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REVIEW

Invasion theory and biological control

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Abstract

Recent advances in the mathematical theory of invasion dynamics have much to offer to biological control. Here we synthesize several results concerning the spatiotemporal dynamics that occur when a biocontrol agent spreads into a population of an invading pest species. We outline conditions under which specialist and generalist predators can influence the density and rate of spatial spread of the pest, including the rather stringent conditions under which a specialist predator can successfully reverse a pest invasion. We next discuss the connections between long distance dispersal and invasive spread, emphasizing the different consequences of fast spreading pests and predators. Recent theory has considered the effects of population stage-structure on invasion dynamics, and we discuss how population demography affects the biological control of invading pests. Because low population densities generally characterize early stages of an invasion, we discuss the lessons invasion theory teaches concerning the detectability of invasions. Stochasticity and density-dependent dynamics are common features of many real invasions, influencing both the spatial character (e.g. patchiness) of pest invasions and the success of biocontrol agents. We conclude by outlining theoretical results delineating how stochastic effects and complex dynamics generated by density dependence can facilitate or impede biological pest control.

Keywords

Predator–prey invasions, pest load, spatial spread, invasion speed.

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INTRODUCTION

Links between invasion biology and biological control of exotic pest species constitute an area of increasing interest among ecologists (e.g. Louda *et al.* 1997; Ehler 1998; Ewel *et al.* 1999; Strong & Pemberton 2000). Recently, Louda *et al.* (1997) demonstrated geographical expansion mediated by host shifts as one of several unintended consequences that followed from the establishment of an herbivorous beetle introduced for biological control of exotic thistles. Moody & Mack (1988) and Hajek *et al.* (1996) discussed the importance of targeting control efforts at the leading edge of invading populations, especially at nascent foci (recently colonized sites in advance of the main body of the invasion, whose spread can speed overall invasion progress). Parker (2000) discussed the difficulties impeding biological control of *Cytisus scoparius*, an invasive weed that lacks life stages particularly sensitive to biological control efforts. Searching for management strategies that would improve the establishment, spatial spread and suppressive effects of biological control agents, Shea & Possingham (2000) suggested several rules of thumb to guide biocontrol releases. Among these rules of thumb are that a few large releases of control agents constitute the optimal strategy against a pest when few sites already feature established populations of the control agent, with many small releases becoming optimal as the probability of establishment of control releases increases. Mixed strategies afford opportunities for learning how innoculum size influences colonization success of the control agent (Shea & Possingham 2000).

In an overview, Ehler (1998) lamented the lack of predictive guidelines that ecological theory has offered to practitioners of classical biological control and outlined several areas in which increased understanding of invasion

processes are sorely needed. Ehler also distinguished between the establishment and spatial spread of exotic pests and 'planned introductions', arguing that species falling in the latter class, which would encompass most releases of biological control agents, make better model systems in which to study the dynamics of the invasion process.

We wholeheartedly agree with Ehler's (1998) call for increased study of the processes underlying biological invasions and better links to classical biological control. However, we also feel that an increasingly general theory of invasion dynamics has much to contribute toward our understanding of both planned introductions and invasions by exotic pests. Through this review, we hope to strengthen the dialogue between theoretical and practical perspectives on biological control.

Citing particular case studies of invasions by the Mediterranean fruit fly (*Ceritits capitata*) and the spotted alfalfa aphid (*Therioaphis maculata*) in California, Ehler (1998) identified several reasons why such exotic pests might make poor model systems from which to develop theories of invasion biology. Among these were a mismatch in the distribution of adult and juvenile forms of a species, difficulty in detecting invaders at low densities, and human-mediated movement of invaders. In contrast, we feel that some of these same areas are ones in which continued development and application of quantitative theories of invasion dynamics may prove especially useful to practitioners of biological control. Our primary intention here is to synthesize existing results from invasion theory, together with several new theoretical results, into a form more accessible to a broad range of ecologists. Accordingly, we review several areas of contact between invasion theory and biological control. Among these are (1) the importance of the relative spatial spread rates of pest and control agent, (2) the critical role of long-distance dispersal, (3) structured population dynamics, (4) the detectability of invading species, and (5) stochasticity (e.g. spatial patchiness) and complex dynamics mediated by Allee effects and other forms of density dependence.

