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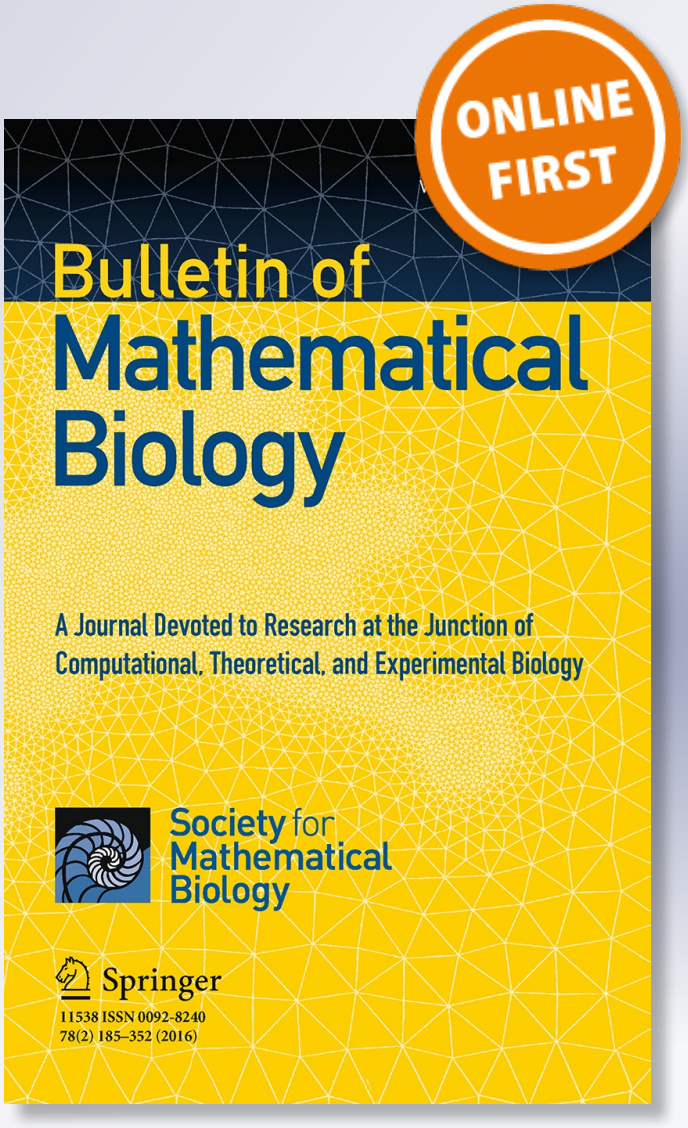

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ORIGINAL ARTICLE

Mate Finding, Sexual Spore Production, and the Spread of Fungal Plant Parasites

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Abstract Sexual reproduction and dispersal are often coupled in organisms mixing sexual and asexual reproduction, such as fungi. The aim of this study is to evaluate the impact of mate limitation on the spreading speed of fungal plant parasites. Starting from a simple model with two coupled partial differential equations, we take advantage of the fact that we are interested in the dynamics over large spatial and temporal scales to reduce the model to a single equation. We obtain a simple expression for speed of spread, accounting for both sexual and asexual reproduction. Taking Black Sigatoka disease of banana plants as a case study, the model prediction is in close agreement with the actual spreading speed (100 km per year), whereas a similar model without mate limitation predicts a wave speed one order of magnitude greater. We discuss the implications of these results to control parasites in which sexual reproduction and dispersal are intrinsically coupled.

Keywords Allee effect · Density-dependent dispersal · Facultative parthenogenesis · Pushed wave · Spore-producing pathogens

Mathematics Subject Classification 92D25 · 92D30 · 35Q92

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1 Introduction

Sexual reproduction and dispersal are often coupled in organisms combining sexual and asexual reproduction, i.e. facultative parthenogens. Quoting Williams (1975), 'where both asexual and sexual reproduction can occur simultaneously, the asexual offspring will develop immediately and near the parent, but dormant, widely dispersed propagules will be produced sexually'. In plants for instance, sexual reproduction and dispersal are intrinsically coupled as seeds are often their most efficient way to disperse (Cheptou 2012). From an evolutionary perspective, sexual reproduction and dispersal may both be selected for in spatially and temporally varying environments, so that these environments could favour a coupling between the two functions (Bonner 1958).

However, self-incompatible sexual reproduction requires finding a mate, which may be challenging at low population density. Such a positive correlation between one component of the *per capita* growth rate (mating success) and population density is termed a component Allee effect (Allee et al. 1949). Mate limitation has actually been reported as the most common mechanism leading to Allee effects (Gascoigne et al. 2009). Allee effects may give rise to a critical density below which the population goes extinct, a phenomenon termed a strong Allee effect (Taylor and Hastings 2005; e.g. Garrett and Bowden 2002; Castel et al. 2014 in fungi). Moreover, Allee effects are known to influence the spread of invading organisms both negatively (critical patch size, Lewis and Kareiva 1993; Vercken et al. 2011) and positively: Roques et al. (2012) showed that with an Allee effect, the population propagates as a pushed wave, which prevents erosion of genetic diversity in the invasion front. Pushed waves are driven by the whole invasion front rather than by the leading edge only, as opposed to pulled waves (Stokes 1976; Morel-Journel et al. 2015). Mate finding as a prerequisite to reproduction and dispersal therefore deserves particular attention.

In plant ecology, the so-called Baker law (Baker 1955) states that long-distance dispersal is more likely associated with self-compatible sexual reproduction, because mate and pollen limitation likely limit self-incompatible reproduction after long-distance dispersal. Although intuitive, this statement is not clearly supported by theory or data (Cheptou 2012; Pannell et al. 2015). In particular, Cheptou and Massol (2009) and Massol and Cheptou (2011a, b) studied the joint evolution of dispersal and self-fertilization with spatially and temporally heterogeneous pollination. They found either complete outcrossing associated with dispersal (the 'dispersal/outcrossing' syndrome) or complete/mixed selfing associated with the absence of dispersal (the 'no-dispersal/selfing' syndrome). Their results contrast with Baker's intuition and provide an explanation for the unexpectedly high frequency of plants with separate male and female individuals on islands (Cheptou 2012).

