# The impact of parasitoids and inter-patch spacing on the rate of spread of hosts

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**Abstract**

Spatially isolated outbreaks of insects should spread into surrounding, unoccupied patches barring the presence of other factors. Two potential factor hindering their spread is the presence of predators or parasitoids and large spacing between habitat patches. We examine under what conditions parasitoids can slow and or prevent the spread of outbreaks of insects. We examine two models: a single population Allee effect model and a host-parasitoid model that has the possibility of creating an Allee-like effect. The presence of an Allee effect is vital in creating significant slowing of spread– increasing habitat spacing without a concomitant increase in dispersal mortality or the presence of parasitoids that do not cause an Allee-like effect has no impact on spatial spread. However, spread cannot be stopped by inherent Allee effects alone. Spread can be stopped only in the presence of significant spacing between patches. Furthermore, only inherent Allee effects in combination with significant habitat spacing can stop spread for an indefinite period of time.

An Allee-like effect of parasitoids can only slow or stop spread for a finite period of time. We compare our results with the spread of a spatially isolated outbreaks of an herbivore outbreak of the western tussock moth (*Orgyia vetusta*). The boundary of the outbreak is located where habitat spacing increases consistent with our hypothesis.

*Key words:* Spatial spread, spatial pattern, host-parasitoid dynamics, habitat spacing, Allee effects

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## Introduction

The cause of spatial patterns of abundance of species is a fundamental question in ecology (Tilman and Kareiva 1997). Such patterns are the result of underlying patterns in ecological factors such as resources and predators and the tendency of species to move. Outbreaks of insect herbivores can be striking examples of such spatial patterns (Ludwig et al. 1979, Berryman 1987, Maron and Harrison 1997). Frequently, such outbreaks spread from an initially small area of high density. Alternatively, other outbreaks persist for periods of time without spreading. Understanding why some outbreaks spread and others do not should reveal important details in the ecology of the herbivores (Hastings 1999).

Most theoretical analyses of spread have focused on the spread of single species and

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have ignored possible strong interactions with other species (Skellam 1951, Fisher 1937, Andow et al. 1990, Kot 1992), though see (Okubo et al. 1990, Cruywagen et al. 1996, Hart and Gardner 1997). This work has emphasized that spread rates are usually dependent on two factors: the amount of long distance dispersal and the intrinsic rate of growth. An exception to this is when there is an Allee effect (Kot et al. 1996, Lewis and Kareiva 1993). In that case, many factors are important including the amount of short distance dispersal and whether habitat is discrete or not. In some cases where there is an Allee effect, the spread can be stopped if the habitat is discrete (Keitt et al. 2001).

In this paper, we develop a series of explicitly spatial models of single species populations and host-parasitoid interactions that we use to explore what role parasitoids play in altering or stopping the spread of their host. In particular, we examine the influence of parasitoids and the spacing of the host habitat and possible interactions between these factors on the spread rate of a herbivore outbreak. We then examine the applicability of the general results to the well studied system of spatial pattern of the Western Tussock Moth (Hastings et al. 1997, Maron and Harrison 1997), especially in examining a possible role for habitat spacing in preventing the spread of the moth.

## Models and Analysis

*Spread in seasonal, single species models, effect of Allee effects and habitat spacing*

We first describe a model of an single population distributed in one spatial dimension. The one dimensional model is used because it allows simple concepts of spread to be demonstrated. Later, we will examine a model of the interaction of a insect population with a parasitoid. Since we are largely concerned with herbivorous insect outbreaks, we use a discrete time model reflecting the fact that many herbivorous insect species that exhibit outbreak dynamics have non-overlapping generations. This situation is well modeled with the use of integro-difference equations (IDE's). The general form for these models is

$$H_{t+1}(x) = \lambda \int_{-\infty}^{\infty} k_H(x,y) F(H_t(y)) dy \tag{1}$$

where $H(x)$ is the density function on x of herbivore numbers, $F(H)$ is the function describing density-dependent population growth in the herbivore and $\lambda$ is its intrinsic rate of increase. $k_H(x,y)$ is the kernel describing the movement of the herbivore from location y to location x (Kot and Schaffer 1986). Movement kernels are typically probability density functions, but need not necessarily integrate to one (reflecting dispersal mortality). Throughout the paper we assume there is no dispersal mortality. Additionally, we, as is typically done for mathematical convenience and parsimony, assume the dispersal function takes the form $k_H(x,y) = k_H(|x-y|)$, reflecting the assumption that dispersal is symmetrical. The spatial spread properties

of Equation 1 are well studied (Kot et al. 1996, Kot 1992, Neubert et al. 2000). The general result is that in the absence of an Allee effect, i.e. the per capita growth rate of the population is maximized at a limit of zero, the rate of spread is only dependent on shape of $k_H$ at its tails ($|x - y| >> 0$) and the value of $\lambda$.

In the presence of an Allee effect, the dynamics of spread are qualitatively different. Kot et al. (1996) used a simple model of an Allee effect to examine these effects. We use a slight variant of this model to examine the effect of habitat spacing on spread. The model assumes that if the population is below a threshold, $c$, in one generation it is at zero in the next, while if it is greater than or equal to $c$, it moves to the carrying capacity, $K$, in the next generation.

$$\begin{aligned} F(H) &= K & H &\geq c \\ &= 0 & H &< c. \end{aligned} \tag{2}$$

This model can be rescaled using $H' = H/K$ and $c' = c/K$ so that

$$\begin{aligned} F_{t+1}(H'_t) &= 1 & H'_t &\geq c' \\ &= 0 & H'_t &< c'. \end{aligned} \tag{3}$$

Using results from Kot et al. (1996), population invasions will have negative velocity (the population range will recede) when

$$\lambda/2 > c', \tag{4}$$

and will have positive velocities (increasing population range) otherwise.

