Host diversification may split epidemic spread into two successive fronts advancing at different speeds

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Host diversification may split epidemic spread into two successive fronts advancing at different speeds

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Abstract Host diversification methods such as within-field mixtures (or field mosaics, depending on the spatial scale considered) are promising methods for agroecological plant disease control. We explore disease spread in host mixtures (or field mosaics) composed of two host genotypes (susceptible and resistant). The pathogen population is composed of two genotypes (wild-type and resistance-breaking). We show that for intermediate fractions of resistant hosts, the spatial spread of the disease may be split into two successive fronts. The first front is led by the wild-type pathogen and the disease spreads faster, but at a lower prevalence, than in a resistant pure stand (or landscape). The second front is led by the resistance-breaking type, which spreads slower than in a pure resistant stand (or landscape). The wild-type and the resistance-breaking genotype coexist behind the invasion fronts, resulting in the same prevalence as in a resistant pure stand. This study shows that host diversification methods may have a twofold effect on pathogen spread compared to a resistant pure stand (or landscape): on one hand they accelerate disease spread, and on the other hand they slow down the spread of the resistance-breaking genotype. This work contributes to a better understanding of the

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multiple effects underlying the performance of host diversification methods in agroecology.

1 Introduction

The negative impacts of pesticides on biodiversity and human health require developing more ecological methods to control plant diseases [42,79,78]. So far, the main alternative to using pesticides against plant pathogens has been to breed genetically resistant plant varieties or cultivars and deploy them as pure stands [77]. Under these conditions, populations of pathogens often evolve and break down resistance genes after a few years, while a breeding program may require at least a decade [8,81]. More sustainable control methods will require diversifying genetic resistance over time (cultivar rotations) [4,49], and/or in space. Spatial diversification of genetic resistance can be achieved either at the field scale (host mixtures) [34,6,65,50,62,63,29,19,43,60,13] or at the scale of the landscape (field mosaics) [66,18,16,41,52,59,58,74].

Host mixtures consist in growing several varieties of the same plant species in the same field and at the same time [76,46]. Mixtures are or have been used against plant pathogens in various regions of the world [21,82,27,56]. Although host mixtures have long been studied both theoretically [37,34] and experimentally [35,76], their design remains to be optimized to be more widely and efficiently used [43,5]. So far, most empirical studies of epidemic control through host diversification have taken place at the field scale, but a few exceptions [48,51] suggest that host diversification at the landscape scale (field mosaics) would also be effective [53].

Host diversification is often achieved with resistant and susceptible plants in which resistance is qualitative, meaning that infection either succeeds or fails (as opposed to quantitative resistance, which only partially decreases the success of infection). Qualitative resistance is often conferred by major resistance genes and driven by gene-for-gene interactions [22,44]. Pathogen genotypes can then be classified into two types: the resistance-breaking type, which can successfully infect both resistant and susceptible hosts, and the wild-type, which can successfully infect susceptible hosts only.

Infection sites may be individual plants, or smaller infection units such as the size of a lesion, if the spatial domain considered is a single field [80]. At a larger spatial scale, albeit with a lower spatial resolution, infection sites may be regarded as fields if the spatial domain considered is a landscape. Previous authors [47] introduced the term “genotype unit area” to denote the contiguous area occupied by a single host genotype [53]. From now on, we will use the term “infection site” to mean the same thing as “genotype unit area” at the landscape scale. Infection sites correspond to either resistant or susceptible host genotypes. If the spatial domain is a field, resistant and susceptible infection sites form a host mixture. If the spatial domain is a landscape, resistant and susceptible infection sites form a mosaic of pure stands in the landscape.

Models can be spatially implicit [34,50,18,29,19,43,16,60,13] or spatially explicit [6,65,62,63,66,41,59,58,52,74]. See [57] for a recent review. Among spatially explicit models, the few that tackle the speed of pathogen spread focus on host mixtures [6,65]. In these models, the pathogen population is assumed to be wild-type, that is unable to infect resistant hosts. In this study, we are interested in comparing the spreading speeds of resistance-breaking and wild-type pathogens invading a spatial domain where resistant and susceptible infection sites are mixed.

An outline of the paper is as follows. We first present the canola-clubroot pathosystem as a motivating example. Then, we describe the epidemic model in Section 3, along with its biological interpretation. We look into the analogous spatially implicit (diffusionless) system in Section 4, and show how the model parameters drive the genetic composition of the pathogen population at equilibrium. We study the spatial spread of the disease in Section 5: we present formal derivations of the spreading speeds, that are checked with numerical simulations. Finally, in Section 6, we present a discussion of our results.

2 Motivating example: Clubroot in Alberta, Canada

Clubroot, caused by the obligate parasite *Plasmodiophora brassicae* Wor., is a soilborne disease of canola/oilseed rape (*Brassica napus* L) and other cruciferous hosts. Infection by *P. brassicae* is associated with the formation of large galls on the roots of susceptible plants, leading to yield losses estimated at 10%-15% globally [15]. In Alberta, Canada, clubroot was first identified on canola in 2003 [71] and by 2019 had been detected in more than 3000 fields [68]. Targeted surveys for the occurrence and spread of clubroot have been conducted annually since 2005, generating a large data set. The total infested area is estimated as the number of fields in which clubroot has been confirmed, multiplied by the standard size (160 acres) of an agricultural field in Alberta. Figure 1 shows the spread of clubroot as the square root of infested acres over time (years). The linear relationship indicates an approximately constant speed of disease spread.

The disease is controlled mainly by planting clubroot resistant canola varieties, which initially became available in 2009 and 2010. Resistance-breaking (RB) genotypes of *P. brassicae* were detected for the first time in Alberta in 2013 [69]. By 2019, RB genotypes had been confirmed in 239 clubroot-infested fields *vs.* 2928 fields infested by the wild-type (WT) genotype. The resistance-breaking designation of each *P. brassicae* isolate is confirmed in a greenhouse test, although given the large number of new cases of clubroot identified every year, only isolates recovered from resistant varieties are tested for their ability to overcome resistance. As above, the spread of the WT and RB genotypes of *P. brassicae* is expressed, respectively, as the square root of WT and RB infested acres over time (years) (Fig. 2). The spread of the RB genotypes is apparently slower than that of the WT. This suggests the RB genotypes might have a lower capacity to propagate compared to the WT.

