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The evolution of plant virus transmission pathways

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HIGHLIGHTS

- We use adaptive dynamics theory to study the evolution of plant viruses.
- Coexistence of infected and healthy plants is impossible in absence of pollen transmission.
- Evolutionary bistability may prevent vector transmission to replace pollen transmission.
- Evolutionary branching may lead to the coexistence of vector borne and non-vector-borne strains.

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ABSTRACT

The evolution of plant virus transmission pathways is studied through transmission via seed, pollen, or a vector. We address the questions: under what circumstances does vector transmission make pollen transmission redundant? Can evolution lead to the coexistence of multiple virus transmission pathways? We restrict the analysis to an annual plant population in which reproduction through seed is obligatory. A semi-discrete model with pollen, seed, and vector transmission is formulated to investigate these questions. We assume vector and pollen transmission rates are frequency-dependent and density-dependent, respectively. An ecological stability analysis is performed for the semi-discrete model and used to inform an evolutionary study of trade-offs between pollen and seed versus vector transmission. Evolutionary dynamics critically depend on the shape of the trade-off functions. Assuming a trade-off between pollen and vector transmission, evolution either leads to an evolutionarily stable mix of pollen and vector transmission (concave trade-off) or there is evolutionary bi-stability (convex trade-off); the presence of pollen transmission may prevent evolution of vector transmission. Considering a trade-off between seed and vector transmission, evolutionary branching and the subsequent coexistence of pollen-borne and vector-borne strains is possible. This study contributes to the theory behind the diversity of plant–virus transmission patterns observed in nature.

1. Introduction

Plant viruses naturally spread through three main transmission pathways: pollen, seed, and vector. Many plant viruses have vectors, providing the means for horizontal transmission from plant-to-plant (Gray and Banerjee, 1999; Bragard et al., 2013). Although various organisms serve as plant viral vectors, insects represent the most important group (Hull, 2014). Seed transmission (Sastry, 2013) serves as a major route for long-distance dissemination, provides an initial local source of inoculum for spread by vectors, and through vertical transmission enables virus survival at times when vector populations crash or go locally extinct. Just over 100 plant viruses are known to be seed-borne (Revers and Garcia, 2015). Virus transmission through pollen is also known (Mink, 1993; Card et al., 2007), which provides a pathway for an indirect form of vertical transmission, i.e. from an infected donor plant to the progeny of a healthy receptor plant. In addition pollen can provide a pathway for direct horizontal transmission. Finally, contact transmission can also occur, but there is little quantitative data on its occurrence in natural settings (Sacristán et al., 2011).

Vector transmission requires an active association with the virus, unlike passive transfer of infected pollen by insects. Specific interactions between virus and vector factors occur regardless of

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the type of virus/vector association, i.e. non-persistent (virus on mouth-parts/stylet of vector leading to short term transmission) or semi-persistent (movement of the virus to the foregut) (Ng and Falk, 2006; Blanc et al., 2011). For stylet-borne viruses the virus determinants for insect transmission reside on the viral coat protein (CP) (Ng and Perry, 2004). Some viruses, such as potyviruses, even have an additional virus-encoded protein, helper-component proteinase (Pirone and Blanc, 1996; Ng and Falk, 2006) which acts as a bridge for direct interaction with receptors on the aphid stylet as well as viral CP to facilitate vector transmission.

Plant viruses combine seed, pollen, and vector transmission pathways in a diverse and puzzling manner, in which it is difficult to discern consistent trends. Appendix A reports contrasting patterns as represented in extant virus species, documented from the literature, although restricted to the case of positive-sense single-stranded RNA (+ssRNA) viruses known to infect hosts with an annual life history. Some plant virus species have no known vector (Table A1), some others seem to be transmitted only by vectors (Table A2), while the majority combine seed/pollen and vector transmission (Table A3). Importantly, strains of the same virus species may be transmitted differently (Evans et al., 1970; Carroll, 1972; Stewart et al., 2005).

There may be a trade-off among modes of virus transmission. For instance, horizontal transmission rate is positively correlated with virulence (measured as the reduction in lifetime viable seed output of the host) in the *Barley stripe mosaic virus* (BSMV) – barley (*Hordeum vulgare*) system (Stewart et al., 2005). This suggests a trade-off between vertical (seed) and horizontal (vector) transmission in this virus species. Similarly in *Cucumber mosaic virus* (CMV) aphid (*Aphis gossypii*) transmission rate positively correlates with virus accumulation in tomato (*Lycopersicon esculentum* Mill.) (Escriu et al., 2000), whereas CMV virulence (measured as the negative effect of infection on plant fecundity) positively correlates with virus accumulation in *Arabidopsis thaliana* (Pagán et al., 2014). Thus when vector transmission correlates positively with virulence (negatively with fecundity) less seed transmission can occur suggesting a trade-off between seed and vector transmission. In addition, sequence variation in viral motifs may enhance and reduce different modes of virus transmission thus leading to a direct trade-off among transmission modes. For example, it was shown that a single amino-acid substitution in the coat protein (CP) coding region of *Soybean mosaic virus* (SMV) can both enhance aphid transmission rate and reduce seed transmission rate (p413-CP2mut; Jossey et al., 2013). More generally, there may be a trade-off between plant (pollen, seed) and animal (vector) transmission.

In this paper we do not address whether vector transmission preceded seed/pollen transmission or the reverse. Rather we concentrate on the mechanisms enabling seed/pollen and vector transmission to coexist in an evolutionarily stable manner, be it at the individual level or at the population level. Also, we investigate under what circumstances vector transmission is selected against pollen/seed transmission or the reverse. In particular, do climatic or latitudinal changes (longer growing seasons) select for vector or pollen/seed transmission (Jansen and Mulder, 1999; Garrett et al., 2006, 2009)? Also, does host adaptation or breeding for tolerance (lower virulence; Boots and Bowers, 1999; Jeger et al., 2006) select for vector or pollen/seed transmission?

To study the ecological and evolutionary interplay between plant virus transmission pathways, we developed a semi-discrete model taking into account vector (horizontal), seed (direct vertical) and pollen (horizontal) transmission pathways. For simplicity, we restricted the model to an annual plant population, with the rationale that in annual plants, fertilization and seed production is obligatory for population persistence. This led us to express an epidemiological invasion threshold based on the basic reproductive number of the virus, taking into account different combinations of the transmission pathways. Next we explored the circumstances under which vector transmission is selected against pollen/seed transmission or the reverse.

2. Ecological model

We focus on annual plants with indeterminate flowering. More specifically, we assume that seed germination and seedling emergence occur on a shorter time scale than vegetative plant growth, flowering and seed set/maturation. Flowering may occur at any time during the growth period, which therefore corresponds to the pollination period as well. At the end of the growth and pollination period, seeds drop, and eventually plant dies. Seeds that survive the overwintering period start a new cycle. We assume there is no seed bank.

There are three methods for viral transmission to a host plant: infected vectors, infected pollen and infected seeds. Vector acquisition of virus and inoculation of host plants may occur during the growth and pollination period. Vector transmission therefore overlaps with pollen transmission. We also assume that virus infection of the plant quickly becomes systemic. In particular, the virus is assumed to quickly spread from the vegetative tissues of the inoculated plant to the seeds.

Based on the transmission assumptions, a semi-discrete model is formulated (Mailleret and Lemesle, 2009; Fabre et al., 2012, 2015). We model the annual life cycle, t to $t+1$, in two parts, the growth and pollination period $t \to t+\tau$ and the survival and germination period $t+\tau \to t+1$. During the growth and pollination period $\tau < 1$ year, hereafter the growing season, a continuous-time model accounts for vector acquisition and inoculation of plants and pollen transmission. During the remainder of the year $1-\tau$, a discrete-time model accounts for seed survival and germination.

Let $H(t)$ and $I(t)$ denote the densities of healthy and infected plants, respectively, at time t, the beginning of the growing season. The total plant density is denoted as $T(t) = H(t) + I(t)$.

To keep the model simple, we assume the virus/vector association is non-persistent (Gray and Banerjee, 1999; Bragard et al., 2013) and leave vector dynamics implicit. Also, vector transmission is assumed to depend on the frequency of healthy plants, whereas pollen transmission is assumed to depend on the density (Appendix B; Thrall et al., 1995). Hence, the vector transmission rate per infected plant is $\beta H/T$ and the pollen transmission rate is αH. We refer to the parameter β as the vector transmission coefficient and to the parameter α as the pollen transmission coefficient.

More specifically, it is shown in Appendix B that one can express the vector transmission coefficient β as the product of five parameters: $\beta = \varepsilon \vartheta \Phi^2 U/\Lambda$, where ε is the probability that a viruliferous vector inoculates the virus to an uninfected plant, ϑ is the probability that a vector feeding on an infected plant acquires the virus, Φ^2 is the square of the vector feeding rate, U is the total vector density and finally, $1/\Lambda$ is the mean time during which transmission occurs. The parameter most subject to evolutionary pressure acting on β may be the acquisition rate ϑ since specific molecular interactions may occur between the virus and the vector receptors.