We further explore the effect of Allee effects by adding discontinuous habitat to the model. We envisage a one-dimensional series of equal-spaced patches with governing

equations:

$$H'_{t+1}(i) = \lambda \sum_{j=-\infty}^{\infty} kD_H(|i-j|)F(H'_t(j)). \tag{5}$$

In order to make comparison between continuous and discontinuous habitats, we convert a continuous kernel $k_H$ into the discrete kernel $kD_H$. If we assume that within a patch, individuals are uniformly distributed, and that $r$ is the size of the patch, then the probability of an individual moving from patch i to patch j is expressed as

$$P(i,j) = \frac{\int_{(i-j)r-\frac{r}{2}}^{(i-j)r+\frac{r}{2}} \int_{-\frac{r}{2}}^{\frac{r}{2}} k_H(x,y)dydx}{2r}. \tag{6}$$

We use a (admittedly crude) approximation of this to simplify the calculation of the dispersal kernel where we substitute the value at the center of the patch for the inside integral such that:

$$kD_H(i-j) = \int_{(i-j)r-\frac{r}{2}}^{(i-j)r+\frac{r}{2}} k_H(x)dx. \tag{7}$$

A biological interpretation of this approximation is that all dispersers leave from the center of their natal patch and move according to the continuous kernel. When they settle, they move to the nearest patch. This form of discretization of habitat, unlike others (Van Kirk and Lewis 1997,e.g.), does not explicitly model regions of space that are habitat and non-habitat. Additionally, there is an implicit assumption that there is no additional mortality as patch spacing increases. This assumption could be relaxed.

After each generation, in a given patch, the population is either 0 or $\lambda$, thus allowing for calculation of the number of patches the herbivore will spread each generation. As before, we assume that, initially, a large area has been colonized and is adjacent to a large uncolonized zone. We consider the patches in the uncolonized zone and label them sequentially starting with 1. Patch i will be colonized if the immigration reaches the threshold, c', i.e., if

$$c' \leq \lambda \sum_{j=-\infty}^{i-1} kD_H(|i-j|). \tag{8}$$

The total number of patches that are colonized per generation (or lost if the population range recedes) is simply the greatest value of i that satisfies Eq.8 and the rate of expansion or contraction is simply the product of this patch number and the inter-patch spacing, $r$.

In order to get quantitative estimates for this spread rate we require an explicit kernel function, $k_H$. We show the results of the impact of spacing on velocity in the case of the Laplacian (or double exponential) kernel,

$$k_H(x) = \frac{\sigma}{2} e^{-\sigma|x|}, \tag{9}$$

in Figure 1. Increases in spacing generally have little effect on spread (either positive or negative), except when spacing gets large enough to prevent spread (see Figure 1). Also, at any given Allee threshold, zero velocities can be attained give the following conditions (see Figure 2):

$$\frac{1}{2} e^{-\frac{\sigma r}{2}} \leq \frac{c'}{\lambda} < 1 - \frac{1}{2} e^{-\frac{\sigma r}{2}}. \tag{10}$$

*Spread in the presence of parasitoids*

In this section we follow a similar analysis to that of the previous section,but we focus on host-parasitoid interactions. We start with a model of a continuous one-dimensional general Nicholson-Bailey interaction for parasitoids and their hosts:

$$\begin{aligned} H_{t+1}(x) &= \lambda \int_0^\infty F(H_t(y))G(F(H_t(y)), P_t(y))k_H(x,y)dy \\ P_{t+1}(x) &= c \int_0^\infty F(H_t(y))(1 - G(F(H_t(y)), P_t(y)))k_P(x,y)dy \end{aligned} \tag{11}$$

here, $P_t(x)$ represents the density function of parasitoids numbers on x, $c$ is the number of parasitoids produced per parasitized host, $G(H, P)$ is the proportion of herbivores parasitized by the parasitoid and $k_P$ represents the dispersal of the parasitoid. When creating discrete time models of host-parasitoid interactions with intra-specific competition in the host, a model necessarily has an assumption about the relative timing of the two events May et al. (1981). We have made the assumption that parasitoids attack after intra-specific competition. This will be the case for a majority of pupal parasitoids, which are generally the most important in outbreaking species of herbivores(Berryman 1988, Cornell et al. 1998).

We are interested in systems that may produce s-shaped nullclines and lead to Allee effects induced by the presence of parasitoids (McCann et al. 2000). We implement this assuming the following functional forms:

$$F(H) = \frac{H_t(y)}{1 + \frac{\lambda - 1}{\lambda}\frac{H_t(y)}{K}} \tag{12}$$

where $K$ is the carrying capacity,

$$G(F(H), P) = e_{-\frac{aP}{1+bF(H)}}, \tag{13}$$

where $a$ is the parasitoid attack rate and $b$ is the strength of the functional response,

$$k_H(x) = \frac{\alpha}{2} e^{-\alpha|x|}, \tag{14}$$

$$k_P(x) = \frac{\beta}{2} e^{-\beta|x|}, \tag{15}$$

where $\alpha$ and $\beta$ are the dispersal parameters for the herbivore and the parasitoid, respectively.The numerical analysis of this model is simplified by rescaling the variable. By proper rescaling the model has only four parameters: $\gamma = Kac$ (the maximal population growth rate of the parasitoid) $\hat{b} = bK$ (the strength of the functional response) $\hat{\beta} = \frac{\beta}{\alpha}$ (the ratio of host movement to parasitoid movement) and $\lambda$. Note that in Function 13, if $\hat{b}$=0, there is a Holling Type I function response and a positive value for $\hat{b}$ reflects a Type II functional response. Depending on the values of $\hat{b}$, we can get the requisite s-shaped nullcline for H (Figure 3).

We analyze these models using numerical simulations. A requirement of such simulations is a discretization of space. In discretizing these equations, we create coupled map lattices, which are representative of isolated patches, thereby making it impossible to simulate exactly continuous habitat. We mediate this effect by doing simulations where inter-patch spacing is much less than the movement of the herbivore. For all the simulations, initial populations of both herbivores and parasitoids were set at near equilibrium levels over an area twice the size of the minimum area needed for the parasitoid to persist (see Appendix). Unless otherwise stated, sim-

ulations were carried out over a period of 300 generations. The rate of spread is determined by how far the threshold density (set to the scaled density 0.0001) of herbivores moved.