CONTACT ZONES BETWEEN INVASION THEORY AND BIOCONTROL

Rates of spatial spread

Efforts to quantify and understand what influences rates of spatial spread constitute a key research area for invasion theory (e.g. Skellam 1951; Okubo 1980; Andow *et al.* 1990; Kot *et al.* 1996; Neubert & Caswell 2000). In contrast, efforts to identify suitable biological control agents have historically placed priority on stable, effective suppression of the pest, with relatively less attention given to factors that influence successful spatial spread of control agents (Huffaker 1976; Murdoch *et al.* 1985; Kareiva 1990; Grevstad & Herzig 1997). Several authors (e.g. Simberloff & Stiling 1996a,b; Strong & Pemberton 2000) discuss how the dispersal ability of potential biocontrol agents constitutes a little-appreciated source of risk associated with their intentional release into new habitats.

Recently, Hastings (2000) has shown how parasitoid spread (through an assumed host population) can be modelled with single species reaction–diffusion (continuous time) or integrodifference (discrete time) models. In such cases, the rate of spread can be related to model inputs, such as the intrinsic growth rate of parasitoids, or their dispersal distances, as described by a dispersal kernel (see Kot *et al.* 1996). In this paper, we explicitly include an additional trophic level, namely that of the host species. It is the nonlinear spatial interaction between host and enemy that determines whether the enemy is effective in controlling the host species. We outline here why spread rates matter in the context of biological control, emphasizing that they depend on both the growth rates and dispersal abilities of the pest and control agent.

A simple model for spatial spread is the reaction-diffusion equation

$$\frac{\partial u}{\partial t} = D_u \frac{\partial^2 u}{\partial x^2} + uf(u), \qquad (1)$$

where $u(x, t)$ is population density of the pest species at location x (in one-dimensional space) and time t, $f(u)$ is the per capita growth rate, and D_u is the pest's diffusion coefficient. The first term in the right hand side of equation 1 governs spatial spread of the species while the second term determines local population growth. If the maximum per capita growth rate occurs at the lowest possible pest density (e.g. in the absence of Allee effects), the asymptotic rate of spatial advance of the spreading population is

$$c_u = 2\sqrt{D_u f(0)} \qquad (2)$$

(Kolmogorov *et al.* 1937; Aronson & Weinberger 1975). The mathematical form of this wave speed indicates that, for this class of models, the asymptotic rate of spread of the invading population is a constant and that the distance covered increases linearly with time. The result can be extended to two spatial dimensions by showing that the square root of area occupied by an invading species also increases linearly with time (Skellam 1951).

Likewise, it is also possible to calculate the wave speed for a biological control agent (or other natural enemy, hereafter 'predator') moving into the pest population when the pests are fixed at their normalized carrying capacity, $u = 1$. We use $v(x, t)$ to denote the population density of the predator species. Assuming that the pest is fixed at its carrying capacity, the predator dynamics are given by

$$\frac{\partial v}{\partial t} = D_v \frac{\partial^2 v}{\partial x^2} + vh(1, v) \tag{3}$$

where $h(u, v)$ denotes the per capita growth rate of the predator. It is assumed that the maximum per capita growth rate for predators occurs at the lowest possible predator density where density-dependent population regulation is at a minimum. As in equation 2 we have

$$c_v = 2\sqrt{D_v h(1, 0)} \tag{4}$$

for the invasion speed of the predator, yielding a form quite similar to the spread rate of the pest itself (equation 2).

When the pest dynamics are included, with the pest density close to its carrying capacity, linearization of equation 3 at the leading edge of predator invasion ($v \approx 0$) yields

$$\frac{\partial v}{\partial t} = D_v \frac{\partial^2 v}{\partial x^2} + vh(1, 0). \tag{5}$$

The solution to this linear equation yields a population of predators spreading (into the pest at carrying capacity) at a speed that is asymptotically given by equation 4. Numerical simulations agree with this linearized estimate of the speed (Fig. 1a–f).

It is informative to compare c_u and c_v when considering the efficacy of a biological control introduction. If $c_u > c_v$ then the pest will 'outrun' the predator. In contrast, if $c_v > c_u$ then the predator will eventually 'catch up' to the pest, even if the pest has a head start. Lines describing spatial extent of the pest and predator invasions as functions of time clarify the consequences of these alternative outcomes (Fig. 2).

Given a predatory species that is fast enough to catch up with a pest, determining when the predator would be expected to catch up to the pest should be of considerable practical interest. For example, the catch-up time (t_S, identified by the intersection of the pest and predator lines in Fig. 2a) delineates a triangle in plots of spatial extent vs. time. The catch-up time is then

$$t_s = \frac{c_v t_I}{c_v - c_u} \tag{6}$$

where t_I is the time of predator introduction.