During the growing season, t to $t+\tau$, the healthy and infected plant densities are modeled as a system of differential equations with initial conditions $H(t)$ and $I(t)$,

$$\frac{dH(s)}{ds} = -\left(\alpha + \frac{\beta}{T(s)}\right) H(s) I(s), \quad \frac{dI(s)}{ds} = \left(\alpha + \frac{\beta}{T(s)}\right) H(s) I(s) \tag{1}$$

for $t \le s \le t+\tau$. Since the total plant density is constant, $T(s) = T(t)$, the healthy and infected plant densities at the end of the growing season can be easily computed (Appendix C):

$$H(t+\tau) = \frac{T(t)}{1 + \left(\frac{T(t)}{H(t)} - 1\right) \exp\left((\alpha T(t) + \beta)\tau\right)}$$
$$I(t+\tau) = T(t) - H(t+\tau). \tag{2}$$

For the remainder of the year, we model the dynamics as a simple difference equation. Let b_H and b_I denote the average number of

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seeds produced per healthy or infected plant, respectively. We assume that the virus infects both the maternal plant and the seeds. Thus, only infected plants produce infected seeds. In addition, seed production by infected plants is lower than healthy plants and on average more than one seed is produced per healthy plant,

$$b_i < b_H \quad \text{and} \quad b_H > 1.$$

If vertical transmission is perfect, all seeds produced by an infected plant are infected but if not, only a proportion p produced is infected and the remaining proportion $q = 1-p$ is not infected. The seeds that survive germinate into either healthy or infected seedlings.

We assume competition and overcrowding between neighboring seedlings reduces the total density of healthy and infected plants (Geritz et al., 1999). Density-dependent effects apply equally to healthy and infected seedlings. We apply a well-known form for plant density-dependence due to de Wit et al. (1960) (also known as Beverton–Holt density-dependence in animal populations). Therefore, the discrete-time model for the remainder of the year, $t+\tau$ to $t+1$, is

$$\begin{aligned} H(t+1) &= \frac{b_H H(t+\tau) + q b_i I(t+\tau)}{1+\lambda T(t+\tau)} \\ I(t+1) &= \frac{p b_i I(t+\tau)}{1+\lambda T(t+\tau)}, \end{aligned} \tag{3}$$

where λ is a density-dependent scaling factor.

Combining Eqs. (2) and (3), the semi-discrete model can be expressed as a difference equation for healthy and infected plants (Appendix C). Table 1 is a list of the parameters and variables for the model.

We summarize some of the dynamics of the ecological model (2) and (3). The disease-free equilibrium (DFE) value for healthy plants is

$$\overline{H} = \frac{b_H - 1}{\lambda}.$$

We define the basic reproductive number of the virus $\mathcal{R}_0$ as the number of infected seeds resulting from the introduction, at the beginning of the growing season, of an infected plant into a fully healthy population, relative to the mean number of seeds produced by a healthy plant:

$$\mathcal{R}_0 = \frac{p b_i}{b_H} \exp\left((\beta + \alpha \overline{H})\tau\right).$$

If viral transmission is purely vertical, limited only to seed transmission ($\alpha = 0 = \beta$), then it can be seen that $\mathcal{R}_0 < 1$. That is, this simple model shows that purely vertical transmission of a virus through the seed cannot maintain the virus in the host population (Fine, 1975). However, if viral transmission is either both pollen/seed-transmitted but not vector-transmitted ($\beta = 0$), or both vector/seed-transmitted but not pollen transmitted ($\alpha = 0$), then $\mathcal{R}_0$ may be greater than 1. Our annual plant model shows that pollen or vector transmission may be able to maintain the virus within the host population. But there are differences due to the transmission mechanisms. That is, pollen transmission coefficient (α) is associated with plant density $\overline{H}$ (density-dependent transmission), whereas this is not the case for vector transmission coefficient (β; frequency-dependent transmission).

Table 1
Model parameters and variables.

Variable	Definition	Parameter	Definition
t	Time in years, $t = 0, 1, 2, \ldots$	b_H	Number of seeds per healthy plant
$T(t)$	Total plant density	b_I	Number of seeds per infected plant
$H(t)$	Healthy plant density	$p = 1-q$	Seed transmission probability
$I(t)$	Infected plant density	α	Pollen transmission coefficient
		β	Vector transmission coefficient
		λ	Density-dependent scaling factor
		τ	Length of the growing season

Following Lipsitch et al. (1996), we focus on the case $p=1$ (perfect vertical transmission), and $b_i > 1$ (so that the plant population persists). An important quantity is the basic reproductive number of a healthy host introduced into a fully infected population:

$$\overline{\mathcal{R}_0} = \frac{b_H}{b_i} \underbrace{\exp\left(-\left(\alpha \frac{b_i - 1}{\lambda} + \beta\right)\tau\right)}_{\text{Prob. to escape infection}},$$

that we call the dual of $\mathcal{R}_0$. The term dual implies an invasion threshold for healthy plants to invade a population consisting entirely of infected plants. That is, $\overline{\mathcal{R}_0}$ is the expected number of healthy seeds produced by a healthy plant relative to an infected plant given that it may be infected by vector or pollen coming from a fully infected population. If both $\mathcal{R}_0 > 1$ and $\overline{\mathcal{R}_0} > 1$, then infected and healthy plants can invade each other when rare, so coexistence of healthy and infected plants is protected (Kisdi and Geritz, 2003); Fig. 1. More generally, coexistence of healthy and infected plants is possible if and only if $\mathcal{R}_0 > 1$ and $\overline{\mathcal{R}_0} > 1$. Importantly, the coexistence of healthy and infected plants is impossible without pollen transmission ($\alpha = 0$) (Appendix C).

3. Evolutionary analysis

From an evolutionary perspective, one may expect vector (frequency-dependent) transmission (β) to be selected against pollen (density-dependent) transmission (α) at low population density, and conversely (Thrall et al., 1998). However, plant population density in turn depends on the virus characteristics, which creates an eco-evolutionary feedback loop. Furthermore, polymorphism may occur because pollen transmission is density-dependent while vector transmission is frequency-dependent (Thrall and Antonovics, 1997).

To account for these phenomena, we follow an adaptive dynamics approach (Metz et al., 1992; Dieckmann and Law, 1996; Geritz et al., 1998; Dieckmann, 2004). To address the evolution of plant virus transmission pathways, the single-strain model is extended to n virus strains (Appendix D) which differ in their abilities to be seed-transmitted (b_I), pollen-transmitted (α), or vector-transmitted (β). We consider a plant population infected with $n=2$ virus strains, I_i, $i=1,2$. Let $x_1 = (\alpha_1, \beta_1, b_1)$ be the resident phenotype and let $x_2 = (\alpha_2, \beta_2, b_2)$ be the mutant phenotype. We assume the mutant initially represents a relatively small subpopulation as compared to the resident. That is, $I_2 \ll I_1$.

Following Metz et al. (1992), we are interested in testing whether the mutant can invade. In particular, if the long-run growth-rate is negative, i.e.

$$\lim_{t\to\infty} \frac{1}{t} \log\left(\frac{I_2(t)}{I_2(0)}\right) < 0, \tag{4}$$

the mutant cannot invade the resident. Assuming that the resident population with phenotype x_1 is at an ecological equilibrium corresponding to coexistence of healthy and infected plants, we define an evolutionary invasion condition (Appendix D) as

$$\log\left(\frac{I_2(1)}{I_2(0)}\right) = \underbrace{\log(b_2) - \log(b_1)}_{\text{seed transmission}} + \underbrace{(\alpha_2 - \alpha_1)\tilde{H}_1 \tau}_{\text{pollen transmission}} + \underbrace{(\beta_2 - \beta_1)\frac{\tilde{H}_1}{\hat{T}_1}\tau}_{\text{vector transmission}} > 0, \tag{5}$$

where the terms $\tilde{H}_1$ and $\hat{T}_1$ are defined as the mean healthy host and the total host density at the ecological equilibrium corresponding to the resident phenotype $x_1 = (\alpha_1, \beta_1, b_1)$, respectively.