The presence of parasitoids reduces the rate of spread of an herbivore over this duration of time (Figure 4). The effect on spread speed increased with increasing $\gamma$ and decreasing $\lambda$. The pattern depending on $\hat{b}$ is more complex and it depends on $\gamma$. When $\gamma$ is small, large values of $\hat{b}$ lead to faster spread. As $\gamma$ increases, there is no longer a monotonic effect of $\hat{b}$. At intermediate values of $\hat{b}$ there is the lowest spread rate. The ability of parasitoids to stop the spread of outbreaks increases as their dispersal ability increases (Figure 5). This effect typically occurs when parasitoids move an order of magnitude greater than their host.

The slowing effect on spread can be so great as to completely stop the invasion of herbivore populations. Herbivore spread is only prevented when there is Type II functional response (i.e. $\hat{b} > 0$) and when there is a significantly large spacing between patches (Figure 6). These two requirements are nearly identical to those in the single species model, where an Allee effect and large spacing is required. The effect of spacing is slightly different in the presence of parasitoids. As spacing is increased there is continuous slowing of spread toward zero, rather than the constant (with variation) decrease in the single species model.

So far, we have emphasized the spread rates over short period of time (200 generations). If we examine the effect over a longer time period, however, the results change dramatically. In fact, over long periods of time, there is no effect of the presence of parasitoids(Figure 7). The zone of negative population growth around the outbreak

is of limited spatial extent. However, according to the model described, dispersal of the host is infinite. As a result, herbivore populations will emerge separated from the main population (Figure 8). This exemplifies how spread over long time periods is fundamentally determined by the long dispersal events and subsequent population growth.

## A Comparison of Predictions with a Real System

Applying models to a specific system allows us to examine the predictions of the model present. We look specifically at the prediction that spacing of patch in concert with a parasitoid- induced Allee effect can lead to the stopping of spread of herbivore populations. The system we investigate involves the spatially limited outbreak of *Orgyia vetusta* present in the coastal grasslands of the Bodega Marine Laboratory on the coast of California (Brodman et al. 1997, Harrison 1997, Hastings et al. 1997, Maron and Harrison 1997). McCann et al. (2000) have described the aspects of this system that tend it towards zero spread rate due to the presence of parasitoids: female *O. vetusta* are flightless and disperse on a small scale (on the order of 4-8m per generation (Harrison 1997)), while the parasitoids disperse over a much wider area (Brodman et al. 1997). Additionally, there is strong evidence of a an Allee effect caused by the presence of parasitoids emerging from the outbreak. Just beyond the edge of the outbreak, parasitism rates are much higher than within the outbreak, then decrease further away from the outbreak (Brodman et al. 1997, Maron and Harrison 1997).

Two hypotheses now exist as to why the parasitoid induced Allee effect leads to the preventing of spread of herbivore outbreaks. One previously postulated reason for the zero spread in continuous habitat has been the presence of a reflecting boundary (Hastings et al. 1997). Our work here suggests that patch spacing may be important. A simple way to assess the plausibility of this hypothesis is to compare the density of *O. vetusta* and the spacing of its habitat. In this system, we consider each individual host plant (bush lupines of either *Lupinus arboreus* or *L. chamissonis*) bush to be an independent patch, which is reasonable given that larvae generally stay on on bush throughout development. Our prediction from this work is that the spread would be halted when spacing of plants increases to a point where actual movement to adjacent bushes is greatly reduced. Figure 9 shows that, at least visually, there appears to be a concordance between these two variables. The rapid change in herbivore density occurs at an inter-patch spacing of approximately 4-6 meters.

## Discussion

The concepts of spatial spread and spatial pattern have generally been considered separately. Generally, theoretical examinations of spatial pattern have focused on diffusive instabilities, which examine the propensity for spatially homogeneous equilibrium to give way to heterogeneous patterns across the entire landscape. Our approach focuses on an individual area of high density and the role parasitism plays in slowing or stopping the spread of that area.

This approach allowed us to show that the extent of the outbreak of *O. vetusta*

*O. vetusta* may be explained by an inability to spread due to interaction between the activity of parasitoids and the spatial arrangement of their host plant. The importance of parasitism in this system has been the focus of previous work(Maron and Harrison 1997, Hastings et al. 1997, Wilson et al. 1998). However, our results emphasize that a secondary factor, in addition to the parasitoid induced Allee effect, is necessary to prevent the spread of regions of high density. Further experiments and observations of this system, and other similar systems, should examine the impact of the spacing of habitat patches of the host.

This raises the issue of what allowed for stable pattern in earlier models of this system and in other models of spatial patterning. Hastings et al. (1997) showed that the presence of a reflecting boundary was one such mechanism. The early models of spatial patterning in predator-prey systems used a different mechanism (e.g. Mimura and Murray 1978). The pattern in these models was repeated through space. Therefore, from the perspective of an individual region of high density, spread was halted due to the presence of a nearby region of high density. In the case of *Orgyia vetusta* such a region was not present. The location of the outbreak on a point of land provided boundaries on at least one side suggesting that the mechanism of Hastings et al. (1997) may be important in determining the size of the outbreak.

There is a clear similarity between the model we examine and that which is described by Keitt et al. (2001). Indeed, from their analysis of a single species model with an Allee effect, they suggested that habitat spacing may play an important role in the patterning of *O. vetusta*. Important insights are gained by examining the two species case. Clearly, it reveals that parasitoids can produce the Allee effect necessary for

spatial patterning, a prediction already tested in the *O. vetusta* system (Maron and Harrison 1997). It also predicts that spatially isolated outbreaks will have a minimum spatial extent (see Appendix) that will support a parasitoid population. Outbreaks below this size will spread unimpeded by either parasitoids or the spacing of their habitat.

An even more important difference, however, is that the parasitoid induced Allee effect is present over a spatially restricted area surrounding the outbreak. The main result of this difference is that spread inhibition is not indefinitely effective. Long distance dispersal by the herbivores will eventually lead to establishment of populations at a distance from the original outbreak. In this framework, spatial patterns are not permanent features of the environment, but rather transient phenomena. An analogy can be made from this slowing of spread to the culling of fox as a means to delay the spread of rabies (Murray et al. 1986).

