1}{\gamma} + \frac{\delta - 1}{\delta} \left( \frac{1}{\gamma} - q_{2|2} - q_{1|2} \right) \right) \right] - \rho_1 \left[ 1 - q_{1|1} - q_{1|2} - q_{2|1} - q_{2|2} \right] \left[ \mu + \frac{\delta - 1}{\delta} \theta q_{1|2} \right].
\] (B.10)

Eqs. (B.2), (B.3), (B.7), (B.8), and (B.10) constitute the pair-approximation dynamics.

To analyze invasion, we introduce species 2 at near-zero density. Invasion succeeds or fails before the next introduction
Next, we address the density of open sites when the invader is rare. Given invader rarity, it has no effect on either the resident's equilibrium global density ($\rho^*$) or the equilibrium frequency of paired residents ($q^*$). Setting $\rho_2 = 0$ in Eq. (B.11) and (B.14) yields

$$\rho^* = \frac{\delta - \frac{1}{2} \mu}{\frac{1}{2} \gamma},$$

and

$$q^* = \frac{1}{\frac{1}{2} \gamma}.$$  

We use these results to find the other conditional probabilities. Let $x = q_{22}$, $y = q_{12}$, and $w = q_{02} = 1 - x - y$. Then

$$\dot{x} = \frac{2xw}{\delta} + xy\theta_1 \left[ 1 - \frac{2(\delta - 1)}{\delta} \right] - 2xyw - \mu x,$$

$$\dot{y} = \left( 1 - \frac{\mu}{\delta} \right) (x + 2y)w + y \left[ 2yw - (\delta - 1) \right].$$

Solving for the equilibria, we have

$$x^* = \frac{2xw}{\delta(2w + \mu + 2w\theta_1(\delta - 2))},$$

$$y^* = \frac{w(x + 2y)}{2(\mu + 2w\delta) + \theta_1 - \theta_2 - \theta_2 O(\delta - 2)}.$$  

Given the independence of $x^*$ and $y^*$, we evaluated $(\rho_2^*)_{y=0} > 0$ numerically. Using Eq. (B.12), we obtain the invasion criterion, Eq. (22) in the text. The importance of deriving $x^*$ and $y^*$ lies in demonstrating that at the neighborhood scale, both species' level of interference ($\theta_1$ and $\theta_2$) affects the likelihood that species 2 can invade the resident species 1.