Eq. (5) shows the relative importance of the differences between the b_i's and the α_i's or the β_i's, $i=1,2$. The difference

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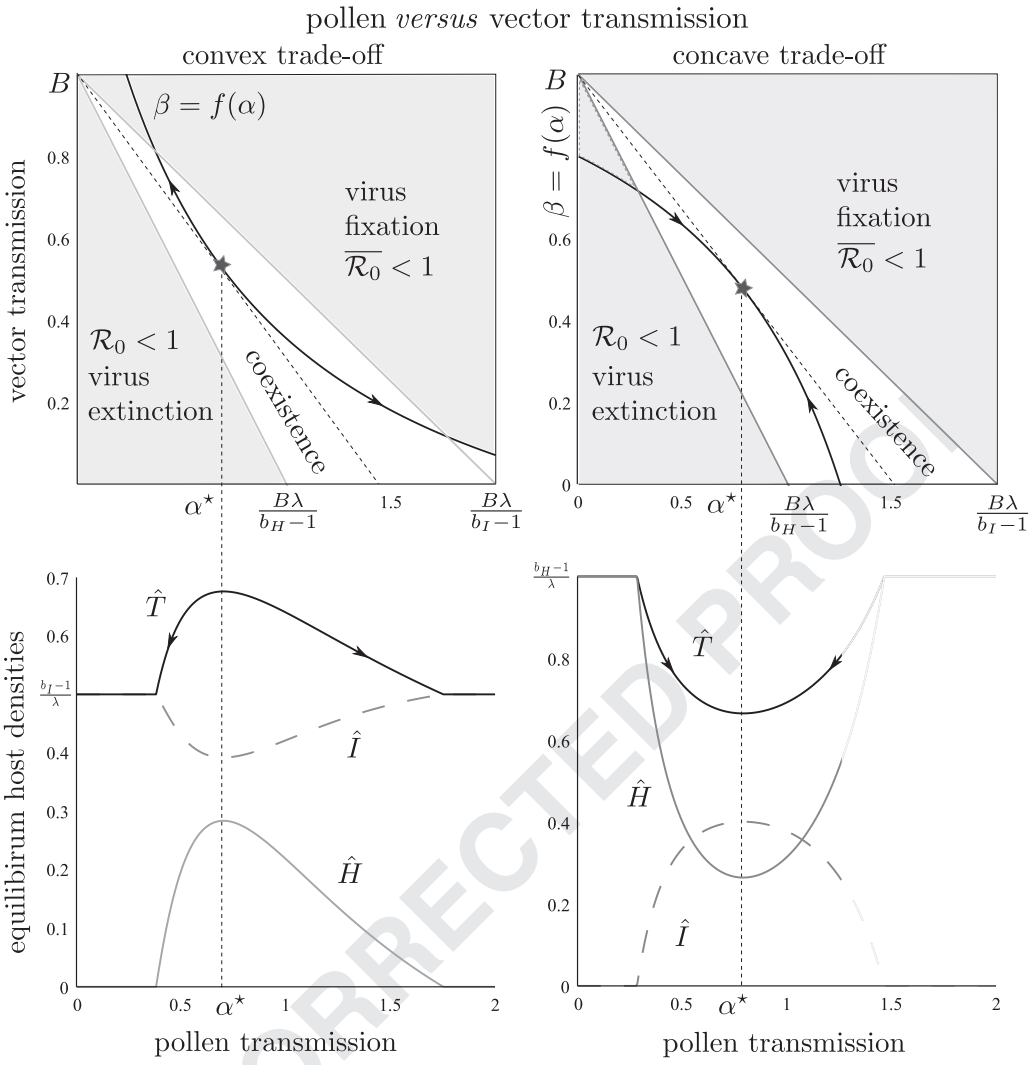

Fig. 1. *Upper row*: Evolutionary dynamics in the plane (α,β) along the trade-off curve $\beta=f(\alpha)$ (vector versus pollen transmission). The arrows represent the direction of evolution. The evolutionarily stable point indicated by a $\star$ (corresponding to $\alpha^\star$) is an evolutionary repelling point (convex trade-off) or an evolutionary endpoint (concave trade-off). It is such that the tangent of the trade-off function (dashed line) passes through the point $(0,B)$ (the upper left corner). The white triangle region corresponds to coexistence of healthy and infected plants ($\mathcal{R}_0>1$ and $\overline{\mathcal{R}_0}>1$), while the light gray regions correspond to either virus extinction ($\mathcal{R}_0<1$ and $\overline{\mathcal{R}_0}>1$), or virus fixation in the plant population ($\mathcal{R}_0>1$ and $\overline{\mathcal{R}_0}<1$). Parameter values are: $B=1$, $b_H=2$, $b_I=1.5$, $\lambda=1$ (so $\tau\approx 0.3$), and $f(\alpha)=(1-a\alpha)/(d+c\alpha)$, with $a=1/2.5$, $d=1/1.5$, and $c=1$ (convex trade-off), and $a=1/1.25$, $d=1/0.8$, $c=-0.6$ (concave trade-off). *Lower row*: Total, healthy and infected host densities at equilibrium $\hat{T}$ (solid black curves), $\hat{H}$ (solid gray curves) and $\hat{I}$ (dashed gray curves) for a monomorphic resident population, as a function of its trait α (pollen transmission), for the remaining parameter values fixed. *Lower left panel*: $\hat{T}$ lower bound corresponds to the susceptible-free equilibrium (SFE; virus fixation in the plant population), i.e. $\hat{T}=\overline{I}=(b_i-1)/\lambda=0.5$. *Lower right panel*: $\hat{T}$ upper bound corresponds to the disease-free equilibrium (DFE; absence of virus in the plant population), i.e. $\hat{T}=\overline{H}=(b_H-1)/\lambda=1$.

between the α_i's and the β_i's are respectively weighted by the mean density and the mean frequency of healthy hosts at the equilibrium shaped by the resident population. For instance, the greater the mean healthy host density at equilibrium $\tilde{H}_1$, the greater the value of pollen transmission α as compared to seed transmission b_I.

Pollen versus vector transmission: To consider a trade-off between pollen and vector transmission, we assume that seed transmission is a constant ($b_1=b_2=b_i$) and let

$$\beta_i=f(\alpha_i),\ i=1,2,$$

with f being a decreasing function ($f'(\alpha)<0$). Let the difference between healthy and infected plants intrinsic growth rates be

$$B=\frac{1}{\tau}\log\left(\frac{b_H}{b_i}\right)=\frac{\log b_H}{\tau}-\frac{\log b_i}{\tau}.$$

To make sure that the resident equilibrium corresponds to a case of coexistence between healthy and infected plants, we restrict our attention to resident α values such that $\mathcal{R}_0>1$ and $\overline{\mathcal{R}_0}>1$, i.e. to α values such that

$$B-\alpha\frac{b_H-1}{\lambda}<f(\alpha)<B-\alpha\frac{b_i-1}{\lambda}.$$

That is, we start from a (α,β) point within the triangular region formed by the three points (Fig. 1):

$$(0,B),\quad \left(\frac{B\lambda}{b_H-1},0\right),\quad \left(\frac{B\lambda}{b_i-1},0\right).$$

The area of the coexistence region between healthy and infected plants is:

$$\mathcal{A}=\frac{1}{2}B^2\lambda\left(\frac{1}{b_i-1}-\frac{1}{b_H-1}\right)=\frac{1}{2}\left[\frac{1}{\tau}\log\left(\frac{b_H}{b_i}\right)\right]^2\lambda\left(\frac{1}{b_i-1}-\frac{1}{b_H-1}\right).$$

Note that it is proportional to λ (the density dependent scaling factor for plants) and inversely related to τ (the proportion of the year corresponding to the growing season).

We use the following invasion fitness proxy, sign-equivalent to the invasion condition (5):

$$s(\alpha_1,\alpha_2)=(\alpha_2-\alpha_1)\hat{T}(\alpha_1)+f(\alpha_2)-f(\alpha_1), \tag{6}$$

with $\hat{T}(\alpha_1)=\hat{T}_1$ (the total equilibrium host density as a function of the resident trait α_1). The dynamics of $s(\alpha_1,\alpha_2)$ as a function of α_2 determine the evolutionary trajectory.

We now take advantage of the fact that we have an expression of $\hat{T}(\alpha)$ (Appendix C):

$$\hat{T}(\alpha)=\frac{B-f(\alpha)}{\alpha}. \tag{7}$$

Note that the total plant density at endemic (or healthy and infected plants coexistence) equilibrium $\hat{T}(\alpha)$ does not depend on λ (intraspecific competition among plants). Using this expression in the invasion fitness proxy (6) yields:

$$\begin{aligned} s(\alpha_1,\alpha_2)&=\frac{\alpha_2-\alpha_1}{\alpha_1}(B-f(\alpha_1))+f(\alpha_2)-f(\alpha_1),\\ &=\alpha_2\frac{B-f(\alpha_1)}{\alpha_1}-B+f(\alpha_2),\\ &=\alpha_2\hat{T}(\alpha_1)-B+f(\alpha_2). \end{aligned}$$

Since the invasion fitness proxy is both 1-dimensional and monotone in the environmental variable $\hat{T}$, there is an optimization principle (Metz et al., 2008; Gyllenberg and Service, 2011): in the pollen versus vector trade-off case, evolution minimizes total host plant density $\hat{T}$ (Fig. 1). An evolutionary singular point $\alpha^\star$ is such that

$$\hat{T}'(\alpha^\star)=-\frac{1}{\alpha^\star}\left(f'(\alpha^\star)+\hat{T}(\alpha^\star)\right)=0,$$

which can be expressed in an equivalent form as

$$B-\alpha^\star f'(\alpha^\star)=f(\alpha^\star).$$

Therefore, at an evolutionary singular point, the tangent line of the trade-off function passes through the point $(0,B)$ (Fig. 1) (Smith and Fretwell, 1974; van Baalen and Sabelis, 1995).

Also, we have

$$\hat{T}''(\alpha)=-\frac{1}{\alpha}f''(\alpha).$$

The latter equation shows that the convexity of the trade-off function $f(\alpha)$ is the opposite to that of the function $\hat{T}(\alpha)$, which determines the direction of evolution. For example, if the trade-off function is convex (concave) for all possible α values, then the total host density $\hat{T}(\alpha)$ is concave (convex) for all possible α values as well. If there exists an evolutionary singular point $\alpha^\star$, $\hat{T}(\alpha)$ has therefore a maximum (minimum), which is an evolutionary repelling (attracting) point (Fig. 1). More generally, for the trade-off between pollen and vector transmission, two evolutionary outcomes are possible.