In animal ecology, Shaw and Kokko (2015) explored how mating system, strength of an Allee effect, and dispersal evolution influence invasion speed. From individual-based simulations, they found that mating system differences can dramatically alter the spread rate. In particular, they found that removing the mate finding Allee effect by introducing parthenogenesis can increase the rate of population spread by almost one order of magnitude (actually by a factor 8). Although the literature addresses density-dependent dispersal in general (Gurney and Nisbet 1975; Travis et al. 1999; Lutscher 2008), we are aware of only one additional study explicitly accounting for dispersal as

conditioned by a mating event. More specifically, Veit and Lewis (1996) focused on the spread of house finches in North America and showed that positive density-dependent dispersal associated with an Allee effect explains their initially slow invasion speed.

Fungi are major biological invaders (Desprez-Loustau et al. 2007; Fisher et al. 2012) and major plant parasites (Brown and Hovmøller 2002; Soubeyrand et al. 2009). Most fungi combine sexual and asexual spore production. Sexual and asexual spores usually differ in their shapes, sizes, and therefore dispersal abilities. For instance, *Mycosphaerella fijiensis*, the causal agent of the Black Sigatoka disease of banana plants which recently invaded all banana-growing regions, produces two types of spores: ascospores are the product of sexual reproduction, while conidia are produced through asexual reproduction. While ascospores are dispersed by wind from several hundred metres to several kilometres, conidia are dispersed by rain-splash up to a few metres only (Rieux et al. 2014).

In many species, the successful fusion of gametes can only occur between haploids carrying functionally different mating-type alleles (say $+$ and $-$), a phenomenon termed heterothallism. In haploid heterothallic fungi (including most ascomycetes), sexual spore production thus results from the interaction between two individuals with compatible mating types ($+$ and $-$) (Billiard et al. 2011). Genomic studies now increasingly support the idea that this mating system is shared by many plant and animal (including human) parasites (Ene and Bennett 2014).

How mating precisely occurs can be specific to the species considered and may be unknown even in ecologically or economically important species. In *M. fijiensis*, mating occurs through a direct contact between two adjacent lesions on the same leaf. By contrast, some fungi such as the causal agent of the poplar rust, *Melampsora larici populina*, mate remotely through gamete-like propagules (spermatia) transported by wind or by insects (Bultman et al. 1995; Pernaci et al. 2014). Some fungi even induce the plants that they infect to produce pseudo-flowers to attract insects, and sometimes provide them a nectar-like reward for outcrossing service (Roy 1994).

The way fungi spread has been the object of a number of studies, including (Frantzen and van den Bosch 2000; Aylor 2003; Powell et al. 2005; Burie et al. 2006; Cunniffe and Gilligan 2008; Mundt et al. 2009). Yet, whether mate finding significantly limits the spread of plant pathogenic fungi remains unknown, despite its potential practical consequences. For instance, the causal agent of Ash Dieback, *Hymenoscyphus fraxineus*, a lethal disease of ash trees which is currently invading Europe at the approximate speed of 75 km per year (more than 1200 km travelled within 16 years in Europe; Fig. 1), has been recently reported to be heterothallic (Gross et al. 2014). Again, this implies that mating between two compatible sexual partners must take place for spores to be dispersed. As a case study, we focus on the Black Sigatoka disease of banana plants (*M. fijiensis*), whose spreading speed is around 100 km per year (Halkett et al. 2010), and which similarly produces dispersive spores only when two compatible ($+$ and $-$) lesions meet on the same leaf.

We aim at evaluating the impact of mate limitation on the spreading speed of fungal plant parasites through a mathematical model combining sexual and asexual reproduction. First, we explore an original reaction-diffusion model accounting for mate limitation in plant pathogenic fungi and show how it can be parameterized using data available in the literature, using *M. fijiensis* as case study. Taking advantage of

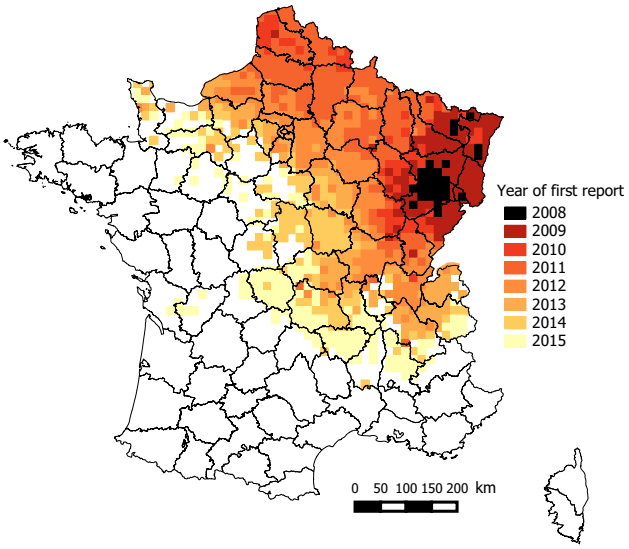

Fig. 1 (Color Figure Online) The spread of Ash Dieback (*H. fraxineus*) in France, from 2008 to 2015 (*source*: DSF, French Forest Health Survey System)

the fact that we are interested in invasion dynamics over large spatial and temporal scales we successively approximate the original model by two simpler models. The third model allows us to get an explicit expression of the spreading speed as a function of the ratio of asexual to sexual spore production. We finally compare the spreading speed obtained with mate limitation to that obtained from an analogous model without mate limitation.

2 Models

We focus on plant pathogenic fungi to make assumptions explicit and to parameterize the model, but we keep it as simple as possible for the sake of generality. A detailed description of an analogous spatially implicit model, including the choice of an appropriate mating function, can be found in (Ravigné et al, unpublished manuscript). Thus, we make a concise presentation of the spatially explicit model here.

Let $i(x,t)$ be the density of infected leaves at time $t > 0$ and location $x \in (-\infty, +\infty)$ (i.e. we adopt a unidimensional conception of space for simplicity). Let n be the total leaf density. Note that the term 'leaf' is a shorthand for the 'leaf part that a lesion occupies', so that multiple infections cannot occur in the model. Let α be the number of asexual spores (conidia) produced per infected leaf per unit time, and $0 < p < 1$ be their infectivity, i.e. the probability that an asexual spore in contact with a susceptible leaf succeeds to infect it. Similarly, let σ be the number of sexual spores (ascospores) produced per infected leaf per unit time, and $0 < q < 1$ be their infectivity. However, sexual spore production is conditioned to the local presence of a mating partner, whose probability is $(i/2)/n$, assuming a balanced mating-type ratio.