The size of the catch-up triangle (measured in units of distance × time) is a measure of the spatiotemporal scope of maximal pest densities. The total scope of pest damage would include this triangle, plus another polygon that sums damage after the arrival of biocontrol agents when pests are reduced to some lower density, $u_S < 1$. Mathematically, a general representation of total pest load is

$$\mathbf{L(t)} = \int_{\mathbf{0}}^{\mathbf{t}} \int_{\mathbf{0}}^{\mathbf{c_u \tau}} \mathbf{u(x, \tau) dx d\tau}. \tag{7}$$

In the special case where the enemy and pest have constant speeds as in Fig. 2a, equation 7 simplifies to

$$2L(t) = \begin{cases} c_u t^2 & \text{if } t \le t_I \\ c_u t^2 - c_v (t - t_I)^2 (1 - u_s) & \text{if } t_I \le t \le t_s \\ c_u t_s^2 - c_v (t_s - t_I)^2 (1 - u_s) + c_u t^2 - t_s^2 u_s & \text{if } t_s \le t. \end{cases} \tag{8}$$

In any practical setting, quantifying the relative speeds of pests and predators and understanding the catch-up times they predict would also help identify those cases in which invaders spreading fast enough or with enough head start could reach spatial limits imposed by environmental constraints (e.g. the extent of an agricultural region in which a particular crop is grown) long before any biological control agent could overtake them. However, even in such discouraging cases, fast spread of biological control agents would be a desirable trait because it would reduce the time invaded habitats are exposed to the exotic pests.

Releases of a biocontrol agent at multiple sites, often undertaken with the goal of increasing the likelihood of a successful introduction (see below), are also important from the perspective of spread rates. In the scenarios discussed above, having multiple successful release sites for the biocontrol agent could lead to a pronounced increase in the total rate of invasion. This is because, initially at least, the biocontrol release sites would behave like individual invasions, with each release reducing pest densities within its own local, but expanding, area. Consequently, the effect of multiple release sites could be quite strong initially when the total area invaded would be a scalar multiple of the expansion area of a single release. However, this benefit would eventually wear off as invasion foci of the control agent coalesced. In this sense, multiple releases of