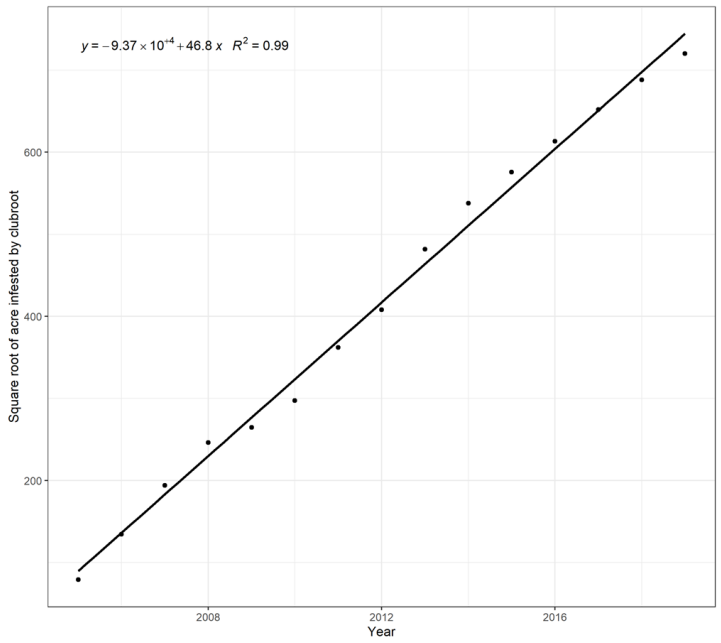

Fig. 1 Spread of clubroot (*Plasmodiophora brassicae*) in Alberta, Canada, expressed as the square root of *P. brassicae*-infested acres over time. The total number of infested acres was calculated by multiplying the number of fields with confirmed clubroot infestations by 160 (standard acreage of an agricultural field in Alberta). The linear regression equation is shown on the figure.

3 Model

We consider a spatial domain where susceptible and resistant infection sites are homogeneously mixed for simplicity.

There are two pathogen genotypes: the wild-type (WT) and the resistance-breaking (RB) genotype. The WT can only infect susceptible hosts, whereas the RB genotype can infect both susceptible and resistant hosts. For the sake of simplicity, we do not allow for coinfections in the model, meaning that each site can be infected by at most one pathogen genotype. This assumption fits resistant infection sites which cannot be infected by the WT. However, this is a strong assumption with respect to susceptible fields at the landscape scale. This is a weaker assumption for susceptible sites at the field scale, depending on whether the infection is systemic (as in viruses) or localized in the plant (as in rust fungi, which form very small lesions that may be defined as infection sites as well).

The total density of infection sites N in the spatial domain is assumed to be homogeneous and constant. A fraction p of the infection sites is resistant and the other fraction $1-p$ is susceptible. The density of susceptible and resistant sites infected by the RB genotype are I_s and I_r, respectively. The density of susceptible sites infected by the WT is J_s. Plant pathogens are generally

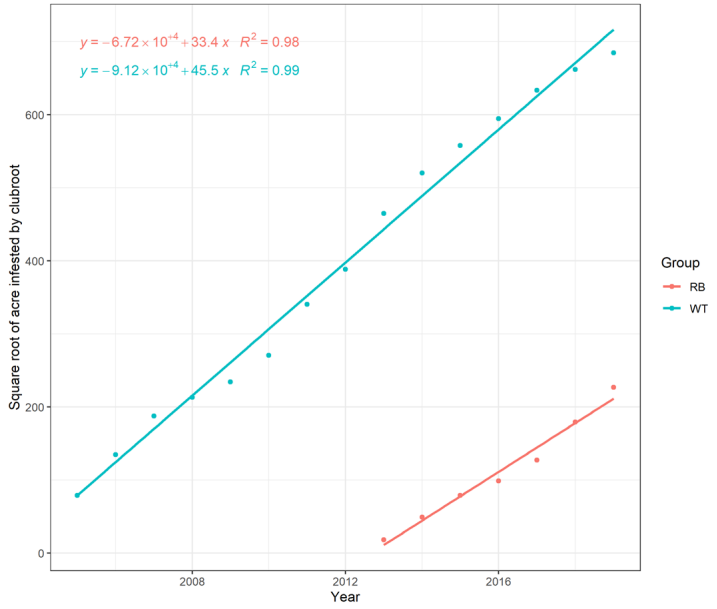

Fig. 2 Spread of wild-type (WT) and resistance-breaking (RB) genotypes of the clubroot pathogen *Plasmodiophora brassicae* in Alberta, Canada, expressed as the square root of WT (black) and RB (red) infested acres over time. The total number of infested acres was calculated by multiplying the number of fields with confirmed infestations of each genotype by 160 (standard acreage of an agricultural field in Alberta). The linear regression equations are shown in black and red, respectively, for the WT and RB data.

dispersed as free-living propagules such as fungal spores, or by insect vectors in the case of viruses. For simplicity we do not model possible insect vectors explicitly. The density of infectious propagules produced by the WT and the RB genotype are W and V, respectively. Each site infected by the WT emits infectious propagules at rate ζ.

We assume the RB genotype incurs a cost c which reduces the propagule emission rate ζ by a factor $0 \leq 1-c \leq 1$ relative to the WT. Similarly, a cost k reduces the infection efficiency of the propagules. The idea of a cost as a counterpart to the ability of breaking a resistance gene originated as a theoretical hypothesis to explain the often observed maintenance of polymorphism in pathogen populations, in both agricultural and wild ecosystems [73,64,24,70,8]. Since then, such a cost has been demonstrated and measured in a number of parasites, including bacteria [14,75], fungi [11,72,2,28,10,9,7], viruses [36,33,23,55,31,38], nematodes [12], and oomycetes [45].