We assume that only sexual spores diffuse; asexual spores do not diffuse. Let then $u(x,t)$ be the density of sexual spores (ascospores), with diffusion coefficient κ, and rate of deposit upon leaves μ. Letting t and x subscripts represents derivatives with respect to these variables, the model is:

$$\begin{aligned} \overbrace{u_t}^{\substack{\text{rate of change}\\ \text{of spores}}} &= \overbrace{\frac{1}{2}\frac{i}{n}}^{\text{mating}} \times \overbrace{\sigma i}^{\text{takeoff}} + \overbrace{\kappa u_{xx}}^{\text{diffusion}} - \overbrace{\mu u}^{\text{settling}}, \\ \underbrace{i_t}_{\substack{\text{rate of change}\\ \text{of lesions}}} &= \Big(\underbrace{q\mu u}_{\substack{\text{infection by}\\ \text{sexual spores}}} + \underbrace{p\alpha i}_{\substack{\text{infection by}\\ \text{asexual spores}}} \Big) \times \underbrace{(n-i)/n}_{\substack{\text{healthy fraction}\\ \text{of leaves}}} . \end{aligned} \tag{1}$$

2.1 Parameterization

To later evaluate whether model predictions are compatible with observed data, we parameterize the model with values for the Black Sigatoka disease of banana plants (Ravigné et al, unpublished manuscript), after (Stover 1980; Fouré 1982; Robert 2012; Landry 2015). We only focus on orders of magnitude here, since full parameterization would require dedicated experiments.

We first show that all parameters but the diffusion coefficient can be estimated through relatively common plant pathology laboratory measurements. The causal agent of Black Sigatoka (*M. fijiensis*) produces on average 200 asexual conidia per lesion. The average duration of the infection is 65 days (including the latent period, which is left implicit for simplicity), so we let $\alpha = 200/65 \approx 3$ asexual spores per day. In addition, the fungus may produce 4000 sexual ascospores per lesion, so we let $\sigma = 4000/65 \approx 60$ ascospores per day. Burie et al. (2008) report that spores of *Erysiphe necator* (a comparable ascomycete causing Powdery mildew of grape) lifted in the atmosphere fall within 30 min, so we let $\mu = 48$ per day as well. We assume the same infection efficiency for ascospores as for conidia: $p = q = 0.01$ (Landry 2015, and references therein).

The diffusion coefficient may be derived from gene flow in population genetics (the standard deviation of the distribution of parent-offspring distances; Mallet 2001). More specifically, Rieux et al. (2013) used neutral genetic cline theory to estimate gene flow in *M. fijiensis* as 1.2 km/generation$^{1/2}$. The time from leaf infection to ascospore release (about 50 days) represents a large part of the parasite generation time during which it does not diffuse, as accounted for in the immobile part of the model (i stage). To estimate the ascospore diffusion coefficient (u stage), we therefore consider only the actual time of diffusion (30 min on average). This yields $\kappa \approx 1.2^2 \times 48 \approx 70$ km^2 day^{-1} (3 km^2 hour^{-1}).

2.2 Dimensionless form of the Equations

We define T and L to be the temporal and geographical scales over which we are interested in the invasion process and rescale variables according to

Table 1 Dimensional variables and their estimated values for *M. fijiensis*

Label	Meaning	Unit	Value
t	Time		
x	Spatial location		
n	Total host density	Per unit area	
$i(x,t)$	Infected host density		
$u(x,t)$	Sexual spores density		
α	Asexual spore production rate	Per unit time	3 per day
σ	Sexual spore production rate	Per unit time	60 per day
p	Asexual spores infectivity	None	.01
q	Sexual spores infectivity	None	.01
κ	Sexual spores diffusion coefficient	Per unit area per unit time	70 km^2 per day
μ	Sexual spores deposition rate	Per unit time	48 per day
c	Spreading speed	Length unit per time unit	

$$t^* = \frac{t}{T}\,, \quad x^* = \frac{x}{L}\,, \quad i^* = \frac{i}{n}\,, \quad u^* = \frac{2\mu}{\sigma n} \times u\,, \tag{2}$$

and

$$a = \frac{2}{q\sigma T}\,, \quad \varepsilon = \frac{1}{\mu T}\,, \quad d = \sqrt{\frac{\kappa}{\mu L^2}}\,, \quad b = 2\frac{p\alpha}{q\sigma}. \tag{3}$$

The variable and parameters introduced are dimensionless. Dropping the asterisks for convenience, model (1) reads:

$$\begin{aligned} a i_t &= (u + bi)(1 - i)\,, \\ \varepsilon u_t &= i^2 - u + d^2 u_{xx}\,. \end{aligned} \tag{4}$$

We consider the case where the infection probability q is small and the rate of spore deposition μ is comparable to the rate of spore emission σ so

$$\frac{\varepsilon}{a} = \frac{q\sigma}{2\mu} \ll 1\,.$$

For instance, with the parameter values corresponding to *M. fijiensis* (Table 1) $\epsilon = 5.71 \times 10^{-5}$, $a = 9.13 \times 10^{-3}$, $b = 0.1$, and $d = 1.2 \times 10^{-2}$ with $L = 100$ km and $T = 365$ days. Also, we consider $0 < \epsilon \ll d < 1$.

2.3 Nonlocal Integrodifferential Equation Approximation

Because we are interested in the asymptotic spread of the population over long timescales, we apply the quasi-steady-state approximation to the second equation of (4) to yield the density of sexual spores u directly in terms of the density of infected leaves i as

$$u(x,t) = \int_{-\infty}^{\infty} H(x-y) i^2(y,t)\,\mathrm{d}y\,. \tag{5}$$

Here

$$H(z) = \frac{1}{2d}\exp\left(-\frac{|z|}{d}\right) \tag{6}$$

is the fundamental solution to the modified Helmholtz equation

$$d^2 H_{xx} - H = -\delta(x-y)\,, \tag{7}$$

where δ is the Dirac delta function.

This yields a nonlocal integrodifferential equation for the rate of change of density of infected leaves with respect to time

$$a i_t = (1-i)\left(bi + \int_{-\infty}^{\infty} H(x-y) i^2(y,t)\,\mathrm{d}y\right). \tag{8}$$

2.4 Degenerate Reaction Diffusion Approximation

Looking at the spread of the population over a large spatial scale, we can take advantage of the small size of d and expand (5) in a Taylor series to give (see "Appendix" for more details):

$$u(x,t) \approx \left(i^2(x,t) + d^2 \frac{\partial^2}{\partial x^2}\left(i^2(x,t)\right)\right), \tag{9}$$

which yields a reaction diffusion model with a cubic reaction term and a nonlinear diffusion term which is degenerate at $i = 0$:

$$a i_t = (1-i)\left(bi + i^2(x,t) + d^2 \frac{\partial^2}{\partial x^2}\left(i^2(x,t)\right)\right). \tag{10}$$

Equivalently, a second-order Taylor expansion as d goes to zero in (8) yields the same equation. This also corresponds to considering a diffusion kernel $\tilde{H}(z) = \delta(z) + d^2\delta''(z)$ (Medlock and Kot 2003) instead of the Laplace kernel (6) in (5).