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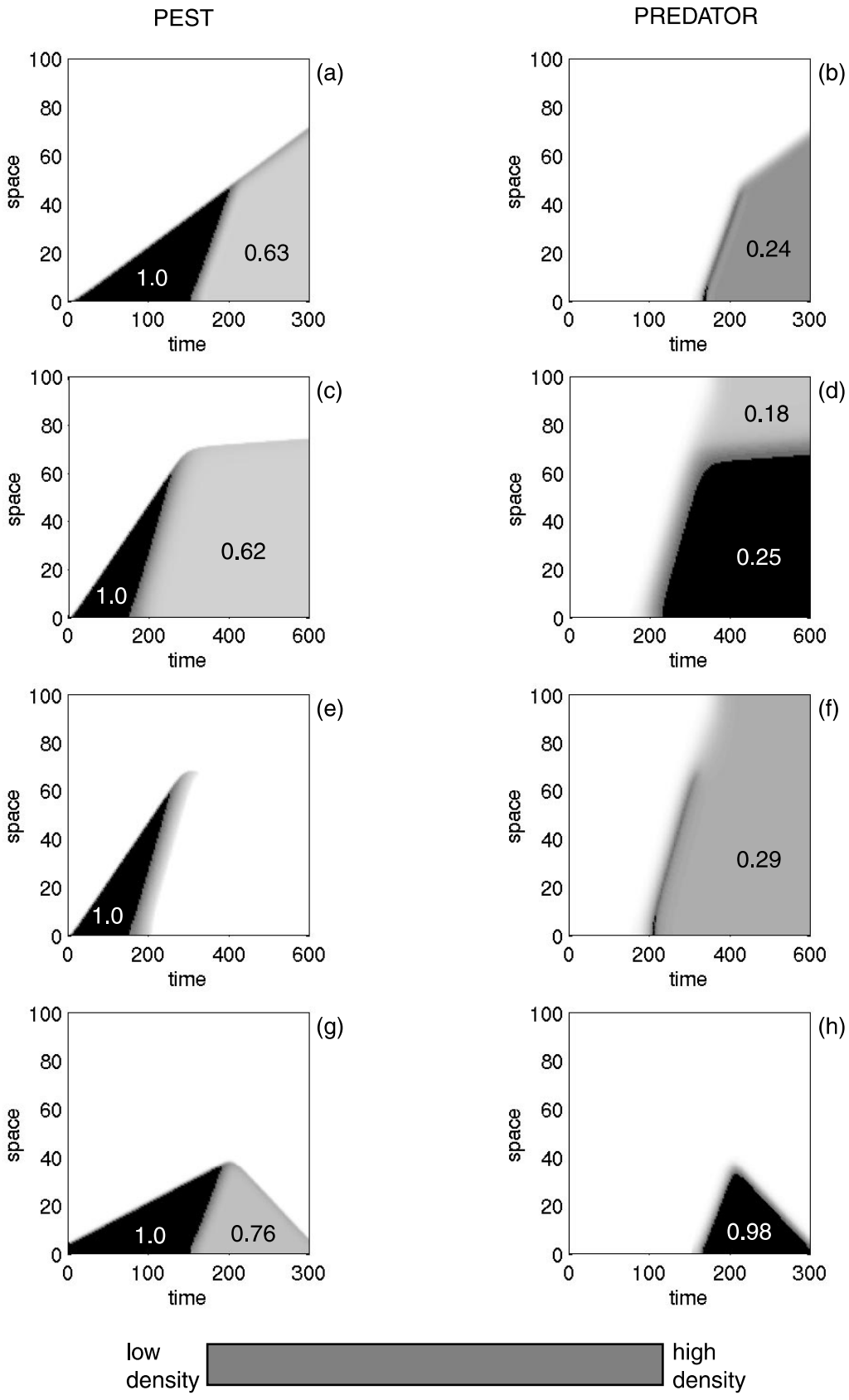
Figure 1 Numerical solutions of partial differential equation representations of biological control agents spreading into populations of invading pest species. Panels (a, b): logistic growth in the pest with specialist predator: the predator catches up to the pest, which continues expanding at a reduced density. Panels (c, d): logistic growth in the pest but a weakly generalized predator: after catching up, the predator slows the pest's rate of advance. Panels (e, f): logistic growth in the pest with a more strongly generalized predator: after catching up, the predator eliminates the pest but continues its own advance. Panels (g, h): strong Allee effect in the pest with a specialist predator: the predator catches up to the pest and then causes its population to contract spatially. We used the equation $\frac{\partial u}{\partial t} = \varepsilon D_v \frac{\partial^2 u}{\partial x^2} + f(u, v)$ for the pest and $\frac{\partial v}{\partial t} = D_v \frac{\partial^2 v}{\partial x^2} + h(u, v)$ for the predator. For (a), (c) and (e), $f = u(1 - u) - \frac{1.1uv}{.1+u}$, whereas for (g), $f = 13.2u(1 - u)(u - 0.45) - uv$. For (b), $h = \frac{5.5uv}{.1+u} - 4.75v$. For (d), $h = \frac{0.033uv}{.1+u} + 0.0715v(1 - \frac{v}{0.179})$. For (f), $h = \frac{0.033uv}{.1+u} + 0.0715v(1 - \frac{v}{0.286})$. For (h), $h = v(u - 0.76)$. In (a), (c) and (e), $\varepsilon = 0.01$ whereas in (g), $\varepsilon = 0.5$. In all cases, $D_v = 1$, the domain size was 100 and predators were introduced at $t_I = 150$.



biocontrol agents help compensate for the problems associated with long lag times between the beginning of the pest's invasion and the initial release of biocontrol agents. Achieving short lag times has historically been difficult (Ehler 1998; Ewel *et al.* 1999) because invasions are often hard to identify in their early stages and because of the inherent laboriousness of isolating and evaluating the specificity of appropriate control agents.

What happens after catch-up?