Detecting transient pattern was facilitated by our use of numerical simulations. An alternative analysis would be to examine traveling wave solutions to the equation (e.g. Kot 1992). While this has not been done for two variable IDE's, traveling wave solutions have been examined for reaction-diffusion equations of predator-prey interactions (Owen and Lewis 2001, Dunbar 1986). The most relevant to our case is that of Owen and Lewis (2001) which examined the joint invasion of predator and prey. In accord with the results presented here, they found that discrete habitat was necessary to allow stable solutions. In contrast to our results, the presence of predators alone could not slow the invasion of prey species without the further slowing effect of Allee dynamics in the prey species. Owen and Lewis (2001) assume

a traveling wave solution and analyze the requirements on the speed of that solution. This approach emphasizes the spread of low densities a long distance from the area of higher density, which in a sense is an analysis of the spread over long periods of time. Our results over long periods of time agree with the traveling wave analysis. Also, Owen and Lewis (2001) predict that spread can be reversed by the predators. We have not found this under the parameter values we have tried. If this were the case, there would be an outbreak size that would be stable. This can be reasoned because there is a minimum size of the outbreak to support parasitoid populations. As the invasion front recedes, the number of parasitoids supported by the prey population declines until recession rate goes to zero. Under this situation, there would be a spatial scale of stable spatial pattern.

The model we analyze is deterministic and randomness may have an influence on our results. Stochastic models of single species spread show that spread is slower in such systems compared to deterministic ones due to the long term population growth rate being reduced in stochastic models (Neubert et al. 2000, Lewis 2000). We suspect that a similar effect could occur in a two species model. In addition, random fluctuations in dispersal and fecundity could be important in pushing populations above the parasitoid induced Allee threshold, further reducing the stability of the pattern, or alternatively, speeding the spread of the outbreak. We know of no analysis of this type of effect in either multi species or single species model.

Our approach to habitat spacing takes into consideration only one type of effect. Several additional effects may or may not exist in specific biological systems. Ovipositing females may select areas of high host density (Root 1973) further reducing den-

sities in isolated patches. Changes in the demographic rates, especially increased mortality (Yamamura and Yano 1999) have been identified among herbivores and this effect can make widely spaced patches uninvadable (Van Kirk and Lewis 1997). Parasitoids could have similarly higher mortality in widely spaced patches as foraging behavior can be risky (Heimpel et al. 1997). Also, the additional travel time between individual patches reduces the foraging efficiency of parasitoids and can have dramatic effects on population dynamics (Weisser and Hassell 1996).

Parasitoids have been a key tool in biocontrol of invading pest herbivore species (Waage and Greathead 1989). Traditionally, their role has been to suppress already established high density populations of herbivores rather than to limit their spread. Their ability to do this has been mixed, with some parasitoids becoming established without significant reduction in the density of the pest species, while others either fail to establish or establish with little effect on their target host (Waage and Greathead 1989). Our results suggest that even in the case where parasitoids do not control established populations, the parasitoids may prove a tool to slow the spread of the herbivore, as long as they disperse over a much wider area than their host. While halting and reversing invasions is the most desired result of managers, slowing invasions can provide large economic benefits, despite not preventing the eventual invasion Sharov and Liebhold (1998), Sharov et al. (1998). Also, such slowing could provide additional time to examine other methods to manage the invasion.

A frequent criticism of ecological theory is that it oversimplifies systems in its pursuit of explanation. Murdoch et al. (1997) have proposed methods of developing sets of models of varying complexity to relate to each others. Comparison of these models

to real systems allows for the understanding of the effect of complex mechanisms that are difficult to test in real systems. We see our current model as a relatively complex part of such a family of models that include host-parasitoid models and models of single species spatial spread models (Hastings 1997, Wilson et al. 1998, McCann et al. 2000, Keitt et al. 2001, Owen and Lewis 2001). By placing our current study in the context of these previous studies we have been able to isolate the role of habitat spacing in determining the spatial pattern of outbreaks of insect herbivores and provide a testable hypothesis for describing the extent of the outbreak of *O. vetusta* at Bodega Head.

## Appendix

We describe our method of determining the minimum outbreak size to support a parasitoid population. The methods for this were described by Kot and Schaffer (1986) for single species model, and are similar to those in continuous equations (Kierstead and Slobodkin 1953, Skellam 1951, Okubo 1980). We use the nondimensionalized form of the equations 12, 12 and 13 and 15 scaling so that $h = \frac{H}{K}$, $p = aP$, $\hat{x} = x\alpha$ , $\hat{y} = y\beta$ $\gamma = Kac$, $\hat{b} = bK$, $\hat{\beta} = \frac{\beta}{\alpha}$. The model is

$$\begin{aligned} h_{t+1}(\hat{x}) &= \lambda \int_0^\infty F(h_t(\hat{y}))e^{-G(F(h_t(\hat{y})),p_t(\hat{y}))}\frac{1}{2}e^{-|\hat{x}-\hat{y}|}d\hat{y} \\ p_{t+1}(\hat{x}) &= \gamma \int_0^\infty F(h_t(\hat{y}))(1-e^{-G(F(h_t(\hat{y})),p_t(\hat{y})}\frac{1}{2}\hat{\beta}e^{-\hat{\beta}|\hat{x}-\hat{y}|}d\hat{y}. \end{aligned} \tag{16}$$

where $F(h) = \frac{h}{1+\frac{\lambda-1}{\lambda}h}$ is the host density dependence function and $G(h,p) = \frac{p}{1+\hat{b}h}$ is the parasitism function.

In the absence of parasitoids the herbivore will attain a positive equilibrium when $h = \lambda$. We now assume the herbivore densities are constant, and taking the parasitoid equation separately:

$$p_{t+1}(x) = \gamma \int_0^L \lambda(1 - e^{-\frac{p_t(\hat{y})}{1+\hat{b}\lambda}})\frac{1}{2}\hat{\beta}e^{-\hat{\beta}|\hat{x}-\hat{y}|}d\hat{y}. \tag{17}$$

We examine the stability of the solution $p = 0$ over values of L. If the zero solution is stable, then parasitoids cannot persist. The value of L where the solution becomes zero represents the minimum outbreak size to allow for parasitoid colonization. Taking results from Kot and Schaffer (1986) we get

$$L^* = \frac{2}{2}\hat{\beta}\sqrt{\frac{1+\hat{b}\lambda}{\gamma\lambda}}\arctan\sqrt{\frac{\gamma\lambda}{1+\hat{b}\lambda}}. \tag{18}$$

Hence, we can predict the minimum outbreak size to allow the persistence of parasitoids.