3 Analysis

3.1 Comparing Original and Approximate Models

We compared numerically the original model (1), its integrodifferential approximation (8), and its lower-order reaction-diffusion approximation (10). It turns out that the latter approximation holds for $b \ll 1$; otherwise, the integrodifferential approximation is much more accurate (Figs. 2, 3). Since the dimensionless parameter b corresponds to the ratio of asexual to sexual spore production (Eq. 3), this means that model (10) fits species reproducing predominantly in a sexual manner (which actually corresponds to *M. fijiensis*, where $b \approx 0.1$). From now on, we assume $0 \leq b \ll 1$.

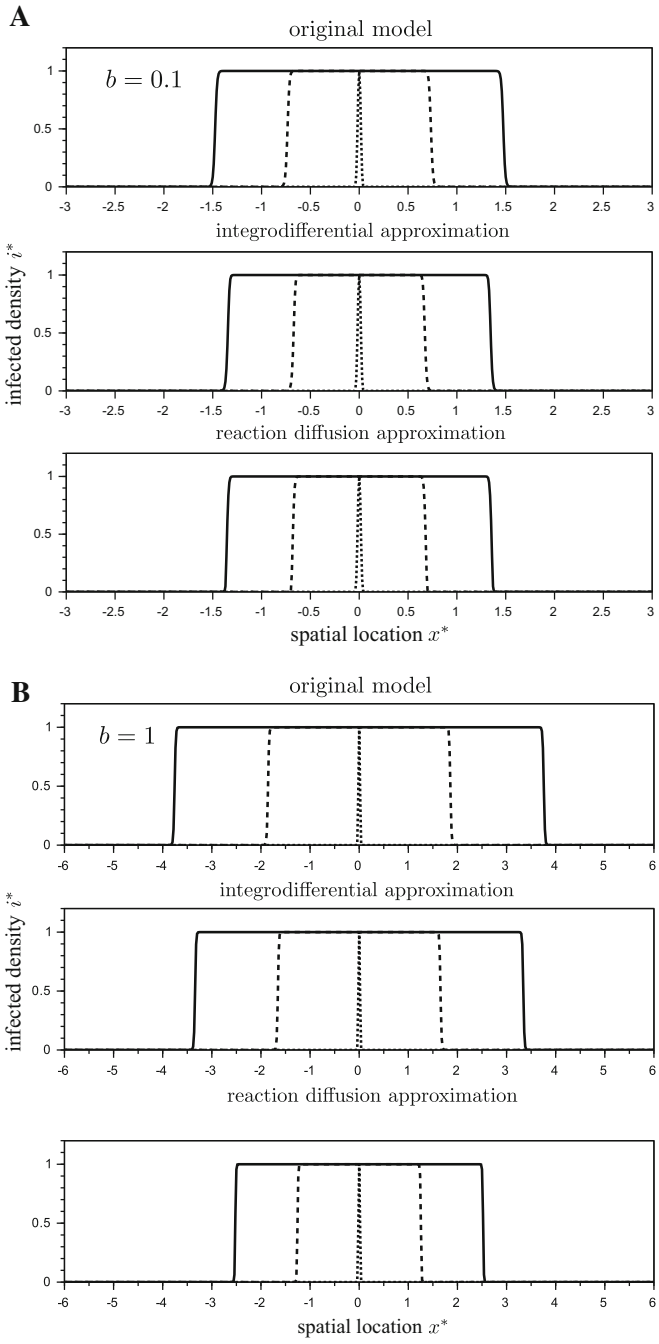

Fig. 2 The solutions of the original model (4), its integrodifferential approximation (8), and its lower-order reaction-diffusion approximation (10), for $t^* = 0$ (dotted), $t^* = 100$ (dashed), and $t^* = 200$ (solid), with $d = 10^{-2}$, $\epsilon = 10^{-5}$, $a = 10^{-2}$, and $b = 0.1$ (A), or $b = 1$ (B). This figure shows that the disease spreads as a travelling wave, and that the approximations are better for low b values

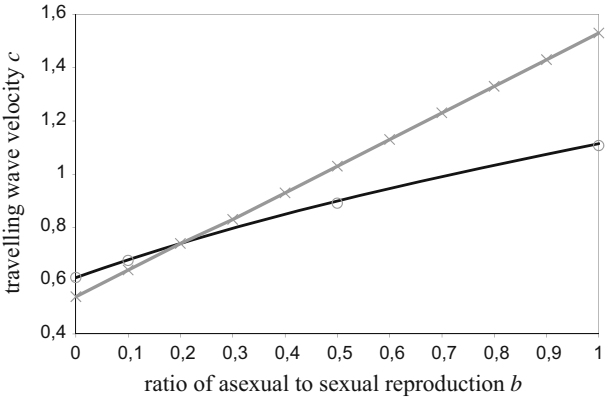

Fig. 3 Travelling wave velocity c^* as a function of the ratio of asexual to sexual spore production b. The black curve corresponds to the reaction-diffusion approximation (10), and the grey curve corresponds to the integrodifferential approximation (8), which is numerically very close to the original model (4) for $\epsilon = 10^{-5}$. Circles and crosses represent numerically calculated points for $d = 10^{-2}$, and $a = 10^{-2}$

3.2 Travelling Wave Form

We are interested in a travelling wave solution such that $i(x,t) = I(z)$, where $z = (x - ct)/d$, and c is the travelling wave velocity. Let $\hat{c} = ac/d$. Eq. (10) becomes

$$-\hat{c}I' = (1-I)\left(bI + I^2 + \left(I^2\right)''\right), \tag{11}$$

where the prime denotes differentiation w.r.t. z. This equation can be analysed in the phase plane with the travelling wave as a heteroclinic orbit connecting the invaded steady state $I = 1$ to the uninvaded steady state $I = 0$.