Clearly, the biocontrol agent will have some impact on the pest species when they interact, although historical evidence suggests that, on average, biocontrol agents are unlikely to completely eradicate target pests (Murdoch *et al.* 1985; Murdoch & Briggs 1996; Ewel *et al.* 1999). The detailed dynamics of spatially explicit predator–prey systems are notoriously complex and include limit cycles, cyclical travelling waves and spatiotemporal chaos (Hassell *et al.* 1991; Neubert *et al.* 2000; Sherratt 2001). However, the question of impact becomes simpler when couched in the context of spatial spread: once the biocontrol agent has caught up with the pest species, can it slow or reverse the spread of the pest species? In the context of Fig. 2, the question is: what will be the slope of the shared predator-pest line after the predator and pest lines intersect? Will it be equal to the earlier pest slope (indicating an unchanging pest spread rate), less than the earlier pest slope (indicating a reduction in pest spread rate), or even negative (indicating a reversal of pest spread)? Owen & Lewis (2001) investigated these questions in a general context for predator–prey systems.

Under the assumption that the system has a stable coexistence equilibrium (i.e. does not cycle endlessly when predator and prey are both present), Owen & Lewis (2001) used models to show that whether the population spreads at undiminished speed, slows or reverses depends crucially upon properties of the pest population dynamics that can be tested in the absence of predation. If the predator is a specialist (consumes no or few other species) then pest populations with a 'weak' Allee effect (reduced, but still non-negative, per capita growth rate at low densities) can have their spread rate slowed by predation while only those with a 'strong' Allee effect (negative per capita growth rate at low densities) can have their spread reversed by predation (Fig. 1g, h). By way of contrast, those with no Allee effect can neither be slowed nor reversed, even in the presence of strong predation (Fig. 1a, b). Thus, with a specialist predator, it is the dynamics of the pest, more than the biocontrol agent that determine the level to which spread can be reduced once the biocontrol agent catches up to the pest. Despite the stringency of this condition for reversal of the pest invasion, we note that, in practice, it may be possible to induce an Allee effect in the pest species through the use of sterile insect releases (Lewis & van den Driessche 1993) and related techniques. Thus, combining methods of biological control within a strategy of integrated pest management may prove especially useful when attempting to eradicate a pest from a landscape.

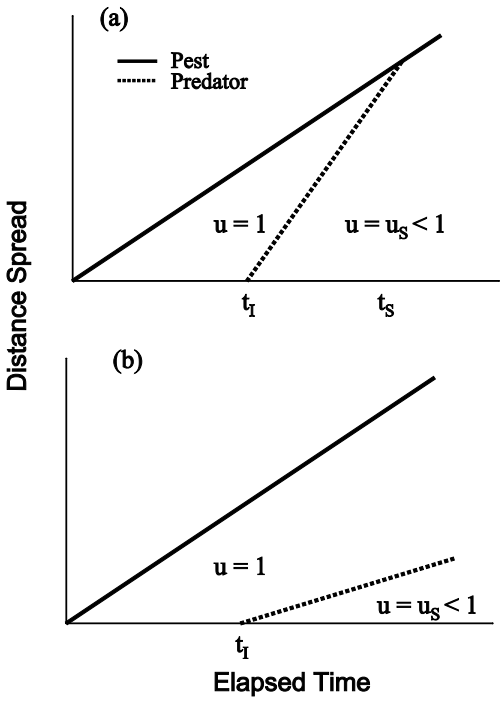


Figure 2 Heuristic plots showing spatial extent of invasions of a pest and its predator (biocontrol agent) as functions of time for dynamics governed by diffusive spread. In (a), the predator's spread rate exceeds the pest and the predator invasion eventually catches up, despite the pest's head start. The area denoted $u = 1$ indicates the spatiotemporal scope of maximal pest density. In (b), the predator's spread rate is less than the pest's and the predator will not catch up to the pest before the pest reaches the limits of available habitat.

In the case of a predator with a generalized diet, spread of the pest population can be slowed (Fig. 1c, d) or the pest population can be eradicated (Fig. 1e, f), regardless of the pest dynamics, providing the predator can persist at a sufficiently high density in the absence of the pest. This highlights one advantage of using a generalized biocontrol agent when attempting to spatially confine the spread of a pest species. The intuitive idea is that by exploiting other prey species, a generalist biocontrol agent can persist in front of the spreading pest population, driving down local growth rates of the pest at the leading edge of the invasion process. On the other hand, a specialist biocontrol agent cannot persist at high densities at the leading edge of the spreading pest population (where pest densities are low) and thus cannot slow or reverse the pest spread unless the pest species is already susceptible to reduced growth rates at low

density. Of course, additional practical difficulties are sometimes involved with using generalist predators for biocontrol of invasive pests. Among these are that native generalist predators may not have much affinity for an introduced pest, and managers may be hesitant to introduce non-native generalists because of the potential for collateral impacts (e.g. Howarth 1991; Louda *et al.* 1997).