The diffusion coefficient of infectious propagules is D. Each infectious propagule dies or deposits at rate δ. Their infection rate is θ. Infected sites recover or are replaced with uninfected sites at rate α. We assume no disease-induced mortality for simplicity, as is the case for most plant viruses and many other parasites. Time is denoted as t and space is denoted as $x \in (-\infty, +\infty)$ (i.e. we adopt a uni-dimensional conception of space for simplicity).

The model is:

$$\begin{aligned}
\frac{\partial W}{\partial t} &= \zeta J_s - \delta W + D\frac{\partial^2 W}{\partial x^2}\,,\\
\frac{\partial V}{\partial t} &= (1-c)\zeta(I_s + I_r) - \delta V + D\frac{\partial^2 V}{\partial x^2}\,,\\
\frac{\partial J_s}{\partial t} &= \theta W((1-p)N - J_s - I_s) - \alpha J_s\,,\\
\frac{\partial I_s}{\partial t} &= (1-k)\theta V((1-p)N - J_s - I_s) - \alpha I_s\,,\\
\frac{\partial I_r}{\partial t} &= (1-k)\theta V(pN - I_r) - \alpha I_r\,.
\end{aligned}$$

3.1 Non-dimensionalization

Let

$$w = \frac{\delta W}{\zeta N}\,, \quad v = \frac{\delta V}{\zeta N}\,, \quad a = \frac{J_s}{N}\,, \quad y = \frac{I_s}{N}\,, \quad z = \frac{I_r}{N}\,,$$

and

$$t^* = t\delta\,, \quad x^* = x\sqrt{\frac{\delta}{D}}\,,$$

and

$$R = \frac{\theta\zeta N}{\alpha\delta}\,, \quad \mu = \frac{\alpha}{\delta}\,.$$

Dropping the asterisks, and using subscripts to denote partial differentiation with respect to t or x, the model can be expressed as:

$$\begin{aligned}
w_t &= a - w + w_{xx}\,,\\
v_t &= (1-c)(y+z) - v + v_{xx}\,,\\
a_t &= \mu[Rw(1-p-a-y) - a]\,,\\
y_t &= \mu[(1-k)Rv(1-p-a-y) - y]\,,\\
z_t &= \mu[(1-k)Rv(p-z) - z]\,.
\end{aligned} \tag{1}$$

4 Analysis of the diffusionless system

To evaluate the underlying dynamical behavior, we first explore the diffusionless system, in which the dot represents differentiation over time:

$$\begin{aligned}
\dot{w} &= a - w\,,\\
\dot{v} &= (1-c)(y+z) - v\,,\\
\dot{a} &= \mu[Rw(1-p-a-y) - a]\,,\\
\dot{y} &= \mu[(1-k)Rv(1-p-a-y) - y]\,,\\
\dot{z} &= \mu[(1-k)Rv(p-z) - z]\,.
\end{aligned} \tag{2}$$

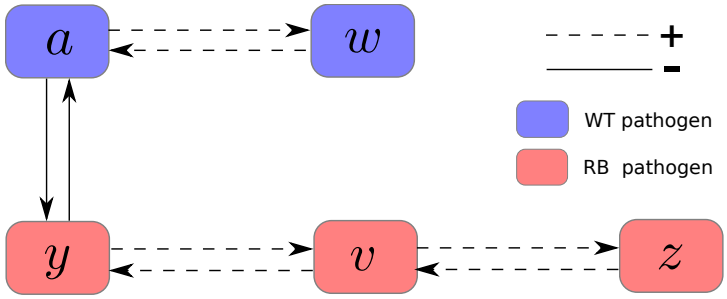

Fig. 3 Influence graph corresponding to System (1).

This system of ODE's is similar to that of [13], except that we consider explicit propagule compartments, which allows us to distinguish two types of resistance-breaking costs (c and k).

We define $P = a + y + z$ as the prevalence of the disease.

4.1 The system is Cooperative

System (2) has "Defined Feedback Relations" [67] meaning that the rates of change of all variables depends either positively or negatively of other variables (no sign change). More specifically, the rate of change of each variable in (w, v, a, y, z) depends non-negatively on other variables with two exceptions: the rate of change of a depends negatively on y, and the rate of change of y depends negatively on a (sign symmetry). Figure 3 shows the influence graph corresponding to System (2). One can check that every loop in the signed and undirected influence graph has an even number of negative edges. Hence, system (2) is cooperative [67]. Therefore, the solution of the system necessarily converges to an equilibrium (there is no attracting periodic orbit for instance).

4.2 Equilibria

System (2) has four equilibria:

- the "disease-free" equilibrium $(w, v, a, y, z) = (0, 0, 0, 0, 0)$.
- the "WT-only" equilibrium

$$(\bar{w}, 0, \bar{a}, 0, 0) := \left(\frac{R(1-p)-1}{R}, 0, \frac{R(1-p)-1}{R}, 0, 0 \right) ,$$

which is positive and therefore biologically exists if and only if

$$R(1-p) > 1 \,. \tag{3}$$

The prevalence of the disease is $P_W = (1-p) - 1/R$.

– the "RB-only" equilibrium $(0, \bar{v}, 0, \bar{y}, \bar{z})$, in which

$$\begin{aligned}
\bar{v} &:= \frac{R(1-c)(1-k)-1}{R(1-k)}\,,\\
\bar{y} &:= (1-p)\frac{R(1-c)(1-k)-1}{R(1-c)(1-k)}\,,\\
\bar{z} &:= p\frac{R(1-c)(1-k)-1}{R(1-c)(1-k)}\,.
\end{aligned}$$

The RB-only equilibrium is positive and therefore biologically exists if and only if

$$R(1-c)(1-k) > 1\,. \tag{4}$$

The prevalence of the disease is $P_R = 1 - 1/(R(1-c)(1-k))$.