1. If the trade-off is locally convex ($f''(\alpha^\star)>0$), then the evolutionarily singular point $\alpha^\star$ is an evolutionary repelling point.
2. If the trade-off is locally concave ($f''(\alpha^\star)<0$), then the evolutionarily singular point $\alpha^\star$ is a potential evolutionary endpoint.

As a corollary of the optimization principle, beginning from a monomorphic population, evolutionary branching (through an attracting but unstable evolutionarily singular point) and the divergence of strains specializing on pollen or vector transmission, is not possible. Also, our graphical analysis (Fig. 1) shows that evolutionary extinction (Boots and Sasaki, 2003; Parvinen, 2005) is not possible for simple convex or concave trade-off functions. More generally, evolutionary extinction through the collision of two possible equilibria (here the endemic and disease-free equilibria) cannot occur through optimizing selection (Gyllenberg and Parvinen, 2001; Parvinen and Dieckmann, 2013); but see Boldin and Kisdi (2015).

In the case of a simple convex trade-off, evolution leads to virus fixation in the plant population (Fig. 1) unless lower and upper bounds on pollen transmission coefficient prevent the dynamics from leaving the coexistence region. In this case there is evolutionary bistability: minimum pollen/maximum vector and maximum pollen/minimum vector transmission coefficients are both potential evolutionary endpoints, depending on initial conditions.

In the case of a simple concave trade-off, there is an intermediate evolutionarily stable mix between pollen and vector transmission ($\alpha^\star$). It is not possible to express $\alpha^\star$ as a function of the parameters without specifying a trade-off function. Nevertheless, our graphical analysis (Fig. 1) shows that $\alpha^\star$ does not depend on λ (intraspecific competition among plants). Also, the investment into pollen (density-dependent) versus vector (frequency-dependent) transmission coefficient $\alpha^\star$ increases with $B=\log(b_H/b_i)/\tau$. The total plant density $\hat{T}(\alpha)$ in Eq. (7) also increases with B, which favors pollen transmission. Therefore, climatic or latitudinal changes that increase the growing season length τ (Jansen and Mulder, 1999) promote vector over pollen transmission, and conversely. Also, if virulence is defined as the relative impact of infection on host plant fecundity (OKeefe and Antonovics, 2002; OKeefe, 2005), then selecting for host tolerance (decreasing virulence b_H/b_i) also selects for vector transmission over pollen transmission.

Seed versus vector transmission: A similar analysis performed for a trade-off between seed and vector transmission does not lead to the same conclusions (Appendix D). Firstly, numerical results show that evolutionary branching is possible, leading to coexistence of non-vector-borne and vector-borne virus strains (Fig. 2). Moreover, an evolutionary repelling point may prevent the early evolution of vector transmission (Fig. 2D lower left corner). Secondly, even when evolution leads to a monomorphic intermediate investment into vector transmission, the dependence of the evolutionarily stable vector transmission coefficient β^* on the parameters is dramatically changed. For instance, Fig. 3 shows that with a concave trade-off between seed and vector transmission, the vector transmission coefficient may

- decrease with increasing growing season length (τ),
- decrease with decreasing virulence (defined as b_H/b_i),
- increase with increasing intraspecific competition among plants (λ).

4. Discussion

Plant viruses have evolved various means for transmission. Focusing on annual plant hosts, some +ssRNA viruses have evolved mechanism(s) for transmission via vectors, pollen and seed (Table A3) whereas others seem incapable of being transmitted in this manner and seem transmitted only by vectors (Table A2). Yet other +ssRNA viruses have evolved mechanisms for both seed and pollen transmissibility (Table A1). Viruses vary in the modes of transmission, which is likely due to genetic differences among species and strains of virus among host species and within a host population (Johansen et al., 1994, 1996; Domier et al., 2007, 2011). Restricting our study to the case of annual plants, we addressed the questions: under what circumstances does vector transmission make pollen transmission redundant? Can evolution lead to the coexistence of multiple virus transmission pathways? How do climatic changes and host adaptation/breeding influence vector transmission?

Ecological points: The semi-discrete ecological model we developed included three modes of transmission: vector, pollen and seed. The basic reproductive number $\mathcal{R}_0$ was derived, and hence conditions for its value to be greater than one. When limited only to seed transmission the value was less than one, indicating that purely vertical transmission through seed cannot maintain the virus in the host population; this result is due to the assumption of fecundity costs

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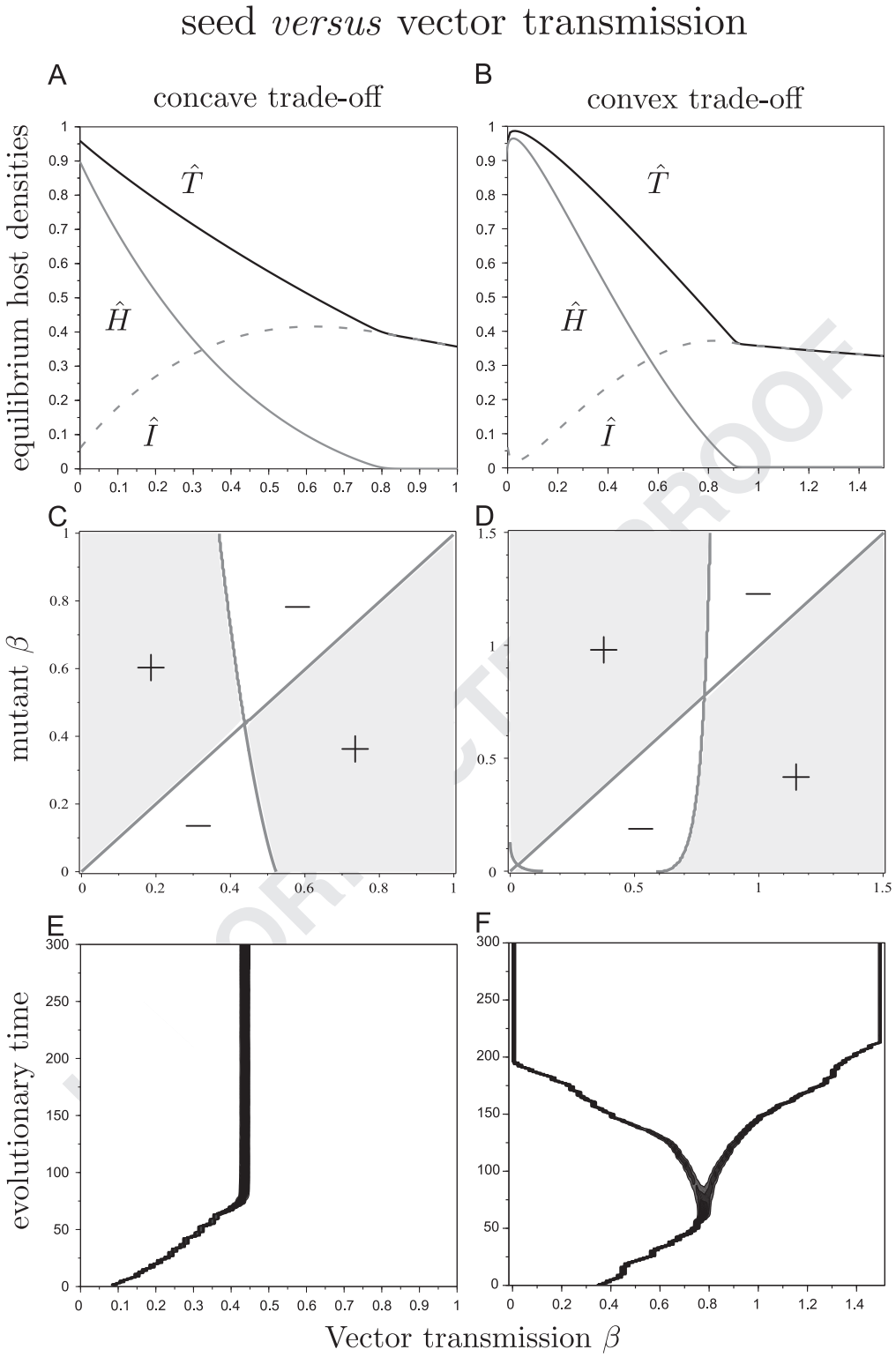

Fig. 2. (A and B) Total, healthy and infected host densities at equilibrium $\hat{T}$, $\hat{H}$ and $\hat{I}$ for a monomorphic resident population, as a function of its trait β (vector transmission; x-axis). (C and D) Pairwise Invasibility Plot (PIP) representing the sign of the invasion fitness proxy $s(\beta_1, \beta_2)$ in the plane (β_1, β_2); the resident trait β_1 is on the x-axis and the mutant trait β_2 is on the y-axis. (E and F) Evolutionary trajectories based on the multi-strain model (Appendix D) in the (β, m) plane, where m is the number of random mutations which occur at a frequency of 1/10,000 year; the y-axis is the evolutionary time. The virus population is structured on trait β, which can take $n=100$ values between 0 and 1 (E) or 0 and 1.5 (F) on the x-axis. The initial population is monomorphic with trait $\beta \approx 0.1$ (E) or $\beta \approx 0.4$ (F) and gradually evolves up to an evolutionary singular point $\beta^\star$, which is either an evolutionary endpoint (E) or a branching point (F). In (A–F), parameter values are $b_H=2$, $\alpha=1$, $\lambda=1$, and $\tau=0.3$, with trade-off function between vector (β) and seed (b_I) transmission equal to $b_i = \overline{b_i}\exp(g(\beta))$, with $g(\beta) = -0.1\beta^a$, $\overline{b_i}=1.5$ and $a=1.5$ (A, C, and E; $g(\beta)$ concave) or $a=0.5$ (B, D, and F; $g(\beta)$ convex).