Long distance dispersal and human-aided spread of pests

Recent developments in the theory of ecological invasions have outlined some shortcomings of models like equation 1 that assume diffusive movement (Kot *et al.* 1996). In particular, contrasts between diffusion models and alternative models of spatial spread involving integrodifference equations highlight how important long-distance dispersal events can be to the overall rate of spread of an invading population. Such events, even when rare relative to the fraction of seeds or offspring dispersing locally, can effectively determine rates of spatial spread across a landscape. Indeed, given long distance dispersal that is sufficiently common and sufficiently extreme relative to the dispersal distances of most propagules, the wave speed of an invading population ceases to be constant (as in equations 2 and 4) and instead accelerates over time.

The potential importance of invasions driven by long distance dispersal has gained recent notoriety in discussions of postglacial recolonization of temperate forest landscapes (Clark *et al.* 1998). However, long distance dispersal is likely of even greater consequence in agricultural landscapes or situations in which human-aided transport is possible. For example, Mack (1981) demonstrated that railroad networks were critical to the spread of cheat grass in the western United States. Ehler (1998) makes similar arguments concerning the spread of spotted alfalfa aphids paralleling major trucking routes in California. Likewise, repeated, intentional introduction of mosquitofish (usually *Gambusia affinis*) for control of pest insects has led to their present distribution that is essentially world-wide, across large and disconnected landscapes through which they could not have dispersed independently (Courtenay & Meffe 1989).

Such dispersal may limit the utility of pest species as models systems in which to study some biological aspects of 'natural' invasions (Ehler 1998). Nevertheless, long-distance dispersal abilities, including human-aided dispersal, can be crucial determinants of invasion dynamics, and understanding their potential consequences is critical to successful management. In the context of biological control, the effects of long distance dispersal on spread rates and catch-up times become paramount. Clearly, a pest whose invasion speed accelerates with time will be much harder to catch up to than one exhibiting a constant rate of expansion (Fig. 2). For example, any attempt to reduce damage from and halt the spread of such species across a habitat would likely require a control agent that itself is capable of rapid spread via long distance dispersal and/or quick establishment following release at large numbers of sites dispersed throughout the invaded area. Either of these requirements could greatly restrict the choice of control agents. Likewise, long-distance dispersal abilities of pests could also be problematic in that they would likely facilitate reinvasion of 'controlled' areas. Medfly infestations in California and the metapopulation-like dynamics of *Opuntia–Cactoblastis* in Australia are cases in which (natural or human-aided) long distance recolonization likely plays a critical role (Stiling 1997).

Possible disadvantages of fast spreading biocontrol agents

As discussed above, increased consideration of spatial spread rates could prove beneficial to the practice of biological control. However, an enhanced emphasis on spread rates might also have unintended negative consequences. For example, it is not unreasonable to expect that good dispersers might be deficient in other traits, perhaps making them overall poor choices as control agents. In particular, increases in spatial spread rate might incur a trade-off with ability to locally suppress pest populations. Thus, control agents able to spread throughout a large region of pest infestation might have only a meagre impact locally, whereas species able to enforce substantial pest losses locally might be unable to provide regional control.

A literature survey seeking data on the spatial spread rates of pest species and biocontrol agents released against them identified eight cases for which data on local suppression of pests were also available (Table 1). These data revealed ratios of pest spread rate to predator spread rate spanning four orders of magnitude (Fig. 3). This range includes only two cases in which predator spread rates were vastly slower than those of their target pest species (loosestrife beetle attacking purple loosestrife and *Typhlodromus manihoti* attacking cassava green mite). The highest levels of local suppression were associated with cases in which a predator's spread rate exceeded that of its targeted pest, and we found weak evidence for a suppression–dispersal advantage trade-off. However, this data set probably harbours a strong reporting bias in that few published records would likely be available concerning biological control agents that either fail to disperse widely or fail to suppress the pest.

Another issue is that an emphasis on identifying fast spreading control agents might inadvertently lead to the selection of candidate agents whose diets are insufficiently narrow to be effective (and ecologically safe) upon release. For example, species with generalized diets might more easily deal with spatial and temporal vagaries in resource

Table 1 Pest-biological control agent pairs yielding suppression data and spatial spread rates.