– the "coexistence" equilibrium $(\hat{w}, \hat{v}, \hat{a}, \hat{y}, \hat{z})$, in which

$$\begin{aligned}
\hat{w} &:= \frac{c-p{+}k(1-c)}{c{+}k(1-c)}\,,\\
\hat{v} &:= \frac{Rp(1-c)-c{-}k(1-c)(Rp+1)}{Rc{+}kR((1-c)(1-k)+c)}\,,\\
\hat{a} &:= \frac{c-p{+}k(1-c)}{c{+}k(1-c)}\,,\\
\hat{y} &:= \frac{Rp(1-c)-c{-}k(1-c)(Rp+1)}{Rc{+}kR(1-c)}\,,\\
\hat{z} &:= \frac{Rp(1-c)-c{-}k(1-c)(Rp+1)}{R(1-c)(1-k)}\,.
\end{aligned}$$

The coexistence equilibrium is positive and therefore biologically exists if and only if

$$c{+}k(1-c) > p \quad \text{and} \quad p > \frac{c{+}k(1-c)}{R(1-c)(1-k)}\,, \tag{5}$$

which implies that conditions (3) and (4) are satisfied.
The prevalence of the disease is again $P_R = 1 - 1/(R(1-c)(1-k))$.

Figure 4A summarizes the picture in the plane (p, c) for $R = 5$ and $k = 0$. Figure 4B shows the prevalence of the disease (P) as a function of the fraction of resistant sites (p) for $R = 5$, $c = 0.5$, and $k = 0$. The shape of the graphs are qualitatively unchanged for other parameter values. As previously shown by [13], the prevalence decreases with respect to p (the proportion of resistant sites) until a threshold p value is attained, above which the prevalence remains constant.

To make clear under which conditions coexistence occurs, we next perform a mutual invasion analysis [61].

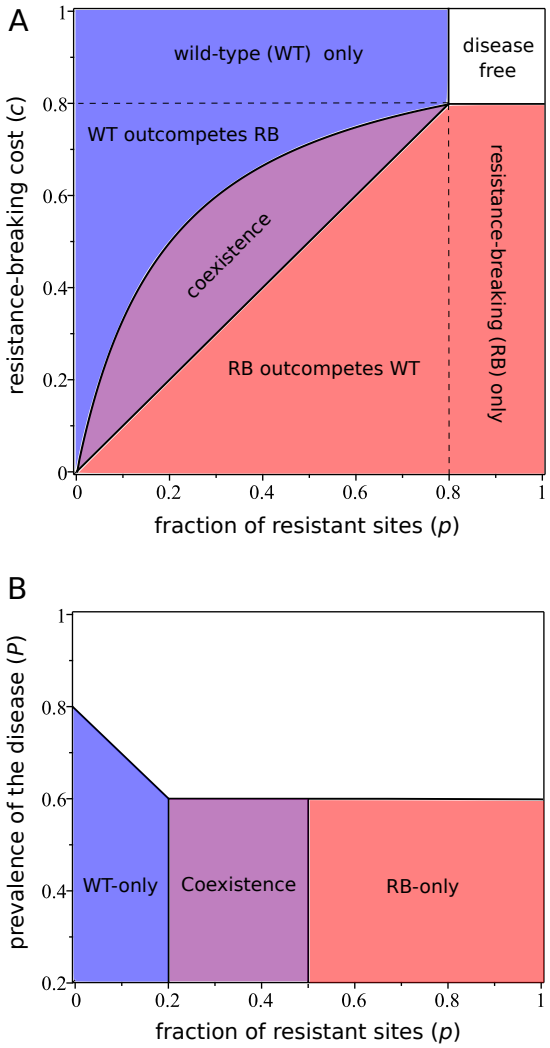

Fig. 4 Parameter values: $R = 5$, $c = 0.5$, and $k = 0$.

4.3 Can the WT invade the RB genotype?

First we wonder whether the WT can initially invade the RB-only equilibrium (assuming $R(1-c)(1-k) > 1$). The sub-model associated with this question is

$$\dot{w} = a - w\,,$$
$$\dot{a} = \mu[Rw(1-p-a-\bar{y}) - a]\,,$$

with

$$\bar{y} = (1-p)\frac{R(1-c)(1-k)-1}{R(1-c)(1-k)}\,.$$

This yields

$$\begin{aligned}\dot{w} &= a - w\,,\\ \dot{a} &= \mu\left[Rw\left(\frac{1-p}{R(1-c)(1-k)} - a\right) - a\right]\,.\end{aligned}$$

The corresponding Jacobian matrix is

$$J_{(w,a)} = \begin{pmatrix} -1 & 1 \\ \mu R\left(\frac{1-p}{R(1-c)(1-k)} - a\right) & -\mu(Rw+1) \end{pmatrix}\,,$$

which we evaluate at $(w,a) = (0,0)$:

$$J_{(0,0)} = \begin{pmatrix} -1 & 1 \\ \mu\frac{1-p}{(1-c)(1-k)} & -\mu \end{pmatrix}\,.$$

The trace of $J_{(0,0)}$ is negative and its determinant is

$$\frac{\mu(p-c-k(1-c))}{(1-c)(1-k)}\,.$$

Therefore, the wild-type can invade if and only if

$$c+k(1-c) > p\,. \tag{6}$$

4.4 Can the RB genotype invade the WT?

Now we wonder whether the RB genotype can initially invade the WT-only equilibrium (assuming $R(1-p) > 1$). The sub-model associated with this question is

$$\begin{aligned}\dot{v} &= (1-c)(y+z) - v\,,\\ \dot{y} &= \mu[(1-k)Rv(1-p-\bar{a}-y) - y]\,,\\ \dot{z} &= \mu[(1-k)Rv(p-z) - z]\,,\end{aligned}$$

with

$$\bar{a} = \frac{R(1-p)-1}{R}\,.$$

Letting $u = y+z$, the above system can be equivalently expressed as

$$\begin{aligned}\dot{v} &= (1-c)u - v\,,\\ \dot{u} &= \mu\left[(1-k)Rv\left(1 - \frac{R(1-p)-1}{R} - u\right) - u\right]\,.\end{aligned}$$