associated with viral infection (Busenberg and Cooke, 1993; Lipsitch et al., 1996; Lively et al., 2005; Faeth et al., 2007). If, however, either pollen (density-dependent transmission) or vector transmission (frequency-dependent transmission) are included with seed transmission, then the virus can be maintained in the host population. Conditions were checked for the co-existence of healthy and infected

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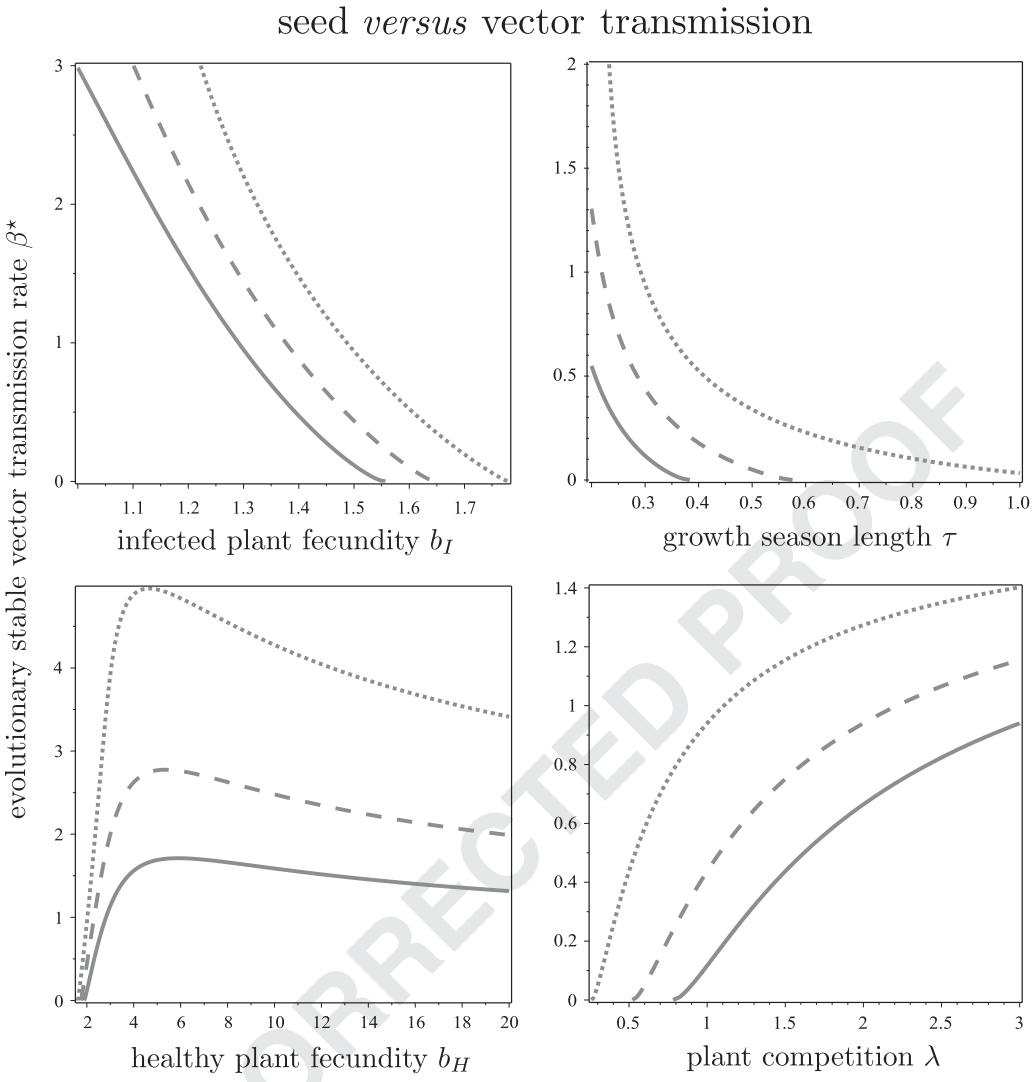

Fig. 3. The evolutionarily stable vector transmission coefficient $\beta^\star$ as a function of infected plant fecundity (b_I), healthy plant fecundity (b_H), the duration of the growing season (τ), and the density-dependent scaling factor λ. Default parameter values are b_H=2, $\alpha = 0.5, 1, 1.5$ (dotted, dashed, and solid curves, resp.), $\lambda = 1$, and $\tau = 0.3$, with concave trade-off function between vector (β) and seed (b_I) transmission equal to $b_i = \overline{b_i}\exp(g(\beta))$, with $g(\beta) = -0.1\beta^a$, $\overline{b_i} = 1.5$ and a=1.5 ($g(\beta)$ concave).

plants. The main conclusions with perfect (100%) vertical transmission in a single host population are summarized below:

(i) Pollen-seed transmission: coexistence of healthy and infected plants is possible.
(ii) Vector-seed transmission: coexistence of healthy and infected plants is not possible.

The results for vector-seed transmission (Appendix C) are consistent with continuous-time theory with vertical transmission and frequency-dependent horizontal transmission (Getz and Pickering, 1983; May et al., 1988; Thrall et al., 1995; Altizer and Augustine, 1997). With frequency-dependent horizontal transmission, the threshold for disease spread does not depend on density. Consequently, either the infection causes healthy host extinction or reproduction of healthy individuals "outstrips" disease reproduction (Thrall et al., 1993).

Evolutionary insights: The ecological model was used to explore the evolution of vector versus pollen/seed transmission. The evolutionary results are summarized in Table 2. We found that mixing vector and pollen/seed transmission may be evolutionarily stable. Besides, non-vector-borne and vector-borne variants may evolve from a single ancestral strain and coexist in the long run. The main conclusions from the evolutionary analysis depend on the particular trade-offs and are summarized below:

(i) *Pollen versus vector transmission*:
(a) *Convex trade-off* : a mix between pollen and vector transmission is evolutionarily repelling; pollen or vector transmission make the other transmission mode redundant. This situation may lead to evolutionary bistability between maximum pollen/minimum vector and minimum pollen/maximum vector transmission.
(b) *Concave trade-off* : evolution promotes a mix between pollen and vector transmission; vector transmission does not make pollen transmission redundant, and conversely. Evolution leads to coexistence of infected and healthy plants.
- climatic changes increasing the growing season length promote vector transmission over pollen transmission
- increasing tolerance (decreasing virulence) promotes vector transmission over pollen transmission

(ii) *Seed versus vector transmission*:
(a) *Convex trade-off* : evolutionary branching between seed and vector transmission is possible.
(b) *Concave trade-off* : an evolutionary stable mix between seed and vector transmission is possible.

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Table 2
Summary of evolutionary results. Depending on whether the trade-off is locally convex or concave, an evolutionarily singular point may be stable or unstable, and attractive or repulsive. In the convex case for seed *vs* vector, the evolutionary singular point may be an unstable attractor, i.e. an evolutionary branching point, or an unstable repeller (Fig. 2D). In the concave case for seed vs. vector, the evolutionary singular point may be both stable and attracting, i.e. a potential evolutionary endpoint (Fig. 2C), yet we cannot rule out the possibility that in some cases it is both stable and evolutionarily repelling, i.e. an evolutionary "Garden of Eden" Dieckmann (2004).

Trade-off	Pollen vs. vector	Seed vs. vector
Locally convex	Unstable repeller	Unstable
Locally concave	Stable attractor	Stable

In the latter case, the impacts of climatic changes or host adaptation/breeding may be at odds with those observed for a trade-off between pollen and vector transmission.

The evolutionary dynamics of pollen (density-dependent) versus vector (frequency-dependent) transmission are comparable to those previously reported by Thrall et al. (1998), who considered additional trade-offs with host mortality or fecundity. Here, we showed that an evolutionarily stable (or repelling) mix of frequency- and density-dependent transmission is possible with no additional trade-off.

Similarly, the evolutionary dynamics of seed (vertical) vs. vector (frequency-dependent horizontal) transmission are comparable to those previously reported by Bernhauerová and Berec (2015), who considered an additional trade-off with host mortality. We showed that evolutionary branching is possible with no additional trade-offs. However, this result indicates that a tripartite trade-off between plant (both pollen and seed) and animal (vector) transmission would make it possible for evolutionary branching to occur as well. We also showed that bistability may prevent early evolution of vector transmission. Altogether, our results indicate that a trade-off between vertical and horizontal frequency-dependent transmission may yield comparable evolutionary outcomes as a trade-off between vertical and density-dependent horizontal transmission (Ferdy and Godelle, 2005; van den Bosch et al., 2010).