Equation (11) becomes

$$I' = J\,, \quad -\hat{c}J = (1-I)(bI + I^2 + 2J^2 + 2IJ')\,,$$

or equivalently

$$I' = J\,, \quad 2I(1-I)J' = -\hat{c}J - (1-I)(bI + I^2 + 2J^2)\,.$$

There are singularities at $I = 0\,, 1$ in the second equation. As in (Murray 2002), we remove these singularities by defining a new variable ζ as

$$2I(1-I)\frac{\mathrm{d}}{\mathrm{d}z} = \frac{\mathrm{d}}{\mathrm{d}\zeta}\,,$$

so that Eq. (11) becomes

$$\begin{aligned} \dot{I} &= 2IJ(1-I)\,, \\ \dot{J} &= -\hat{c}J - (1-I)(bI + I^2 + 2J^2)\,, \end{aligned} \tag{12}$$

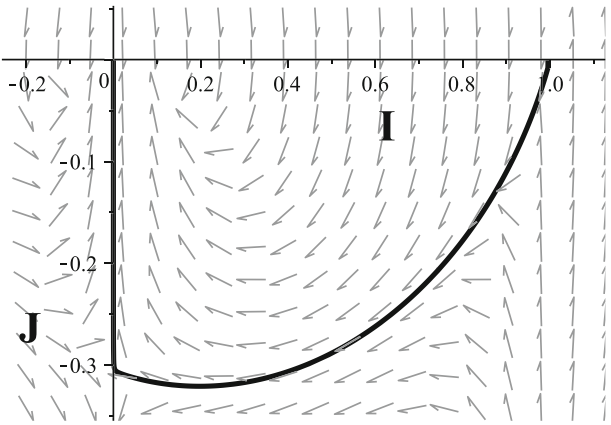

Fig. 4 Phase plane solution for $\hat{c} \approx 0.612$ in the strictly sexual case ($b = 0$)

where the dot denotes differentiation w.r.t. ζ. The equilibria of system (12) are

$$(I, J) = \quad (0, 0)\,, \quad (1, 0)\,, \quad \left(0, -\frac{\hat{c}}{2}\right)\,, \quad (-b, 0)\,.$$

The last equilibrium is not in an admissible region of the state space since I must be positive for biological consistency. Similarly to Murray (2002)'s Section 13.4 (Eq. 13.50 and Fig. 13.4), the travelling wave solution is the one which connects $(1, 0)$ to $(0, -\hat{c}/2)$. We numerically solved this two-point boundary value problem. Figure 4 shows that $\hat{c} \approx 0.612$ in the $b = 0$ case. Also, we numerically solved Eq. (10) and checked that the wave speed $\hat{c}$ indeed is around 0.612 for $b = 0$ (Fig. 5).

Figure 6 shows that, extending the analysis to positive b values, one can approximate the mapping $b \mapsto \hat{c}$ by the following power function

$$\hat{c} \approx .612\,(2.317b + 1)^{1/2}\,,$$

More accurately, a power of 0.5009 fits better in a least squares sense. However, we consider a simple square root function, as it is comparable with the exact solution from the analogous analysis without mate limitation ("Appendix"). We checked that numerical solutions as for $b = 0$ (Fig. 5) indeed agree with this mapping.

We obtain an explicit but approximate expression of the travelling wave speed: in terms of the original dimensional Eq. (15), the wave speed can be expressed as

$$c \approx .306 \left(\frac{\kappa}{\mu} q\sigma\,(4.634 \times p\alpha + q\sigma)\right)^{1/2}\,. \tag{13}$$

3.3 Impact of Mate Limitation on the Spreading Speed

Our aim was to get an explicit expression for the spreading speed of organisms that rely on sexual reproduction to produce their propagules, so as to evaluate the impact of

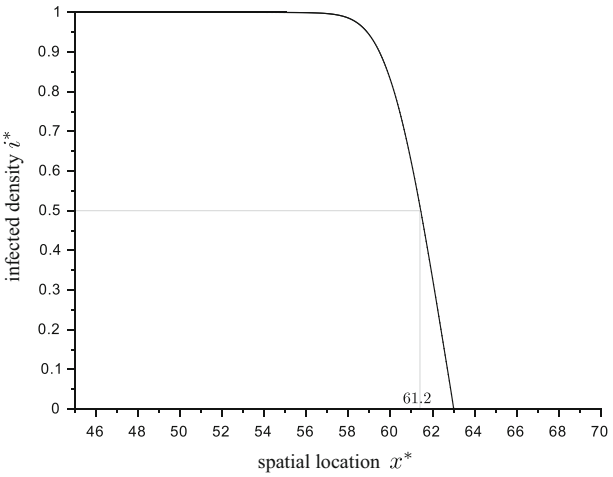

Fig. 5 The *solid line* corresponds to Eq. (10) numerical solution at $t^* = 100$ in the strictly sexual case ($b = 0$), with initial conditions $i^*(x^*) = 1$ for $-.01 \leq x^* \leq .01$, $i^*(x) = 0$ elsewhere. The wave front is at $x^* \approx 61.2$, which confirms the mathematical analysis ($c^* = d\hat{c}/a = 0.612$ with $d = 10^{-2}$, and $a = 10^{-2}$)

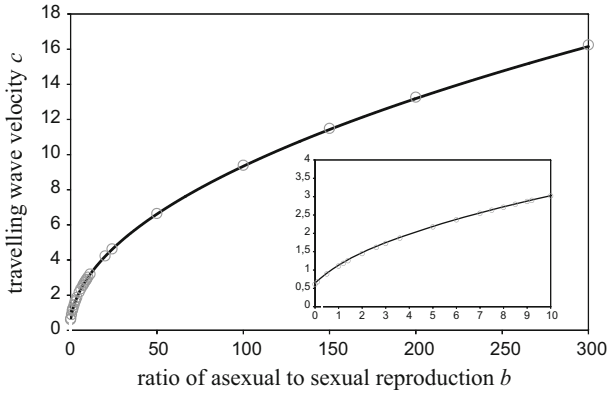

Fig. 6 Travelling wave velocity $\hat{c}$ as a function of the ratio of asexual to sexual spore production b, from the reaction-diffusion approximation (10). Calculated points (Sect. 3.2) are represented by open circles. The curve corresponds to $\hat{c} = .612\sqrt{2.317b + 1}$

mate limitation on their spreading speed. Without mate limitation (other assumptions being equal; "Appendix"), the spreading speed is

$$c = 2\left(\frac{\kappa}{\mu} q\sigma(p\alpha + q\sigma)\right)^{1/2}. \tag{14}$$

Equations (13) and (14) qualitatively agree: Regardless of mate limitation, the spreading speed scales linearly with sexual spore production ($q\sigma$), while it scales with the square root of asexual spore production ($p\alpha$). Quantitatively, mate limitation

slows the expected spreading speed of a strictly sexual parasite ($\alpha = 0$) by a factor $2/.306 \approx 6.5$. Also, with mate limitation, local asexual buildup contributes 4.634 times more to the spreading speed than without mate limitation.