The corresponding Jacobian matrix is

$$J_{(v,u)} = \begin{pmatrix} -1 & 1-c \\ \mu(1-k)R\left(1-\frac{R(1-p)-1}{R}-u\right) & -\mu((1-k)Rv+1) \end{pmatrix},$$

which we evaluate at $(v,u)=(0,0)$:

$$J_{(0,0)} = \begin{pmatrix} -1 & 1-c \\ \mu(1-k)(Rp+1) & -\mu \end{pmatrix},$$

The trace of $J_{(0,0)}$ is negative and its determinant is

$$\mu(1-(1-k)(Rp+1)(1-c)).$$

Therefore, the WT can invade if and only if

$$p > \frac{c+k(1-c)}{R(1-c)(1-k)}. \tag{7}$$

4.5 Conclusions from the diffusionless system

The WT and RB pathogen can invade the disease-free equilibrium if and only if conditions (3) and (4) hold, respectively, which we assume (otherwise, we would not be concerned with these pathogen genotypes).

The WT can initially invade the RB-only equilibrium if condition (6) holds. The RB genotype can initially invade the WT-only equilibrium if condition (7) holds. Therefore, if both conditions (6) and (7) hold, the RB and WT can invade each other, and they are led to coexist. Conditions (6) and (7) are the same as conditions (5) implying the existence of the coexistence equilibrium. Since system (2) is cooperative and other equilibria are unstable, the coexistence equilibrium is globally asymptotically stable when it exists. We refer the reader to [13, Section S4] for more details on how the theory of cooperative systems can be used to show the global stability of the coexistence equilibrium in a related model.

5 Spreading of interacting pathogen genotypes with multiple fronts

We now get back to the spatial model (with diffusion). Although we are not able to rigorously prove the existence of travelling wave fronts, our numerical evidence strongly suggests that such fronts exist (Figure 5). Therefore we consider the possibility of travelling fronts for our system. Figure 5 shows the six possible patterns of travelling waves. We posit that, as can be found in some similar cooperative degenerate systems [40,20] (see also [26,17]), the front speeds are linearly determined as given as minimum possible wave speed based on the linearization at the leading edge of the wave. In this section, we apply the minimum wave speed approach [39,25,3,32] to linearized sub-models for finding the pathogen spreading speeds as a critical point in each sub-model, and then test the theoretical results against numerical simulations.

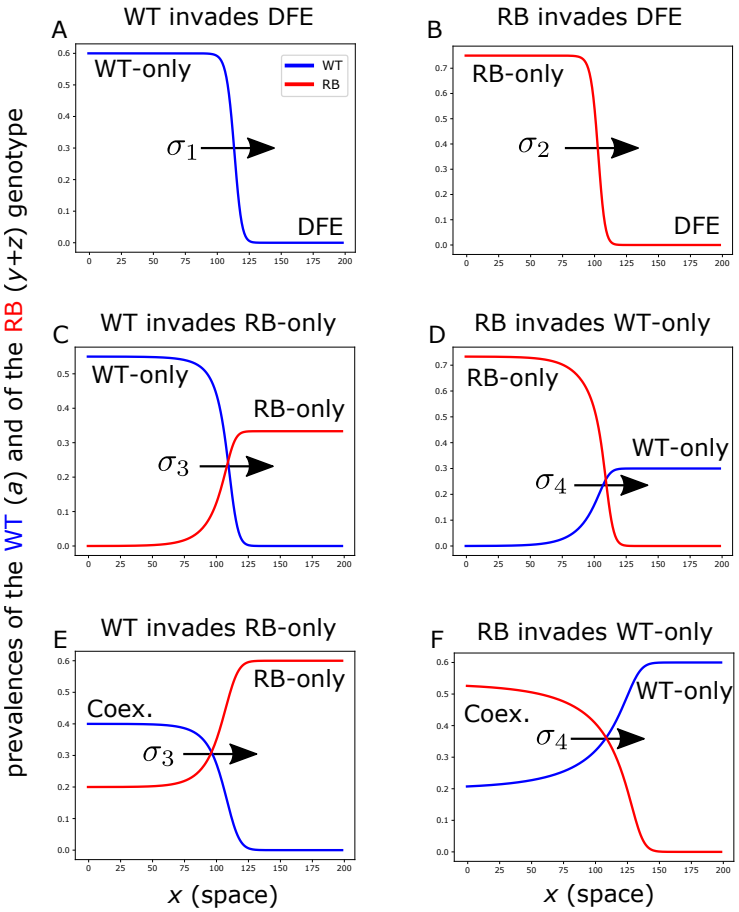

Fig. 5 Six possible patterns of travelling waves. Panel (A) shows a travelling wave solution connecting the disease-free equilibrium (DFE) to the WT-only equilibrium. Panel (B) shows the connection betwenn the DFE and the RB-only equilibrium. Panel (C) shows the connection between the RB-only and the WT-only equilibrium. Panel (D) shows the symmetric situation in which the RB genotype invades the WT. Panel (E) shows the connection between the RB-only and the Coexistence equilibrium. Panel (F) shows the connection between the WT-only and the Coexistence equilibrium. The invasion fronts spread to the right at speeds σ_1 (A), σ_2 (B), σ_3 (C, E), and σ_4 (D, F). Parameter values: $p = 0.2$, $c = 0.9$ (A), $p = 0.9$, $c = 0.2$ (B), $p = 0.25$, $c = 0.7$ (C), $p = 0.5$, $c = 0.25$ (D), $p = 0.3$, $c = 0.5$ (E), $p = 0.2$, $c = 0.25$ (F), and $R = 5$, $\mu = 0.1$, $k = 0$ (A–F).

5.1 Invading the disease-free equilibrium

In this section, we derive the spreading speeds of the WT and RB genotype when invading the disease-free equilibrium (Fig. 5A-B).