Our results may also be interpreted in terms of virulence (defined as having a negative impact on host fitness; Froissart et al. (2010). Indeed, we associated seed (vertical) transmission with infected host fecundity due to the assumption that vertical transmission is perfect. Selecting for seed transmission (or infected plant fecundity) versus vector transmission corresponds to selecting against virulence in our study. Rephrasing our results, we found that a trade-off between virulence and vector transmission may lead to the emergence and co-existence of virulent vector-borne strains and less virulent, non-vector borne strains.

Figs. 1 and 2 show that for a trade-off between pollen and vector transmission, evolution tends to maximize infected host density and to minimize healthy host density, whereas it is not the case for a trade-off between vector and seed transmission; there is no optimization principle in this case. From an epidemiological and control perspective, our results indicate that a trade-off between pollen and vector transmission may lead to a higher prevalence of infection than a trade-off between vector and seed transmission. It is therefore important to further investigate possible trade-offs in plant viruses.

Limits and perspectives: Several restrictions were made in developing the ecological model. Foremost among them was the restriction to an annual plant species. In principle the model could easily be extended to a biennial system, where flowering and hence seed set normally occurs in the second year (e.g. some compositae have a rosette form as a first year seedling). Although seed and pollen transmission have been reported for many woody perennials (Bristow and Martin, 1999; Guerri et al., 2004; Card et al., 2007; Aramburu et al., 2010; Martin et al., 2010), investigating a perennial system would rather require a continuous-time model. The role of a seed bank could also be considered, depending on whether data are available on virus survival in seed.

Second, our annual plant model focused on non-persistent vector transmission and ignored vector migration. The latter may be important in cases where there is asynchronous planting and an available source of susceptible hosts (e.g. in the tropics). Alternatively, the virus may be acquired from wild perennials where there is a virus reservoir. Our model is a better fit for plant viruses specific to annual plants in temperate climates. Extending our study to semi-persistent or persistent vector transmission would require making vector population dynamics explicit (Appendix B). This extension would increase the model complexity, but may lead to more general results in the pollen versus vector trade-off case.

Third, our model did not account for possible Allee effects associated with pollen-limitation. That is, the observation that as total plant density decline, pollination efficiency may decline as well and the plant population may eventually go extinct. If evolution decreases total host density (as in Fig. 1), such an Allee effect might result in the evolutionary extinction of both the plant and virus populations. We leave this issue for future research.

Last, we made the common assumption that plant viruses are parasites which exploit host resources. Thus an infected host cannot be more fit than a non-infected host ($b_i < b_H$)). However, considering a virus that has neutral or positive effects on plant fecundity ($b_i \geq b_H$) together with imperfect vertical transmission ($p < 1$) (otherwise the virus trivially invades the plant population) is a promising avenue to address timely questions related to viral symbiosis evolution (Ferris et al., 1989; Michalakis et al., 1992; Xie et al., 1994; Friess and Maillet, 1996, 1997; Remold, 2002; Xu et al., 2008; Roossinck, 2011; Prendeville et al., 2014; Roossinck, 2015).

Acknowledgments

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Appendix A. Data

A list of 30 plant viruses that are positive-sense single-stranded RNA (+ssRNA), have a primary host with an annual life history, and that are transmitted both vertically (via seed or pollen) as well as horizontally, specifically via insect vectors, was made (Table A3). Compilations of vertically transmitted plant viruses, taken from Harris et al. (1980), Mink (1993), Card et al. (2007), and Sastry (2013) were verified and expanded upon based on the current literature (Table A1). Nomenclature was verified based on the International Committee of Taxonomy of Viruses

Table A1
Selected list of positive-sense single-stranded RNA plant viruses with no known insect vector. Viruses have primary hosts with an annual life history and secondary hosts with biennial or perennial life histories and different modes of pollination. Viruses are seed transmissible[a] and some viruses are pollen transmissible.

Species	Order	Family	Genus	Acronym	Life-history[b]	Pollination[c]	Seed (%)	Pollen[d]	References
Crimson clover latent virus	*Picornavirales*	*Secoviridae*	*Nepovirus*	CCLV	A	I	97		(Kenten et al., 1980)
Lucerne (Australian) latent virus				LALV	A, P	I	8–9		(Blackstock, 1978)
Foxtail mosaic virus	*Tymovirales*	*Alphaflexiviridae*	*Potexvirus*	FoMV	A	W	1–2		(Paulsen and Niblett, 1977)
Pepino mosaic virus				PepMV	A, B, P	I	0.005–1.8	No	(Córdoba-Sellés et al., 2007)
White clover mosaic virus				WClMV	A, P	I	6		(Hampton, 1963)
Potato virus T		*Betaflexiviridae*	*Tepovirus*	PVT	A, P	I	0–72	Yes	(Jones, 1982)
Melon rugose mosaic virus		*Tymoviridae*	*Tymovirus*	MRMV	A	I	0.9–3.8		(Mahgoub et al., 1997)
Pelargonium zonate spot virus	Unassigned	*Bromoviridae*	*Anulavirus*	PZSV	A, P	I	5–42	Yes	(Lapidot et al., 2010)
Humulus japonicus latent virus			*Ilarvirus*	HJLV	A, P	W	9		(Scott and Zimmerman, 2006)
Parietaria mottle virus				PMoV	A	I	36	Yes	(Aramburu et al., 2010)
Spinach latent virus				SpLV	A	W	30–95	Yes	(Bos et al., 1980)
Subterranean clover mottle virus		Unassigned	*Sobemovirus*	SCMoV	A, B, P	I	0.5–3	No	(Wroth and Jones, 1992)
Barley stripe mosaic virus		*Virgaviridae*	*Hordeivirus*	BSMV	A	W	0–100	Yes	(Carroll and Mayhew, 1976)

[a] Non-embryo-borne seed transmission is excluded; seed transmission is not a criterion to determine taxonomic assignment.
[b] Host plant life history: annual (A), biennial (B), perennial (P).
[c] Host plant pollination in alphabetical order: insect (I), self (S), wind (W).
[d] Pollen transmission: Yes, No, and blank indicates lack of supportive literature.

Table A2
Selected list of positive-sense single-stranded insect-borne plant RNA viruses with no report of seed transmissibility. Viruses have primary hosts with an annual and secondary hosts with biennial or perennial life histories and different modes of pollination.

Species	Order	Family	Genus	Acronym	Life-history[a]	Pollination[b]	Vector[c]–mode[d]	References
Ranunculus latent virus	*Picornavirales*	*Potyviridae*	*Maclurovirus*	RanLV	A, B, P	I, S	A–NP	Turina et al. (2006)
Red clover mottle virus		*Secoviridae*	*Comovirus*	RCMV	A	I,S	B–SP	Oxelfelt (1976)
Okra mosaic virus	*Tymovirales*	*Tymoviridae*	*Tymovirus*	OkMV	A	I, S	B–SP	Fajinmi and Fajinmi (2010)
Lettuce infectious yellows virus	Unassigned	*Closteroviridae*	*Crinivirus*	LALV	A, P	I	WF–SP	Brown et al. (1990)
Rice yellow mottle virus		Unassigned	*Sobemovirus*	RYMV	A	W	B–SP	Abo et al. (2000)

[a] Host plant life history: annual (A), biennial (B), perennial (P).
[b] Host plant pollination in alphabetical order: insect (I), self (S), wind (W).
[c] Vector: aphid (A), beetle (B), white fly (WF).
[d] Mode of vector transmission: non-persistent (NP), semi-persistent (SP), persistent and circulative (P-C).

(ictvonline.org/virusTaxonomy.asp). For each virus the type of insect vector, mode of vector transmission, percentage of vertical transmission, and life history of the plant host was noted. Virus biology was obtained from the current literature and the Description of Plant Viruses (http://www.dpvweb.net) with record numbers presented), whereas plant life history was determined using the US Plant Database (http://plants.usda.gov). Viruses were excluded from this summary if: (1) the primary host has a perennial life history, or (2) they are transmitted only by mites, fungi, nematodes, thrips, or by mechanical transmission. Where specific cases are directly supported by the literature they are categorized according to: plant (seed, pollen) transmission with no known vector (Table A1), animal (vector) transmission only (Table A2), and both plant (seed, pollen) and animal (vector) transmission (Table A3).

A qualification to be made is that whereas vector relationships are expected to be the same within a virus genus, there is no such expectation for seed/pollen transmission, which is not a taxonomic criterion. Also, the data presented aggregate strains of the same virus species across host species.

Evolution of seed transmission inferred from phylogeny: Vertical transmission of +ssRNA viruses occurs in eight virus families (Tables A1–A3), which suggests that vertical transmission is an ancestral trait or it has arisen multiple times. However, the phylogeny of +ssRNA virus families is a polytomy (Stuart et al., 2006), thus making it impossible to determine the evolutionary origin of seed transmission of viruses. The resolution of virus relationships is greater within families and genera and these phylogenies suggest that seed transmission has arisen multiple times (Codoñer and Elena, 2008; Gibbs and Ohshima, 2010; Thompson et al., 2014). However, since recombination is more likely in some +ssRNA virus families, e.g. *Bromoviridae* and *Potyviridae* (Chare and Holmes, 2006; Codoñer and Elena, 2008) than others, the potential exists that seed transmission was acquired through recombination. Moreover, some viral strains utilized in phylogenetic analyses have derived from "laboratory strain" where domains for vector transmission may have been altered as a result of continuous mechanical transmission by sap inoculation, e.g. Ng and Perry (1999).