4 Discussion

In this study, our aim was to evaluate the impact of mate limitation on the spreading speed of fungal plant parasites which combine sexual and asexual spore production. We built a simple model from first principles and showed how it can be parameterized from data available in the literature, focusing on *M. fijiensis* as a case study. Next we performed several approximations in order to get an explicit expression of the spreading speed as a function of sexual and asexual spore production.

Assuming that spore dispersal occurs on a shorter timescale than plant infection (30 minutes versus 65 days on average), we first made a quasi-steady-state approximation for the spore compartment and obtained an integrodifferential approximation of the original reaction-diffusion model (resulting in one equation instead of two). Focusing on the spread of the parasite over a large spatial scale, we showed that the model may be further approximated by a single reaction-diffusion equation. We compared numerically the original model, its integrodifferential approximation, and its lower-order reaction-diffusion approximation. We found that the latter approximation holds as long as the ratio of asexual to sexual spore production is low; otherwise, the integrodifferential approximation is much more accurate. This means that the reaction-diffusion approximation fits species reproducing predominantly in a sexual manner, which actually corresponds to Black Sigatoka (*M. fijiensis*).

Assuming that the ratio of asexual to sexual spore production is low, we then focused on the reaction-diffusion approximation and found a very good approximation of the spreading speed. Also, we analysed an analogous model without mate limitation and derived an exact expression of the spreading speed in this simpler model ("Appendix"). Both expressions are qualitatively similar. The spreading speed scales linearly with sexual spore production, while it scales with the square root of asexual spore production.

An important question is whether the model predicts a reasonable spreading speed in our case study, the causal agent of the Black Sigatoka disease of banana plants (*M. fijiensis*), for which parameter estimates are available (Table 1). We get $c \approx 542$ km per year without mate limitation and $c \approx 90$ km per year with mate limitation. The spreading speed obtained without mate limitation is almost one order of magnitude greater than the spreading speed obtained with mate limitation (actually greater by a factor 6). From the spatial temporal maps provided by Halkett et al. (2010), one can estimate the spreading speed of *M. fijiensis* to be slightly less than 100 km per year. This is in remarkably good agreement with our model with mate limitation (90 km per year). Therefore, the model taking into account mate limitation makes a better job than the model without mate limitation in estimating disease spreading speed.

Importantly, the model may not be restricted to mating through direct contact between two adjacent lesions on the same leaf, as in *M. fijiensis*. As mentioned earlier, the model may fit fungi which mate remotely through gamete-like propagules (sper-

matia) transported by wind or by insects (Bultman et al. 1995). Our results might, for instance, be applied to Ash Dieback disease (*H. fraxineus*), where asexual spores only serve as spermatia (Gross et al. 2014), and therefore rarely generate infections by themselves ($b \ll 1$). However, our results do not readily extend to this disease as the ash decline is a monocyclic disease (i.e. there is at most one infection cycle per year) which would rather require a discrete-time model. It would be interesting to compare our model to an analogous integrodifference equation (Veit and Lewis 1996). Also, measuring sexual spores production, infectivity, and dispersal of newly invading species would be interesting to compare the theoretical and observed spreading speeds. Indeed, we believe that the strength of this model is its simplicity, which makes it generic and relatively easy to parameterize. However, key to the parameterization was the diffusion coefficient, which came from genetic data on rates of spread. For a new invader this would hardly be available.

Our model ignores trade and focuses on natural dispersion. However, one of the prospects of this study is to show that mate limitation entails a critical patch size (here an initial infected area) below which the disease cannot spread (Lewis and Kareiva 1993). This would be expected to limit the impact of trade or other anthropogenic pathways for disease dispersal when the focus created has a size too small to enable spread. This might explain the very regular wave-like spread of ash decline observed in France (Fig. 1; Husson et al. 2011).

Since the SI epidemiological model and the Verhulst logistic equation are mathematically equivalent, and because the present model was kept as simple as possible, it may apply to other species than fungi. In plants for instance, local asexual reproduction would correspond to vegetative growth and sexual propagule production would correspond to seed production. In self-incompatible plant species, mate finding indeed conditions sexual reproduction. Our diffusion model seems to better fit wind-dispersed species although it may nevertheless be relevant for some water or animal-dispersed plants. The probability for a spore to fall upon a healthy plant would correspond to the probability for a wind-dispersed seed to fall into an empty site. For instance, Cheptou (2012)'s Table 1 reports several examples of long-distance wind-dispersed self-incompatible plants. These include the dwarf birch *Betula nana* and the mountain avens *Dryas octopetala*, which can be found in the Svalbard Archipelago, although they originate from Russia (Alsos et al. 2007).

Last, this paper is likely the first to deal with how fast a species spreads as a function of the ratio of asexual to sexual reproduction (b). We hope that such a shift in perspective will lead to new insights regarding self-incompatible species in which sex conditions dispersal.

Possibilities for future research include:

- demonstrating the convergence of the solutions of (4) and (8) as ε goes to zero,
- exploring travelling wave solutions in the integrodifferential model (8) with nonlinear dispersal (mate limitation) and comparing with the linear (mate unlimited) theory (Medlock and Kot 2003),
- extending our study to a 2-dimensional spatial domain and exploring how the critical patch size (Lewis and Kareiva 1993) depends on the ratio of sexual to asexual reproduction,

– including stochastic mating in the model as it may have important consequences even under a balanced sex ratio (Wilson and Harder 2003).

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Appendix: Analogous Analysis Without Mate Limitation

In this section, sexual spores can be produced *regardless* of the presence of a mate, which amounts to removing the probability $(i/2)/n$ in Eq. (1). Without mate limitation, model (1) becomes

$$\begin{aligned} u_t &= \sigma i - \mu u + \kappa u_{xx}\,, \\ i_t &= (q\mu u + p\alpha i)\,(n-i)/n\,. \end{aligned} \tag{15}$$

We rescale variables according to

$$t^* = \frac{t}{T}\,, \quad x^* = \frac{x}{L}\,, \quad i^* = \frac{i}{n}\,, \quad u^* = \frac{\mu}{\sigma n} \times u\,,$$

and

$$a = \frac{1}{q\sigma T}\,, \quad \varepsilon = \frac{1}{\mu T}\,, \quad b = \frac{p\alpha}{q\sigma}\,, \quad d = \sqrt{\frac{\kappa}{\mu L^2}}\,.$$

Dropping the asterisks for convenience, model (15) reads:

$$\begin{aligned} a i_t &= (u + bi)(1-i)\,, \\ \varepsilon u_t &= i - u + d^2 u_{xx}\,. \end{aligned} \tag{16}$$

Applying the quasi-steady-state approximation to the second equation of (16) yields the following nonlocal integrodifferential equation for i:

$$a i_t = (1-i)\left(bi + \int_{-\infty}^{\infty} H(x-y) i(y,t)\,\mathrm{d}y\right)\,, \tag{17}$$

where H is the Laplace kernel (6).