Case	Pest species		Control agent		Locality	Primary references
1	Purple loosestrife	*Lythrum salicaria*	Loosestrife beetle	*Galerucella* spp.	USA	McAvoy *et al.* (1997); Smith *et al.* (1999); Katovich *et al.* (1999)
2	European rabbits	*Oryctolagus cuniculus*	Myxomatosis	Myxoma virus	Australia	Marshall & Douglas (1961)
3	European rabbits	*Oryctolagus cuniculus*	Rabbit hemorrhagic disease virus	Calicivirus	Australia	Kovaliski (1998); Mutze *et al.* (1998)
4	Casava green mite	*Mononychellus tanajoa*	Predatory mite	*Typhlodromus manihoti*	Central Africa	Yaninek (1988)
5	Casava green mite	*Mononychellus tanajoa*	Predatory mite	*Typhlodromus aripo*	Central Africa	Yaninek (1988); Bellotti *et al.* (1999)
6	Casava mealybug	*Phenacoccus manihoti*	Wasp	*Epidinocarsis lopezi*	Central Africa	Herren *et al.* (1987); Yaninek (1988)
7	Prickly pear cactus	*Opuntia* spp.	Moth	*Cactoblastis cactorum*	Australia	Stiling (1997)
8	Scale insect	*Palaeococcus fuscipennis*	Ladybird beetles	*Novius cruentatus* + *Cryptochetum jorgepastori*	Israel	Mendel *et al.* (1998)

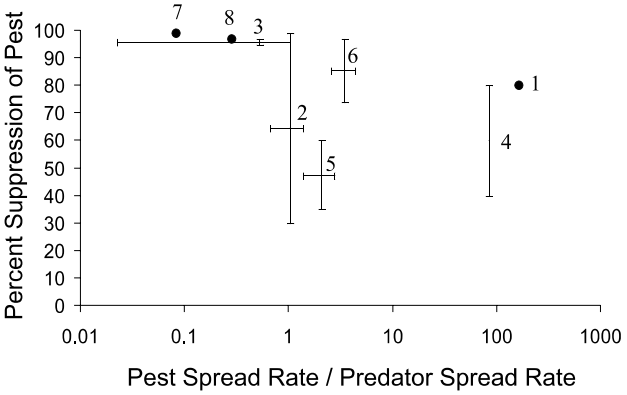


Figure 3 Relationship between pest suppression and spatial spread for eight pest–control agent pairs identified from the literature. Data are point estimates (dots) or ranges (error bars). Data for some pests or control agents were presented as area occupied over time. These were transformed to spread rates by calculating rates of increase in the square root of area occupied (Skellam 1951).

availability. Though such diets could greatly facilitate spread of control agents through a region even when their target pest populations were at low density or patchily distributed, insufficient specialization of control agents can lead to negative impacts on non-target species (e.g. Louda *et al.* 1997). Simberloff & Stiling (1996a,b) suggested cost–benefit analyses prior to introductions of biological control agents as a mechanism to force consideration of possible non-target effects. More recently, Strong & Pemberton (2000) suggested avenues for improvement of governmental oversight of biological control efforts with the joint goals of reducing ecological risk and increasing public confidence in biological control efforts.

Invasion models for structured populations

The importance of population structure for demography is well known (e.g. Caswell 2001). Individuals differ in their vital rates and responses to the environment, and many of those differences are determined by age, size or developmental stage. In studies of invasion, we must also recognize that individuals differ in their dispersal characteristics, and that these dispersal differences are also largely determined by age, size or stage. Recently, promising methods have been developed to deal with population stage structure in mathematical models of invasion (Van den Bosch *et al.* 1990; Diekmann *et al.* 1998; Neubert & Caswell 2000). Ignoring population stage structure typically produces an overestimate of invasion speed. But a more accurate prediction of invasion speed is not the most compelling reason for including stage structure in invasion models. Rather, by including stage structure, invasion speed – and, by our arguments above,

levels of pest suppression – can be connected to processes occurring within the life cycle of the individual. The sensitivity of invasion speed to changes in these processes can then be calculated (Neubert & Caswell 2000), and thereby reveal which stages of a pest species are most important to attack when attempting to slow their spread.