At leading edge invading the disease-free equilibrium, w, v, a, y, z have small positive values. Note that w and v equations in system (1) are linear. We

linearize the other equations at leading edge:

$$\begin{aligned} w_t &= a - w + w_{xx}\,, \\ v_t &= (1-c)(y+z) - v + v_{xx}\,, \\ a_t &\approx \mu[Rw(1-p) - a]\,, \\ y_t &\approx \mu[(1-k)Rv(1-p) - y]\,, \\ z_t &\approx \mu[(1-k)Rvp - z]\,. \end{aligned}$$

Using $u = y + z$, the above system can be equivalently expressed as

$$\begin{aligned} w_t &= a - w + w_{xx}\,, \\ a_t &= \mu[Rw(1-p) - a]\,, \\ v_t &= (1-c)u - v + v_{xx}\,, \\ u_t &= \mu[(1-k)Rv - u]\,, \end{aligned}$$

where (w, a) and (v, u) now form two uncoupled subsystems.

We are interested in traveling wave solutions such that

$$\underline{\ell} = \begin{pmatrix} w \\ a \\ v \\ u \end{pmatrix} = \underline{k} \exp(-s(x - \sigma t))\,,$$

in which $\underline{k}$ is an implicit column vector, σ is the wave speed, and s is the exponential decay rate of the wave profile at leading edge.

Plugging the previous expression in the system, we get

$$s\sigma\underline{\ell} = \underbrace{\begin{bmatrix} -1+s^2 & 1 & 0 & 0 \\ \mu R(1-p) & -\mu & 0 & 0 \\ 0 & 0 & -1+s^2 & (1-c) \\ 0 & 0 & \mu(1-k)R & -\mu \end{bmatrix}}_{A} \underline{\ell}\,,$$

which implies

$$\det \underbrace{\begin{bmatrix} -1+s^2-s\sigma & 1 & 0 & 0 \\ \mu R(1-p) & -\mu - s\sigma & 0 & 0 \\ 0 & 0 & -1+s^2-s\sigma & (1-c) \\ 0 & 0 & \mu(1-k)R & -\mu - s\sigma \end{bmatrix}}_{B = A - s\sigma \mathbb{1}} = 0\,,$$

in which $\mathbb{1}$ is the identity matrix. The above equality is equivalent to

$$\det \begin{bmatrix} -1+s^2-s\sigma & 1 \\ \mu R(1-p) & -\mu - s\sigma \end{bmatrix} \times \det \begin{bmatrix} -1+s^2-s\sigma & (1-c) \\ \mu(1-k)R & -\mu - s\sigma \end{bmatrix} = 0\,,$$

which is equivalent to

$$0 = [(-1 + s^2 - s\sigma)(-\mu - s\sigma) - \mu R(1-p)] \times [(-1 + s^2 - s\sigma)(-\mu - s\sigma) - \mu R(1-c)(1-k)] .$$

We now introduce Q to denote either $R(1-p)$ or $R(1-c)(1-k)$, which are the basic reproductive numbers of the WT and RB genotypes, respectively; see equations (3-4). From now on, we therefore assume $Q > 1$ so that the pathogens can invade the disease-free equilibrium. This allows us to consider a single equality:

$$(-1 + s^2 - s\sigma)(-\mu - s\sigma) - \mu Q = 0 . \tag{8}$$

Next we follow the approach for using this equation to calculate the minimum wave speed as outlined in [25]. Solving equation (8) for $\sigma > 0$ yields

$$\sigma(s, \mu, Q) := \frac{s^2 - \mu - 1 + \sqrt{(s^2 + \mu - 1)^2 + 4\mu Q}}{2s} .$$

Equation (8) can therefore be written to include the dependency of σ on s, μ and Q as

$$\mathcal{P}(\sigma(s, \mu, Q), s) := (-1 + s^2 - s\sigma(s, \mu, Q))(-\mu - s\sigma(s, \mu, Q)) - \mu Q = 0 .$$

Differentiating with respect to s, we have, for all s,

$$\frac{\mathrm{d}\mathcal{P}}{\mathrm{d}s} = \frac{\partial \mathcal{P}}{\partial \sigma}\frac{\partial \sigma}{\partial s} + \frac{\partial \mathcal{P}}{\partial s} = 0 . \tag{9}$$

We are interested in the minimum possible wave speed. Let

$$s^\star(\mu, Q) = \arg\min_s \sigma(s, \mu, Q) ,$$

and

$$\sigma^\star(\mu, Q) = \sigma(s^\star(\mu, Q), \mu, Q) .$$

Since $\sigma^\star$ is such that $\partial\sigma/\partial s = 0$, equation (9) yields

$$\frac{\partial \mathcal{P}}{\partial s}(\sigma^\star(\mu, Q), s^\star(\mu, Q)) = 0 . \tag{10}$$

Since $\mathcal{P}$ is cubic in s, $\partial\mathcal{P}/\partial s$ is quadratic in s. We are interested in the conditions on the coefficients that allow both polynomials to have a common root, $s^\star$. They are given by cancelling the resultant of the two polynomials (which is also the determinant of the Sylvester matrix of the two polynomials). Letting $\mathcal{P} = es^3 + fs^2 + gs + h$ yields $\partial\mathcal{P}/\partial s = 3es^2 + 2fs + g$, and the resultant is

$$r(e, f, g, h) = -e(f^2g^2 - 4eg^3 - 4f^3h + 18efgh - 27e^2h^2) ,$$

as described in equation (4.30) of reference [32]. The coefficients are identified as $e = -\sigma$, $f = \sigma^2 - \mu$, $g = \sigma(1 + \mu)$, and $h = -\mu(Q - 1)$. The equality $r(e, f, g, h) = 0$ can be equivalently expressed as a cubic with respect to σ^2:

$$E(\sigma^2)^3 + F(\sigma^2)^2 + G(\sigma^2) + H = 0 ,$$

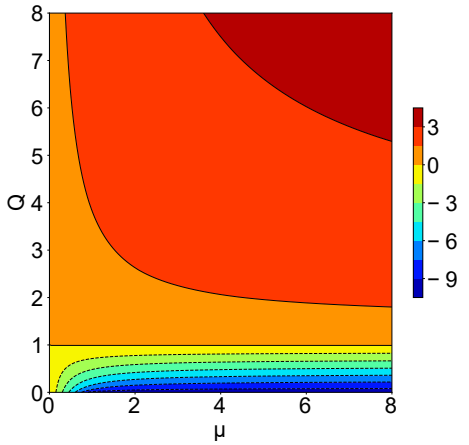

Fig. 6 Value of $\sigma^\star$ with respect to Q and μ, which is non positive if and only $Q \leq 1$. Note that $\sigma^\star$ has no biological meaning for $Q \leq 1$.