A Taylor expansion of $i(y,t)$ at x in (17) yields

$$\begin{aligned}\int_{-\infty}^{\infty} H(x-y)i(y,t)\,\mathrm{d}y = i(x,t)\int_{-\infty}^{\infty} H(x-y)\,\mathrm{d}y \\ + i_x(x,t)\int_{-\infty}^{\infty}(x-y)H(x-y)\,\mathrm{d}y \\ + \frac{i_{xx}(x,t)}{2}\int_{-\infty}^{\infty}(x-y)^2 H(x-y)\,\mathrm{d}y \\ + \cdots . \end{aligned} \tag{18}$$

Using the moments of the Laplace distribution in (18), we get

$$a i_t = (1-i)(bi + i + d^2 i_{xx} + d^4 i_{xxxx} + \ldots)\,. \tag{19}$$

A second-order Taylor expansion as d goes to zero in (19) yields

$$a i_t \approx (1-i)\left(bi + i + d^2 i_{xx}\right)\,. \tag{20}$$

Equation (20) can be expressed in a travelling wave form as

$$-\hat{c} I' = (1-I)\left((b+1)\,I + I''\right)\,, \tag{21}$$

where $\hat{c} = ac/d$, and the prime denotes differentiation w.r.t. $z = (x - ct)/d$. Proceeding as in Sect. 3.2, Eq. (21) can be expressed as a dynamical system:

$$\begin{aligned}\dot{I} &= J(1-I)\,, \\ \dot{J} &= -\hat{c}J - (1-I)(b+1)I\,. \end{aligned} \tag{22}$$

Its equilibria are $(I, J) = (0, 0)$ and $(1, 0)$. Let G be the associated Jacobian matrix:

$$G = \begin{pmatrix} -J & 1-I \\ -(b+1)(1-2I) & -\hat{c} \end{pmatrix}\,.$$

Linearizing around $(0, 0)$, we get

$$G_{(0,0)} = \begin{pmatrix} 0 & 1 \\ -(b+1) & -\hat{c} \end{pmatrix}\,,$$

whose eigenvalues are

$$\lambda_{\pm} = \frac{-\hat{c} \pm \sqrt{\hat{c}^2 - 4(b+1)}}{2}\,.$$

So $(0, 0)$ is a stable node if $\hat{c} > 2\sqrt{b+1}$, and a stable spiral otherwise. From Murray (2002)'s Sect. 13.2 (Eq. 13.12 and Fig. 13.1), we conjecture that there is a trajectory

from $(1, 0)$ to $(0, 0)$ lying entirely in the quadrant $I \geq 0$, $J \leq 0$ with $0 \leq I \leq 1$ for *all* wave speeds $\hat{c} \geq \hat{c}^\star$, with

$$\hat{c}^\star = 2\sqrt{b+1}\,.$$

We numerically checked that only $\hat{c}^\star$ is relevant, greater wave speeds being unstable. In terms of the original dimensional Eq. (15), the wave speed can be expressed as

$$c = q\sigma\sqrt{\frac{\kappa}{\mu}}2\sqrt{\frac{p\alpha}{q\sigma}+1}\,.$$

References

Allee WC, Park O, Emerson AE, Park T, Schmidt KP et al (1949) Principles of animal ecology, 1st edn. WB Saunders Co., Ltd, Philadelphia

Alsos IG, Eidesen PB, Ehrich D, Skrede I, Westergaard K, Jacobsen GH, Landvik JY, Taberlet P, Brochmann C (2007) Frequent long-distance plant colonization in the changing arctic. Science 316(5831):1606–1609

Aylor DE (2003) Spread of plant disease on a continental scale: role of aerial dispersal of pathogens. Ecology 84(8):1989–1997

Baker HG (1955) Self-compatibility and establishment after 'long-distance' dispersal. Evolution 9(3):347–349

Billiard S, López-Villavicencio M, Devier B, Hood ME, Fairhead C, Giraud T (2011) Having sex, yes, but with whom? inferences from fungi on the evolution of anisogamy and mating types. Biol Rev 86(2):421–442

Bonner JT (1958) The relation of spore formation to recombination. Am Nat 92:193–200

Brown JK, Hovmøller MS (2002) Aerial dispersal of pathogens on the global and continental scales and its impact on plant disease. Science 297(5581):537–541

Bultman TL, White JF Jr, Bowdish TI, Welch AM, Johnston J (1995) Mutualistic transfer of Epichloë spermatia by Phorbia flies. Mycologia 87(2):182–189

Burie JB, Calonnec A, Ducrot A (2006) Singular perturbation analysis of travelling waves for a model in phytopathology. Math Model Nat Phenom 1(1):49–62

Burie JB, Calonnec A, Langlais M (2008) Modeling of the invasion of a fungal disease over a vineyard. In: Mathematical modeling of biological systems, volume II, Springer, pp 11–21, http://www6.bordeaux-aquitaine.inra.fr/sante-agroecologie-aquitaine.inra.fr/sante-agroecologie-vignoble/layout/set/print/content/download/3769/36021/file/Pub13-ACL-Burie-07.pdf

Castel M, Mailleret L, Andrivon D, Ravigné V, Hamelin FM (2014) Allee effects and the evolution of polymorphism in cyclic parthenogens. Am Nat 183(3):E75–E88

Cheptou PO (2012) Clarifying baker's law. Ann Bot 109(3):633–641

Cheptou PO, Massol F (2009) Pollination fluctuations drive evolutionary syndromes linking dispersal and mating system. Am Nat 174(1):46–55

Cunniffe NJ, Gilligan CA (2008) Scaling from mycelial growth to infection dynamics: a reaction diffusion approach. Fungal Ecol 1(4):133–142

Desprez-Loustau ML, Robin C, Buée M, Courtecuisse R, Garbaye J, Suffert F, Sache I, Rizzo DM (2007) The fungal dimension of biological invasions. Trends Ecol Evol 22(9):472–480

Ene IV, Bennett RJ (2014) The cryptic sexual strategies of human fungal pathogens. Nat Rev Microbiol 12(4):239–251