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## 1 Figure Captions

Figure 1. Impact of habitat spacing on invasion speed of an herbivore with an Allee effect. When the Allee threshold is greater than 0.5, the population retracts. As habitat spacing increases, the number of patches invaded or lost iteratively decrease, creating the the saw-edge shape.

Figure 2. Conditions for forward, backward and no movement of an herbivore invasion as a function of Allee threshold and inter-patch spacing.

Figure 3. Nullclines for the non-spatial model of host-parasitoid interaction. The herbivore nullcline is the solid line and the parasitoid is dashed. The top figure represents a case where there is a Type 1 functional response ($\hat{b} = 0$) so there is not a hump in the nullcline and no possibility for a spatial Allee effect. Below there is a Type 2 functional response ($\hat{b} = 4$), leading to the hump-shaped nullcline so that the system is susceptible to spatial Allee effects. For these nullclines, $\gamma = 14$ and $\lambda = 10$.

Figure 4. The impact of interaction parameters on the spread of an herbivore outbreak. The spread is measured in non-parametric units of host-dispersal equivalent to the standard deviation of the host kernel, see Equation 5. The horizontal line on top of each graph represents the spread rate in absence of parasitoids. Here, the parasitoid movement is much greater than the herbivore ($\hat{\beta} = .001$).

Figure 5. Effect of relative movement ability of parasitoid on the spread of an herbivore outbreak. For these simulations, $\lambda = 6$ and $\hat{b} = 2$.

Figure 6. Impact of inter-patch spacing on the spread of an herbivore outbreak. The threshold spacing that allows for spread changes greatly with the value of $\gamma$. In both cases, $\hat{b} = 4$.

Figure 7. Change in spread rate of an herbivore invasion over time. The curves represent the point of furthest spread as a given time and the slope of the curve represents the spread. The top curve is an example with no parasitoids present, the middle, where parasitoids are present with Type I functional response, and the bottom with Type II functional response.

Figure 8. Spread of an herbivore outbreak under parasitoid exploitation. Curves depict the distribution of hosts at 20 generation intervals (curves further to the right are later in time). The top shows spread when there is large inter-patch spacing ($r = 2.4$) and the bottom when patches are close ($r = 0.8$). In both cases, there is a region at the edge of the invasion where growth is inhibited by parasitoids, but at larger distances, this inhibition is not important. For these simulations $\lambda = 10$, $\gamma = 12$ and $\hat{b} = 3$.

Figure 9. Relationship between inter-patch spacing and herbivore density in *Orgyia vetusta*. The edge of the outbreak represents an increase in the spacing of the host plant, *Lupinus arboreus*, (a) and the concurrent herbivore density (b). In both (a) and (b) the pattern has been emphasized with a locally weighted regression. In (c), we plot the local weighted fits against each other.

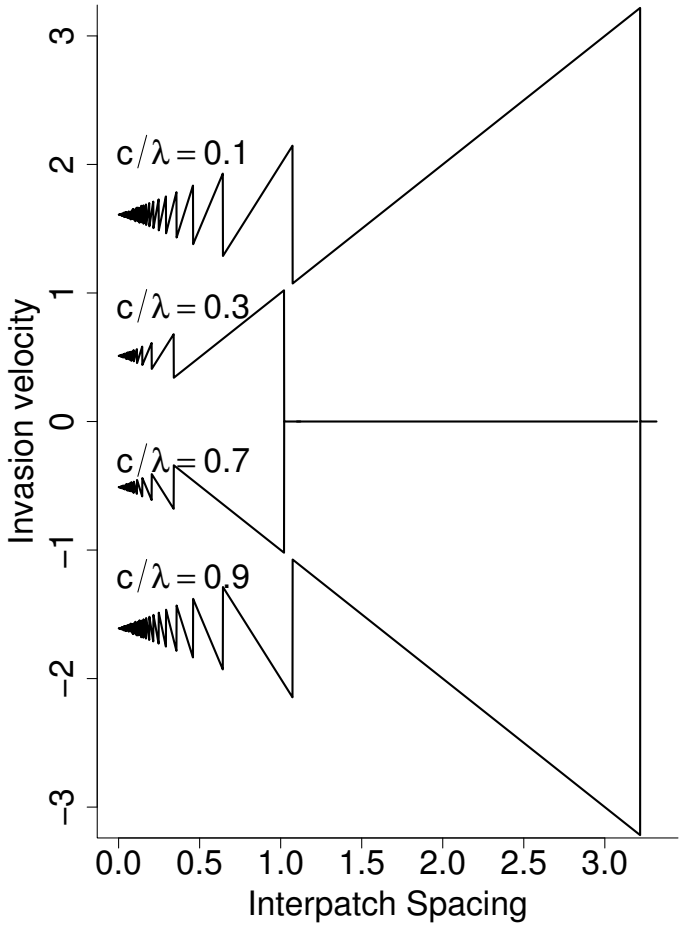
Invasion velocity
3
2
1
0
−1
−2
−3
c/λ = 0.1
c/λ = 0.3
c/λ = 0.7
c/λ = 0.9
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Interpatch Spacing

Fig. 1.