Evolution of pollen/seed transmission: Whether seed/pollen transmission preceded vector transmission during the course of evolution is a challenging issue (Power, 2000). In most cases, the information available refers to extant crop viruses of recent origin, sometimes only decades to centuries old (Gibbs and Ohshima, 2010). Very little information is available on viruses in wild plant species, where the influence of agriculture in its many manifestations has not been so pronounced. Based on the phylogenetic evidence alone (Appendix A), it is not possible to conclude on the evolutionary trajectories that have led to the extant plant viruses: i.e. some seed/pollen transmitted viruses may have their origin as

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Table A3
Selected list of positive-sense single-stranded RNA plant viruses that are seed transmissible.[a] Viruses have primary hosts with an annual life history and secondary hosts with biennial or perennial life histories and different modes of pollination. Viruses are seed transmissible, some viruses are pollen transmissible and all viruses are vector-borne via different modes of transmission.

Species	Order	Family	Genus	Acronym	Life-history[b]	Pollination[c]	Seed (%)	Pollen[d]	Vector[e]–mode[f]	References
Bean pod mottle virus	*Picornavirales*	*Secoviridae*	*Comovirus*	BPMV	A	I, S	0.1		B-SP	Giesler et al. (2002)
Broad bean stain virus				BBSV	A, P	I	0.06–32		B–NP	Cockbain et al. (1976)
Cowpea mosaic virus				CPMV	A	I	0–84		B–NP, SP	Porta et al. (2003)
Cowpea severe mosaic virus				CPSMV	A, P	I	3.3–10		B–SP	Ndiaye et al. (1993)
Squash mosaic virus				SqMV	A	I	6–93		B–NP, SP	Alvarez et al. (1978)
Broad bean wilt virus 1, 2			*Fabavirus*	BBWV1, 2	A, B, P	I	0.4-0.6		A–NP	Makkouk et al. (1990)
Cowpea mild mottle virus	*Tymovirales*	*Betaflexiviridae*	*Carlavirus*	CPMMV	A	I	0.5–90		WF–NP	Jeyanandarajah and Brunt (1993)
Pea streak virus				PeSV	A, P	I	1.7		A–NP	Ford (1966)
Turnip yellow mosaic virus		*Tymoviridae*	*Tymovirus*	TYMV	A, B, P	I	2–72		B–NP	de Assis Filho and Sherwood (2000)
Alfalfa mosaic virus	Unassigned	*Bromoviridae*	*Alfamovirus*	AMV	A, P	I, W	1–100	Yes	A–NP	Hemmati et al. (1977)
Broad bean mottle virus			*Bromovirus*	BBMV	A, P	I	1.7		B	Fortass and Bos (1992)
Cucumber mosaic virus			*Cucomovirus*	CMV	A, B, P	I	1-100		A–NP	Yang et al. (1997)
Peanut stunt virus				PSV	A	I	0.2–4	No	A–NP	Troutman et al. (1967)
Bean common mosaic necrosis virus[g]		*Potyviridae*	*Potyvirus*	BCMNV	A	I, S	36–54		A–NP	Larsen et al. (2005)
Bean common mosaic virus				BCMV	A, B, P	I, S	1–94		A–NP	Morales and Castano (1987)
Bean yellow mosaic virus				BYMV	A, P	I, S	1–47		A–NP	McKirdy et al. (2000)
Cowpea aphid-borne mosaic virus				CABMV	A, B, P	I	0–35		A–NP	Konate and Neya (1996)
Lettuce mosaic virus				LMV	A, B, P	I, S	0–15	Yes	A–NP	Dinant and Lot (1992); Ryder et al. (1964)
Maize dwarf mosaic virus				MDMV	A, P	W	0.02–1.6	No	A–NP	Mikel et al. (1984)
Papaya ring spot virus				PRSV	A, P	I	15		A–NP	Gonsalves (1998)
Pea seed-borne mosaic virus				PSbMV	A	I	0–90	No	A–NP	Wang and Maule (1992)
Peanut mottle virus				PeMoV	A	I	0.02–20		A–NP	Adams et al. (1977)
Soybean mosaic virus				SMV	A	I, S	1–92		A–NP	Domier et al. (2007)
Sugarcane mosaic virus				SCMV	A, P	W	0.1–5	Yes	A–NP	Li et al. (2007)
Sunflower mosaic virus				SuMV	A	I	12		A–NP	Gulya et al. (2002)
Watermelon mosaic virus				WMV	A	I	2		A–NP	Laney et al. (2012)
Zucchini yellow mosaic virus				ZYMV	A, P	I	1–18.9		A–NP	Desbiez and Lecoq (1997)
Cowpea mottle virus		*Tombusviridae*	*Carmovirus*	CPMoV	A	I	0.4–10.3		B–SP	Allen et al. (1982)
Maize chlorotic mottle virus			*Machlomovirus*	MCMV	A	W	0–0.002		B–SP	Jensen et al. (1991)
Southern bean mosaic virus		Unassigned	*Sobemovirus*	SBMV	A, B, P	I	1–40	No	B–PC	Uyemoto et al. (1977)

[a] Non-embryo-borne seed transmission is excluded; seed transmission is not a criterion to determine taxonomic assignment.
[b] Host plant life history: annual (A), biennial (B), perennial (P).
[c] Host plant pollination in alphabetical order: insect (I), self (S), wind (W).
[d] Pollen transmission: Yes, No, and blank indicates lack of supportive literature.
[e] Vector: aphid (A), beetle (B), white fly (WF).
[f] Mode of vector transmission: non-persistent (NP), semi-persistent (SP), persistent and circulative (P–C).
[g] It may be a strain of *Bean common mosaic virus*.

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purely plant viruses, with the vector association evolving subsequently (Table A3) or not (Table A1). The obverse interpretation of Table A1 would be that these viruses have gained the ability to be seed/pollen transmitted but lost the ability to be vector-transmitted.

Appendix B. Frequency-dependent vector transmission and a simplification

Following Ross (1911) and Bacaër (2011), let $I(s)$ and $V(s)$ be the infected plant and viruliferous vector densities at time s in the growing season, respectively. For simplicity, we assume that total plant and vector densities T and U are constant during the growing season. Let Φ be the vector feeding rate, ϑ be the probability that when a vector feeds on an infected plant it acquires the virus, ε be the probability that a viruliferous vector inoculates the virus to an uninfected plant, and Λ be the rate at which the vector loses the ability to inoculate the virus. Therefore, the model takes the form:

$$\frac{dI(s)}{ds}=\Phi V(s)\frac{T-I(s)}{T}\varepsilon,\ \frac{dV(s)}{ds}=\Phi(U-V(s))\frac{I(s)}{T}\vartheta-\Lambda V(s). \tag{B.1}$$

Let

$$s^*=\Phi\varepsilon s,\quad I^*=\frac{I}{T},\quad V^*=\frac{V}{U}. \tag{B.2}$$

The dimensionless vector-plant model simplifies to

$$\frac{dI^*}{ds^*}=\frac{U}{T}V^*(1-I^*),\ \varepsilon\frac{dV^*}{ds^*}=\vartheta(1-V^*)I^*-\frac{\Lambda}{\Phi}V^*. \tag{B.3}$$

Assuming $\varepsilon\ll 1$ (the probability to inoculate the virus during a feeding event is low), we apply the quasi-steady state approximation to the second equation to yield the density of viruliferous vector V^* directly in terms of the density of infected plant I^* (Keeling and Rohani, 2008) as

$$V^*=\frac{I^*}{I^*+\dfrac{\Lambda}{\vartheta\Phi}}\approx\frac{\vartheta\Phi}{\Lambda}I^*, \tag{B.4}$$

since $\Lambda\gg\vartheta\Phi I^*$ (the virus is non-persistent). Letting $\beta=\varepsilon\vartheta\Phi^2U/\Lambda$ yields

$$\frac{dI(s)}{ds}\approx\frac{\beta}{T}I(s)(T-I(s)). \tag{B.5}$$

Appendix C. Ecological model

C.1. Dynamics of healthy and infected plants

The total plant population $T(s)$ remains constant within the growing season, $T(s)=T(t)$, $t\le s\le t+\tau$, $T(t)=H(s)+I(s)$. We can substitute $T(t)-H(s)$ for $I(s)$ which yields a single differential equation for infected plants:

$$\frac{dI(s)}{ds}=\left(\alpha+\frac{\beta}{T(t)}\right)I(s)(T(t)-I(s))=(\alpha T(t)+\beta)I(s)\left(1-\frac{I(s)}{T(t)}\right),\ t\le s\le t+\tau. \tag{C.1}$$

The preceding equation is the Verhulst logistic equation, for which an explicit solution is known. At time $t+\tau$, the solution takes the form:

$$I(t+\tau)=\frac{T(t)}{1+\left(\dfrac{T(t)}{I(t)}-1\right)\exp(-(\alpha T(t)+\beta)\tau)}. \tag{C.2}$$

Similarly,

$$H(t+\tau)=\frac{T(t)}{1+\left(\dfrac{T(t)}{H(t)}-1\right)\exp((\alpha T(t)+\beta)\tau)}. \tag{C.3}$$

For simplicity, we focus on the case $p=1-q=1$ (perfect vertical transmission), and $b_i>1$ (infected plants produce in average more than one seed, so the plant population persists). In this case, Eq. (C. 3) simplifies the semi-discrete ecological model to the following discrete-time formulation:

$$H(t+1)=\frac{b_HH(t+\tau)}{1+\lambda T(t+\tau)}=\frac{b_H}{1+\lambda T(t)}\frac{T(t)}{1+\left(\dfrac{T(t)}{H(t)}-1\right)\exp((\alpha T(t)+\beta)\tau)},$$

$$T(t+1)=\frac{b_HH(t+\tau)+b_i(T(t)-H(t+\tau))}{1+\lambda T(t+\tau)}. \tag{C.4}$$

It can be easily demonstrated that solutions are bounded and nonnegative. In addition, if $b_i>1$, the plant population persists; the total plant density is bounded below by a positive constant.