in which $E = 4\mu(Q-1) + (1+\mu)^2$, $F = 4 + 2\mu^3 + (6Q+2)\mu^2 + (18Q-8)\mu$, $G = \mu^4 - (6Q-8)\mu^3 - (27Q^2 - 36Q + 8)\mu^2$, and $H = -4\mu^4(Q-1)$. Since we assume $Q > 1$, we have that E is positive and H is negative, which means that we are in the same configuration as [25]. This implies that $\sigma^\star(\mu, Q)$ is uniquely defined as the square root of the largest root of the above cubic.

Although it is possible to write down the formula for the largest root of a cubic polynomial, we have no simple expression of $\sigma^\star(\mu, Q)$. However, this function only depends on two parameters, μ and Q. Therefore, one can plot $\sigma^\star$ as a function of μ and Q without loss of generality. One can easily check that for all $Q > 1$ and $\mu > 0$,

$$\frac{\partial \sigma^\star}{\partial Q}(\mu, Q) > 0\,, \tag{11}$$

meaning that $\sigma^\star$ increases as Q increases.

As a consequence, $c+k(1-c) > p$, or equivalently $(1-p) > (1-c)(1-k)$, implies

$$\sigma_1 := \sigma^\star(\mu, R(1-p)) > \sigma^\star(\mu, R(1-c)(1-k)) =: \sigma_2\,,$$

meaning that the WT spreads faster than the RB genotype. For $c+k(1-c) < p$, the inequality is reversed, and the RB genotype spreads faster than the WT.

5.2 Invading the RB-only equilibrium

In this section, we derive the spreading speed of the WT when invading the RB-only equilibrium (Fig. 5C-E).

At leading edge invading the RB-only equilibrium, w, a have small positive values while v, y, z are constant (equal to equilibrium values $\bar{v}, \bar{y}, \bar{z}$, resp.). We

linearize the a equation at leading edge:

$$\begin{aligned} w_t &= a - w + w_{xx}\,, \\ a_t &\approx \mu[Rw(1-p-\bar{y}) - a]\,, \end{aligned}$$

with

$$\bar{y} = (1-p)\frac{R(1-c)(1-k)-1}{R(1-c)(1-k)}\,.$$

This yields

$$\begin{aligned} w_t &= a - w + w_{xx}\,, \\ a_t &\approx \mu\left[\frac{1-p}{(1-c)(1-k)}w - a\right]\,. \end{aligned}$$

Proceeding as in Section 5.1, the spreading speed of the WT is

$$\sigma_3 := \sigma^\star\left(\mu, \frac{1-p}{(1-c)(1-k)}\right)\,.$$

As shown in Section 5.1, $\sigma_3 > 0$ is conditioned to the second argument of the function $\sigma^\star$ being greater than one, i.e. $(1-p)/[(1-c)(1-k)] > 1$. This condition is equivalent to (6), which is the condition for the WT to invade the RB genotype. Lastly, one can check that $\sigma_3 < \sigma_1$ is equivalent to condition (4), which is necessarily satisfied since we assumed the RB-only equilibrium exists.

5.3 Invading the WT-only equilibrium

In this section, we derive the spreading speed of the RB genotype when invading the WT-only equilibrium (Fig. 5D-F).

At leading edge invading the WT-only equilibrium, v, y, z have small positive values while w, a are constant (equal to equilibrium values $\bar{w}, \bar{a}$, resp.) in space. We linearize the (y, z) equations at leading edge:

$$\begin{aligned} v_t &= (1-c)(y+z) - v + v_{xx}\,, \\ y_t &\approx \mu[(1-k)Rv(1-p-\bar{a}) - y]\,, \\ z_t &\approx \mu[(1-k)Rvp - z]\,, \end{aligned}$$

with

$$\bar{a} = \frac{R(1-p)-1}{R}\,.$$

Using $u = y + z$, the above system can be equivalently expressed as

$$\begin{aligned} v_t &= (1-c)u - v + v_{xx}\,, \\ u_t &= \mu[(1-k)(1+Rp)v - u]\,. \end{aligned}$$

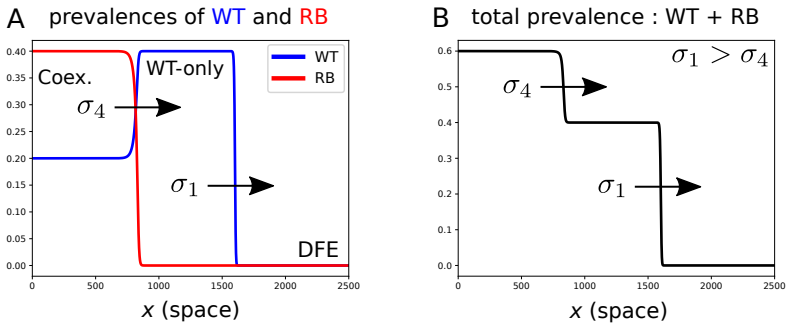

Fig. 7 Numerical simulations showing multiple fronts. See also the supplementary videos. Parameter values: $R = 5$, $c = 0.5$, $p = 0.4$, $\mu = 0.1$, $k = 0$. Initial conditions: $w(x,0) = v(x,0) = a(x,0) = 0.5, y(x,0) = 0.25, z(x,0) = 0.25$ for $0 \leq x \leq 20$, and 0 elsewhere..