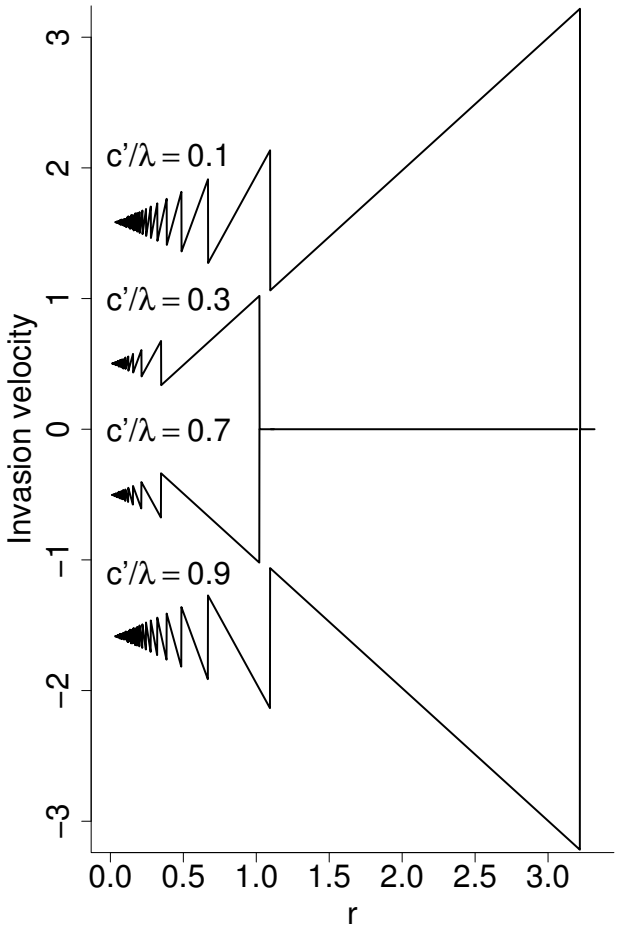
c'/λ = 0.1
c'/λ = 0.3
c'/λ = 0.7
c'/λ = 0.9
Invasion velocity
r
3
2
1
0
−1
−2
−3
0.0
0.5
1.0
1.5
2.0
2.5
3.0

Fig. 2.

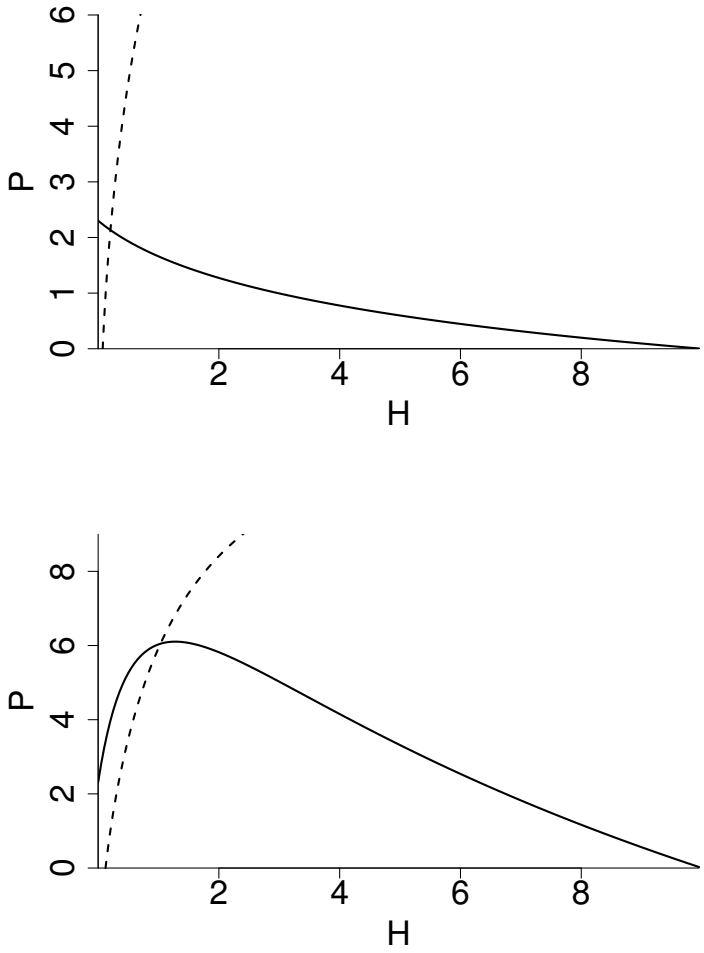


Fig. 3.

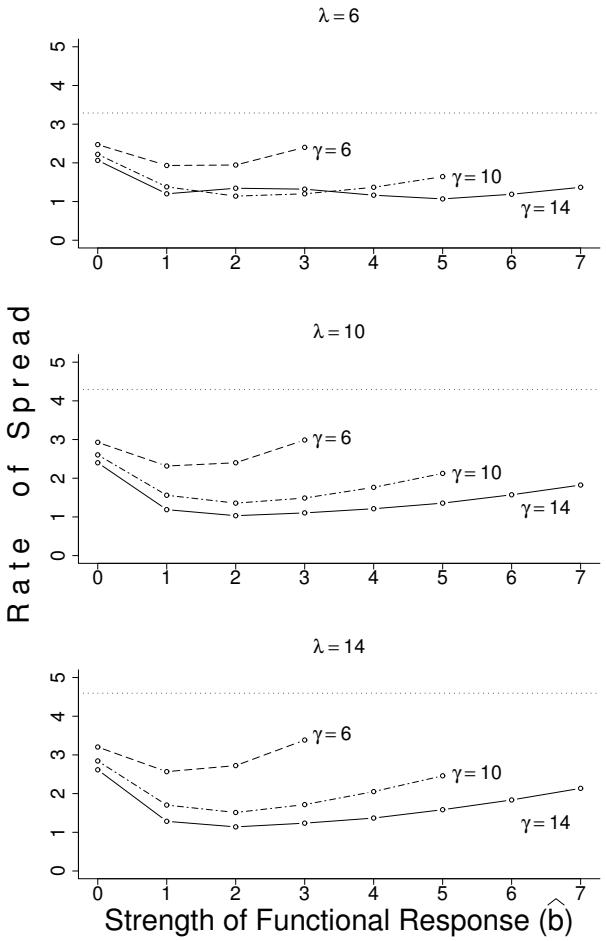
λ = 6
γ = 6
γ = 10
γ = 14
λ = 10
γ = 6
γ = 10
γ = 14
λ = 14
γ = 6
γ = 10
γ = 14
Rate of Spread
0 1 2 3 4 5
0 1 2 3 4 5 6 7
Strength of Functional Response (b̂)

Fig. 4.