Detectability of invasions

Another critical issue relevant to both biological control and invasion biology in general concerns the difficulties of identifying incipient invasions and characterizing the spatial progress of a spreading population. These difficulties are inherent to efforts to detect a species present at low densities. Although invasion theory would be of limited use in the development of better detection protocols, it can provide some useful guidance concerning the nature of spreading populations. For example, a major result in this area of invasion theory is that it is not necessary to detect the furthest dispersed individuals to gauge the spatial extent of an invading population. Instead, data on changes in the spatial distribution of populations exceeding a set 'detection threshold' are themselves informative. In the case of a population spreading via diffusive dispersal, the wave speed for a specific detection threshold will be the same as the wave speeds for detection thresholds corresponding to higher and lower densities. Consequently, the spatial extent determined for a fixed detection threshold will be a constant fraction of the spatial extent of a lower detection threshold throughout the course of the invasion. In contrast, for populations whose invasion speeds accelerate over time, the spatial extent for a given detection threshold will not only underestimate the spatial extent for lower detection thresholds, but the accuracy of this estimate will degrade as time goes on. Importantly, regardless of the specific dispersal characteristics of an invading population, the higher the detection threshold for that species, the greater the area in advance of the zone of detection that is already colonized by the invader.

The roles of stochasticity and complex dynamics

Stochasticity (both demographic and environmental) should be expected to play a major role in invasion dynamics, both in their initiation and over the long term (Hastings 1996; Lewis 1997). For example, demographic stochasticity in association with long distance dispersal typically produces 'patchy' spread (Lewis & Pacala 2000). Pest species whose invasions exhibit significant patchiness may have pronounced advantages over control agents released against them in that localized populations may escape detection or colonization by control agents. Mismatches between dispersal capabilities of pests and control agents could exacerbate this problem. Thus, the patchiness of pest–enemy interactions (which may be valuable in the context of long-term pest control and suppression (Murdoch *et al.* 1985; Murdoch & Briggs 1996)) may greatly complicate biological control of an invading pest. Persistence of a pest species via patchy spatial distribution also underlies the profound difficulties involved in completely eliminating an invader once it has established (Ewel *et al.* 1999). Continued persistence of the prickly pear–*Cactoblastis* interaction in Australia is a prime example (Stiling 1997).

Stochasticity can also play an important role at the very beginning of invasions for both pest and predator species. For example, the number of release sites used and the number of individuals released at those sites can influence the likelihood of persistence of biocontrol agents on a regional basis (Beirne 1975). These same factors can also influence the degree of suppression achieved through biocontrol programs (Shea & Possingham 2000).

While the mathematical theory of biological invasions is still dominated by deterministic models, work on stochastic models is an area of active research. For example, Neubert *et al.* (2000) have developed methods for calculating expected invasion speeds in temporally stochastic environments; Lewis & Pacala (2000) have developed methods for models that incorporate demographic stochasticity (including individual variability in dispersal); and Shigesada & Kawasaki (1997) have studied the effects of spatially variable environments.

In addition to stochasticity, the complex dynamics generated by density dependence can affect the invasion process. For example, Allee effects can control the early stages of an invasion by setting minimum population sizes (or areas) that must be exceeded before spread is possible (Lewis & Kareiva 1993; Kot *et al.* 1996; Veit & Lewis 1996; Lewis 1997; Wang & Kot 2001; Wang *et al.* 2001; see also Shea & Possingham 2000). As in Fig. 1(g) and (h), Allee effects can also determine the subsequent control by the predators. Moller (1996) discusses the advantages that social insects may have in meeting such minimum population size criteria. Allee effects can arise in surprising ways, particularly in predator populations. In many predator–prey models, nonlinear dynamics can generate insidious Allee effects in the predator population through the formation of multiple attractors (Neubert & Kot 1992). When coupled with complex dynamics in the prey population, small changes in parameter values can unexpectedly induce Allee effects in the predator's dynamics (Neubert *et al.* 2000). Complex dynamics (e.g. spatiotemporal periodicity or chaos) in the wake of an advancing predator invasion (Kot 1992; Sherratt 2001) will produce variable levels of pest suppression before control is ultimately achieved.

CONCLUSIONS

Kareiva (1996) argued that ecology has not contributed much to the practice of biological control, with most of the insights instead flowing from biological control to ecology. Theoretical studies of ecological invasions can make contributions to biological control by identifying topics that may have practical importance and suggesting ways in which to study those issues. Couched in the context of invasion dynamics, we have touched on some of these potential linkages between ecological theory and biological control here, emphasizing spatially distributed predator–prey interactions, long distance dispersal, stochasticity, and complex dynamics generated by density dependence. Continued progress toward a generalized theory of invasion dynamics will help equilibrate the flow of information between the disciplines of ecology and biological control.

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