Proceeding as in Section 5.1, the spreading speed of the RB genotype is

$$\sigma_4 := \sigma^\star(\mu, (1+Rp)(1-c)(1-k))\,.$$

As indicated in Section 5.1, $\sigma_4 > 0$ is conditioned to the second argument of the $\sigma^\star$ function being greater than one, i.e. $(1+Rp)(1-c)(1-k) > 1$. This condition is equivalent to (7), which is the condition for the RB genotype to invade the WT. One can lastly check that $\sigma_4 < \sigma_2$ is equivalent to condition (3), which is necessarily satisfied since we assumed the WT-only equilibrium exists.

5.4 Comparing spreading speeds under coexistence conditions

If $c{+}k(1-c) < p$, then the RB genotype invades the disease-free equilibrium faster than the WT and competitively excludes the later behind the invasion front. If $c{+}k(1-c) > p$ and $p < [c{+}k(1-c)]/[R(1-c)(1-k)]$, then the reverse situation happens: the WT invades the disease-free equilibrium faster than the RB genotype and competitively excludes the later behind the invasion front.

In the remaining situation, that is

$$c{+}k(1-c) > p > \frac{c{+}k(1-c)}{R(1-c)(1-k)}\,,$$

the WT invades the disease-free equilibrium faster than the RB genotype (since $c{+}k(1-c) > p$) but does not exclude the later behind the invasion front. Therefore, the RB genotype invades the WT-only equilibrium at speed $\sigma_4 < \sigma_1$ (since $\sigma_4 < \sigma_2$ and $\sigma_2 < \sigma_1$ in this case). Behind this second and slower invasion front, the WT and the RB genotype coexist steadily (Fig. 7). The reverse situation, in which the RB genotype would precede the WT while both are led to coexist, is not possible.

Note that the spreading speed of the RB genotype invading the disease-free equilibrium (σ_2) does not depend on p, so if the WT is faster than RB in a mixture, it is also faster than RB in a pure stand.

5.5 Numerical simulations

Numerical computations of system (1), presented in Figure 5 and Figure 7, are performed using the fourth order Runge-Kutta method for the first derivative with respect to time, combined with the second order finite difference scheme for the second derivative with respect to space.

Figure 7 shows the long-term pattern obtained from numerical simulations with parameters such that both pathogen genotypes (WT and RB) can coexist.

If the WT initially precedes the RB genotype, the WT spreads faster than the RB genotype and generates a first invasion front. For these parameter values, the RB genotype can still invade and coexist with the WT. This leads to the emergence of a second invasion front, advancing more slowly than the first front. Some authors refer to stacked fronts, e.g. [30], or propagating terraces, e.g. [54]. Between the first and the second front, the prevalence of the disease reaches a plateau. The prevalence of the disease is increased behind the second front, and is eventually the same as in a resistant pure stand (or landscape), see Fig. 4 and supplementary videos.

If the RB genotype initially precedes the WT, the WT makes up the delay and overtakes the RB genotype. In the long-run, the WT invasion front precedes the RB invasion front as in Fig. 7.

6 Discussion

Developing new or preexisting methods based on bio-diversification forms the basics of agroecology [1]. Plant host diversification (cultivar mixtures or field mosaics) are one of these. Several models explored the spreading speed of plant diseases in host mixtures [6,65]. In particular, it was shown that the spreading speed is an increasing concave function of the proportion of susceptible hosts [6], which is consistent with the spreading speed we obtained for the WT invading the disease-free equilibrium. However, previous studies did not consider pathogen diversity, specifically the possibility that the WT co-occur with a RB genotype. Since pathogen diversity and the competition between pathogen genotypes for susceptible hosts is a key component of the success of host mixtures [50,43,13], we explored a simple spatial model accounting for pathogen diversity.

The model we explored consists of 2 PDE's coupled to 3 ODE's. We showed that the model is cooperative, which led us to make a formal spreading speed analysis around each possible equilibrium. This allowed us to get a rather complete picture of the behaviour of the model. In particular, we were able to derive the spreading speed of the RB genotype invading an area previously invaded by the WT.

A notable feature of our analysis is that in host mixtures, the disease may spread along two fronts, with maximum prevalence behind the second front. The first front is led by the WT invading the disease-free equilibrium (as in [6]), and the second front (advancing at a lower speed) is led by the RB genotype invading the WT. Both pathogen genotypes eventually coexist at the rear of the front. The first front advances faster than the disease spreading speed in a resistant pure stand (or landscape). This is because the resistance-breaking cost exceeds the proportion of resistant hosts in this case. However, the prevalence just behind the first invasion front is lower than in a pure resistant stand (or landscape). This is because the WT is unable to infect resistant hosts. The second invasion front advances more slowly than the disease spreading speed in a resistant pure stand (or landscape). This is because the spread of the RB genotype is slowed down by the WT due to the competition for susceptible hosts. Behind the second invasion front however, the prevalence is the same as in a resistant pure stand (or landscape). Altogether, our results show that the spatial spread of the disease in host mixtures (or field mosaics) may have a twofold effect compared to its spread in a resistant pure stand (or landscape): on one hand the WT invades the disease-free area faster than the RB would, and on the other hand the spread of the RB genotype is slowed down due to the competition with the WT.

Our study therefore showed that the question of whether host mixtures (or field mosaics) are advantageous compared to resistant pure stands (or landscapes) is not straightforward, be it in terms of spreading speed or prevalence. However, we did not consider other features of host mixtures that are known to be key for their performance in the field, such as priming-induced cross-protection [13]. Moreover, we ignored co-infections of susceptible plants (or fields) by the WT and the RB genotype [74]. This model was a first step in these directions, that are left for future research.

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Conflict of interest

The authors declare that they have no conflict of interest.

Data Availability Statement

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

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