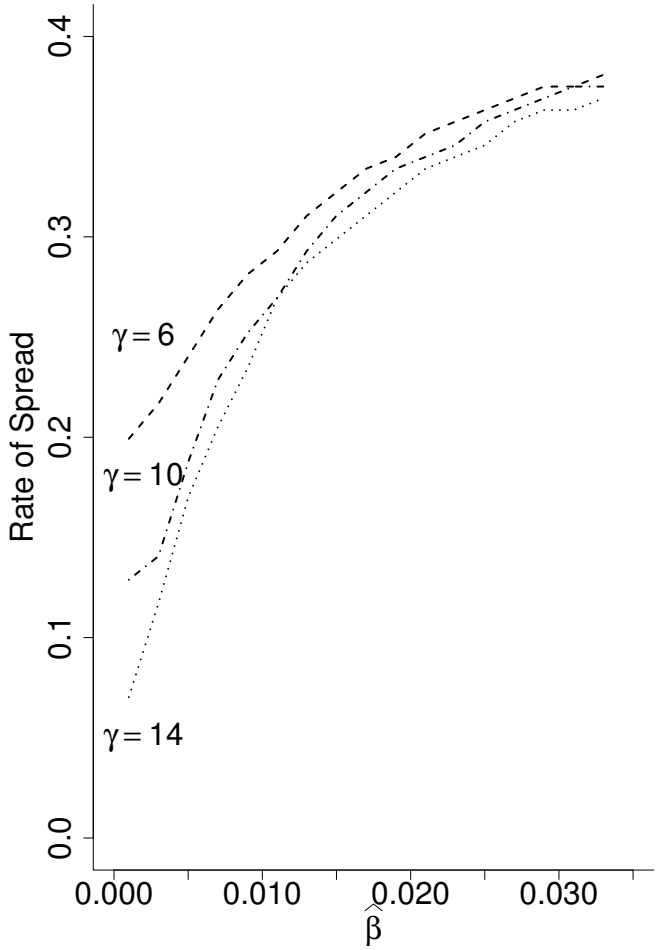


Fig. 5.

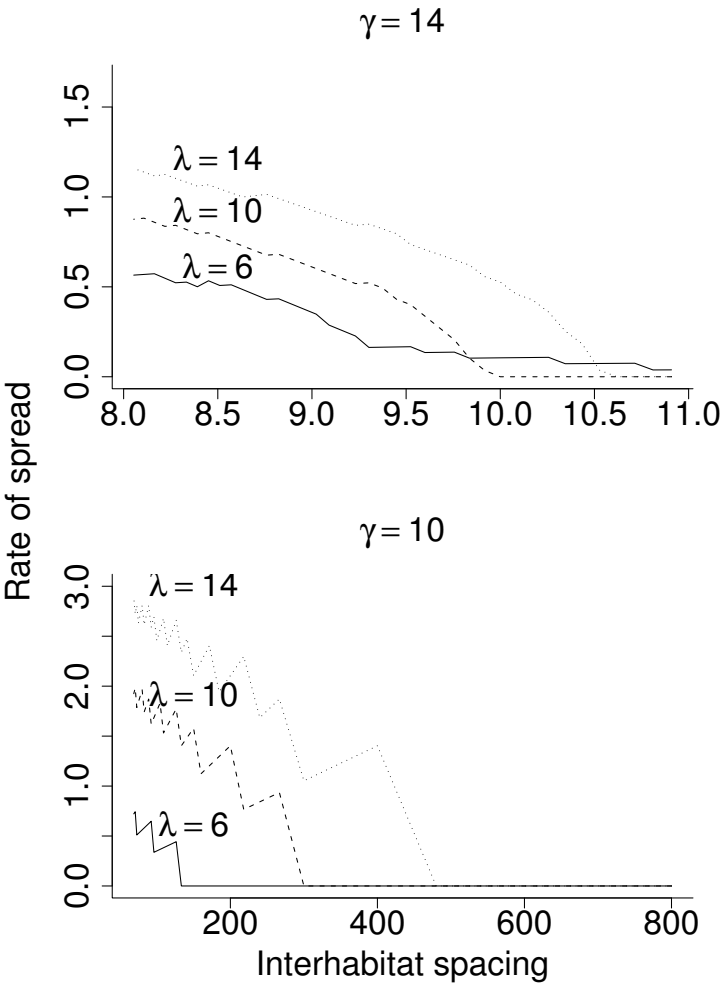
γ = 14
λ = 14
λ = 10
λ = 6
0.0
0.5
1.0
1.5
8.0
8.5
9.0
9.5
10.0
10.5
11.0
γ = 10
λ = 14
λ = 10
λ = 6
0.0
1.0
2.0
3.0
200
400
600
800
Rate of spread
Interhabitat spacing

Fig. 6.

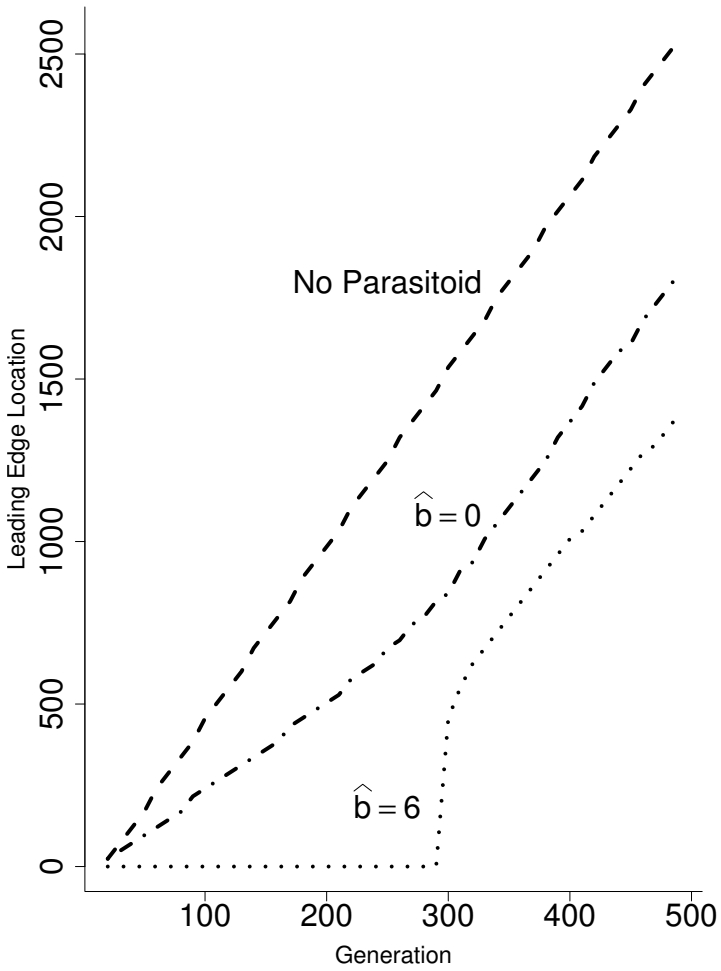
Leading Edge Location
0
500
1000
1500
2000
2500
100
200
300
400
500
Generation
No Parasitoid
$\widehat{b} = 0$
$\widehat{b} = 6$

Fig. 7.