Fisher MC, Henk DA, Briggs CJ, Brownstein JS, Madoff LC, McCraw SL, Gurr SJ (2012) Emerging fungal threats to animal, plant and ecosystem health. Nature 484(7393):186–194

Fouré E (1982) Les cercosporioses du bananier et leurs traitements. comportement des variétés. 1: Incubation et évolution de la maladie. Fruits 37(12):749–766

Frantzen J, van den Bosch F (2000) Spread of organisms: can travelling and dispersive waves be distinguished? Basic Appl Ecol 1(1):83–92

Garrett K, Bowden R (2002) An Allee effect reduces the invasive potential of Tilletia indica. Phytopathology 92(11):1152–1159

Gascoigne J, Berec L, Gregory S, Courchamp F (2009) Dangerously few liaisons: a review of mate-finding allee effects. Pop Ecol 51(3):355–372

Gross A, Holdenrieder O, Pautasso M, Queloz V, Sieber TN (2014) Hymenoscyphus pseudoalbidus, the causal agent of European ash dieback. Mol Plant Pathol 15(1):5–21

Gurney W, Nisbet R (1975) The regulation of inhomogeneous populations. J Theor Biol 52(2):441–457

Halkett F, Coste D, Rivas Platero GG, Zapater MF, Abadie C, Carlier J (2010) Genetic discontinuities and disequilibria in recently established populations of the plant pathogenic fungus Mycosphaerella fijiensis. Mol Ecol 19(18):3909–3923

Husson C, Scala B, Caël O, Frey P, Feau N, Ioos R, Marçais B (2011) Chalara fraxinea is an invasive pathogen in France. Eur J Plant Pathol 130(3):311–324

Landry C (2015) Modélisation des dynamiques de maladies foliaires de cultures pérennes tropicales différentes échelles spatiales: cas de la cercosporiose noire du bananier. PhD thesis, Université des Antilles, http://www.theses.fr/2015AGUY0835

Lewis M, Kareiva P (1993) Allee dynamics and the spread of invading organisms. Theor Pop Biol 43(2):141–158

Lutscher F (2008) Density-dependent dispersal in integrodifference equations. J Math Biol 56(4):499–524

Mallet J (2001) Insect movement: mechanisms and consequences. In: Woiwod I, Reynolds D, Thomas C (eds) Gene flow. CABI, pp 337–360

Massol F, Cheptou P (2011a) When should we expect the evolutionary association of self-fertilization and dispersal? Evolution 65(5):1217

Massol F, Cheptou PO (2011b) Evolutionary syndromes linking dispersal and mating system: the effect of autocorrelation in pollination conditions. Evolution 65(2):591–598

Medlock J, Kot M (2003) Spreading disease: integro-differential equations old and new. Math Biosci 184(2):201–222

Morel-Journel T, Girod P, Mailleret L, Auguste A, Blin A, Vercken E (2015) The highs and lows of dispersal: how connectivity and initial population size jointly shape establishment dynamics in discrete landscapes. Oikos. doi:10.1111/oik.02718

Mundt CC, Sackett KE, Wallace LD, Cowger C, Dudley JP (2009) Long-distance dispersal and accelerating waves of disease: empirical relationships. Am Nat 173(4):456–466

Murray JD (2002) Mathematical biology I: an introduction, interdisciplinary applied mathematics, 3rd edn. Springer, New York

Pannell JR, Auld JR, Brandvain Y, Burd M, Busch JW, Cheptou PO, Conner JK, Goldberg EE, Grant AG, Grossenbacher DL et al (2015) The scope of baker's law. New Phytol 208:656–667

Pernaci M, De Mita S, Andrieux A, Pétrowski J, Halkett F, Duplessis S, Frey P (2014) Genome-wide patterns of segregation and linkage disequilibrium: the construction of a linkage genetic map of the poplar rust fungus Melampsora larici-populina. Front Plant Sci 5:712

Powell JA, Slapničar I, van der Werf W (2005) Epidemic spread of a lesion-forming plant pathogen-analysis of a mechanistic model with infinite age structure. Linear Algebra Appl 398:117–140

Rieux A, Lenormand T, Carlier J, Lapeyre de Bellaire L, Ravigné V (2013) Using neutral cline decay to estimate contemporary dispersal: a generic tool and its application to a major crop pathogen. Ecol Lett 16(6):721–730

Rieux A, Soubeyrand S, Bonnot F, Klein EK, Ngando JE, Mehl A, Ravigne V, de Bellaire LdL (2014) Long-distance wind-dispersal of spores in a fungal plant pathogen: estimation of anisotropic dispersal kernels from an extensive field experiment. PloS ONE 9(8):e103,225

Robert S (2012) Emergence mondiale de la maladie des raies noires du bananier: histoire de l'invasion et stratégie de vie du champignon phytopathogène *Mycosphaerella fijiensis*. PhD thesis, Université de Montpellier 2

Roques L, Garnier J, Hamel F, Klein EK (2012) Allee effect promotes diversity in traveling waves of colonization. Proc Natl Acad Sci USA 109(23):8828–8833

Roy B (1994) The effects of pathogen-induced pseudoflowers and buttercups on each other's insect visitation. Ecology 75(2):352–358

Shaw A, Kokko H (2015) Dispersal evolution in the presence of allee effects can speed up or slow down invasions. Am Nat 185(5):631

Soubeyrand S, Laine AL, Hanski I, Penttinen A (2009) Spatiotemporal structure of host-pathogen interactions in a metapopulation. Am Nat 174(3):308–320

Stokes A (1976) On two types of moving front in quasilinear diffusion. Math Biosci 31(3):307–315
Stover R (1980) Sigatoka leaf spots of bananas. Plant Dis 64(8):751
Taylor CM, Hastings A (2005) Allee effects in biological invasions. Ecol Lett 8(8):895–908
Travis JM, Murrell DJ, Dytham C (1999) The evolution of density-dependent dispersal. Proc R Soc B 266(1431):1837–1842
Veit RR, Lewis MA (1996) Dispersal, population growth, and the Allee effect: dynamics of the house finch invasion of eastern North America. Am Nat 148:255–274
Vercken E, Kramer A, Tobin P, Drake J (2011) Critical patch size generated by allee effect in gypsy moth, *Lymantria dispar* (L.). Ecol Lett 14(2):179–186
Williams GC (1975) Sex and evolution. Princeton University Press, Princeton
Wilson W, Harder L (2003) Reproductive uncertainty and the relative competitiveness of simultaneous hermaphroditism versus dioecy. Am Nat 162(2):220–241