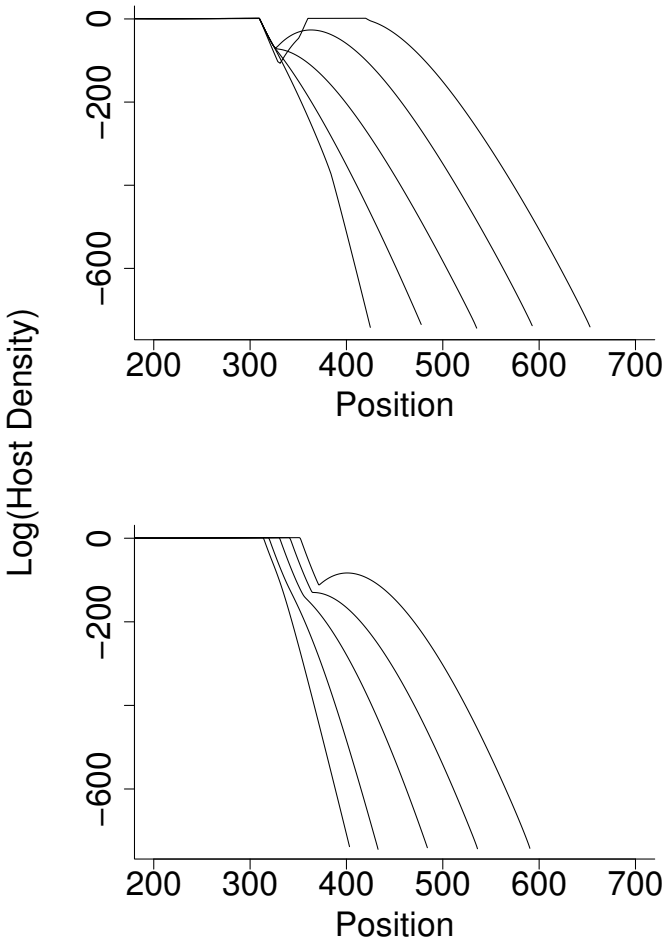
Log(Host Density)
0
−200
−600
200
300
400
500
600
700
Position
0
−200
−600
200
300
400
500
600
700
Position

Fig. 8.

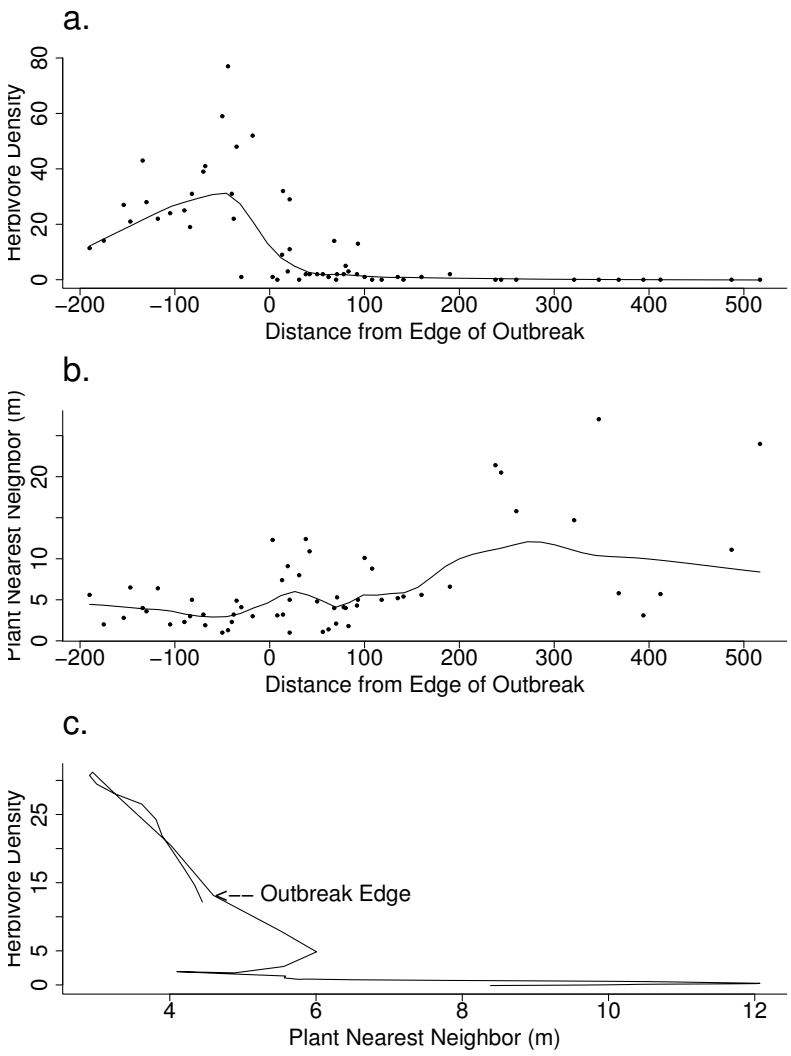
a.
Herbivore Density
0
20
40
60
80
−200
−100
0
100
200
300
400
500
Distance from Edge of Outbreak
b.
Plant Nearest Neighbor (m)
0
5
10
20
−200
−100
0
100
200
300
400
500
Distance from Edge of Outbreak
c.
Herbivore Density
0
5
15
25
Outbreak Edge
4
6
8
10
12
Plant Nearest Neighbor (m)

Fig. 9.