Since $b_i>1$ and $p=1$, there exists a susceptible-free equilibrium (SFE) (in addition to the disease-free equilibrium DFE) which is found by setting $H=0$ and solving for T. The SFE value for $T=I$ is

$$\bar{I}=\frac{b_i-1}{\lambda}. \tag{C.5}$$

Linearizing the difference Eq. (C. 4) for the healthy host H about the SFE, we obtain a reproductive number corresponding to a healthy host introduced into a fully infected population:

$$\overline{\mathcal{R}_0}=\frac{b_H}{b_i}\exp(-(\alpha\bar{I}+\beta)\tau). \tag{C.6}$$

The notation $\overline{\mathcal{R}_0}$ stands for the dual of $\mathcal{R}_0$. If $\overline{\mathcal{R}_0}>1$ then the SFE is unstable. If both $\mathcal{R}_0>1$ and $\overline{\mathcal{R}_0}>1$, then infected and healthy plants can invade each other when rare, so coexistence of healthy and infected plants is protected (Kisdi and Geritz, 2003). It follows that

$$\mathcal{R}_0\overline{\mathcal{R}_0}=\exp(\alpha(\overline{H}-\bar{I})\tau)=\exp\left(\alpha\left(\frac{b_H-b_i}{\lambda}\right)\tau\right)>1. \tag{C.7}$$

Therefore, the conditions $\mathcal{R}_0<1$ and $\overline{\mathcal{R}_0}<1$ are mutually exclusive. Conversely, one or both of the reproduction numbers must be greater than one. When they are both greater than one, coexistence of healthy and infected plants occurs. Moreover, in Appendix C.2 it is shown that there exists a unique endemic equilibrium (EE) if and only if $\mathcal{R}_0>1$ and $\overline{\mathcal{R}_0}>1$. Note that coexistence of healthy and infected plants is impossible in absence of pollen transmission ($\alpha=0$), since in this case, $\mathcal{R}_0\overline{\mathcal{R}_0}=1$. Based on numerical simulations, we conjecture that there are three ecologically relevant cases when $b_i>1$:

1. If $\mathcal{R}_0<1$ and $\overline{\mathcal{R}_0}>1$, then the DFE is globally stable.
2. If $\mathcal{R}_0>1$ and $\overline{\mathcal{R}_0}>1$, then the EE is globally stable.
3. If $\mathcal{R}_0>1$ and $\overline{\mathcal{R}_0}<1$, then the SFE is globally stable.

Simulations performed for $q=1-p\ll 1$ (slightly imperfect vertical transmission) showed similar results to the case $p=1$.

C.2. Endemic equilibrium existence

For model (C. 4) with $b_i>1$ and $p=1$, there exist at most three equilibria in the (H,T) plane, SFE, DFE and EE:

$$(0,\bar{I}),\quad(\overline{H},\overline{H}),\quad\text{and}\quad(\hat{H},\hat{T}), \tag{C.8}$$

with

$$\hat{T}=\frac{\log\left(\dfrac{b_H}{b_i}\right)-\beta\tau}{\alpha\tau}, \tag{C.9}$$

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and

$$\hat{H}=\hat{T}\frac{b_H}{b_H-b_i}\left(\frac{\lambda\left[\log\left(\frac{b_H}{b_i}\right)-(\alpha\bar{I}+\beta)\tau\right]}{\lambda\left[\log\left(\frac{b_H}{b_i}\right)-\beta\tau\right]+\alpha\tau}\right). \tag{C.10}$$

Feasibility of the EE requires $\hat{T}>0$. Given $\hat{T}>0$, the fraction on the right side of the preceding expression is positive if and only if $\overline{\mathcal{R}_0}>1$. But $\overline{\mathcal{R}_0}>1$ implies $\hat{T}>0$. Lastly, $\hat{H}/\hat{T}<1$ if and only if $\mathcal{R}_0>1$. Thus, a unique EE exists if and only if $\mathcal{R}_0>1$ and $\overline{\mathcal{R}_0}>1$. We emphasize this case in the evolutionary model, where each strain may be able to invade the other.

Appendix D. Evolutionary model

D.1. Multi-strain dynamics

We extend the single-strain model (C. 4) to n virus strains, I_i, with traits (α_i,β_i,b_i), $i=1,\ldots,n$. Let the total host population density be $T=H+\sum_{i=1}^{n}I_i$. We assume there is no co-infection of the same plant by two viral strains. During the growing season, the model is

$$\frac{dI_i}{ds}=\left(\alpha_i+\frac{\beta_i}{T(t)}\right)I_i(s)H(s),\ t\le s\le t+\tau, \tag{D.1}$$

for $i=1,\ldots,n$, where $H(s)=T(t)-\sum_{i=1}^{n}I_i(s)$. For $n\ge 2$ strains, an explicit solution in terms of the state variables is not possible. Therefore, unlike the single-strain model, the multi-strain model does not simplify to a difference equation.

We now reintroduce the parameter $p=1-q$, accounting for possibly imperfect vertical transmission, to later stress that the results arising from the evolutionary invasion analysis hold regardless of the perfect vertical transmission assumption ($p=1$, $q=0$).

During the remainder of the year, $t+\tau$ to $t+1$, the model is

$$H(t+1)=\frac{b_H H(t+\tau)+\sum_{i=1}^{n}qb_iI_i(t+\tau)}{1+\lambda T(t)},$$
$$I_i(t+1)=\frac{pb_iI_i(t+\tau)}{1+\lambda T(t)} \tag{D.2}$$

for $i=1,\ldots,n$. The annual cycle (D.1)–(D.2) repeats.

D.2. Evolutionary invasion analysis

We assume that a mutant strain I_2 challenges a resident strain I_1, whose dynamics cycle from year to year, denoted as $I_1^{\circ}(\cdot)$ in the absence of the mutant. The mutant strain is assumed to be initially rare ($I_2(0)\ll I_1(0)$) and to have virtually no impact on the resident dynamics $I_1^{\circ}(\cdot)$ during the first year. Also, let $T^{\circ}(\cdot)$ and $H^{\circ}(\cdot)$ denote the total and healthy plant densities, respectively, that are initially shaped by the resident strain year-to-year equilibrium. That is, $T^{\circ}(0)=\hat{T}_1>0$ and $H^{\circ}(0)=\hat{H}_1$ (Appendix B; the subscript 1 stresses that the equilibrium is shaped by the resident strain I_1). It follows from Eq. (D.1) with $n=2$ strains,

$$\frac{dI_2(s)}{ds}\approx\left(\alpha_2+\frac{\beta_2}{\hat{T}_1}\right)I_2(s)H^{\circ}(s),\ 0\le s\le\tau. \tag{D.3}$$

Denote the mean density of the healthy host at the resident equilibrium as

$$\tilde{H}_1=\frac{1}{\tau}\int_0^{\tau}H^{\circ}(s)\,ds, \tag{D.4}$$

then for $H^{\circ}(s)\approx\tilde{H}_1$ and $T^{\circ}(s)\approx\hat{T}_1$ during the first growing season yields

$$I_2(\tau)\approx I_2(0)\exp\left(\left(\alpha_2+\frac{\beta_2}{\hat{T}_1}\right)\tilde{H}_1\tau\right). \tag{D.5}$$

It follows from Eq. (D.2) with $n=2$ strains,

$$I_2(1)=\frac{pb_2I_2(\tau)}{1+\lambda\hat{T}_1}. \tag{D.6}$$

Thus, applying (D.5) and (D.6), we obtain the following invasion condition:

$$\frac{I_2(1)}{I_2(0)}\approx\frac{pb_2\exp\left(\left(\alpha_2+\frac{\beta_2}{\hat{T}_1}\right)\tilde{H}_1\tau\right)}{1+\lambda\hat{T}_1}>1. \tag{D.7}$$

Using the fact that the resident population I_1 is at an ecological equilibrium, i.e.

$$\frac{I_1(1)}{I_1(0)}\approx\frac{pb_1\exp\left(\left(\alpha_1+\frac{\beta_1}{\hat{T}_1}\right)\tilde{H}_1\tau\right)}{1+\lambda\hat{T}_1}=1, \tag{D.8}$$

simplifies the invasion condition:

$$\frac{I_2(1)}{I_2(0)}\approx\frac{pb_2\exp\left(\left(\alpha_2+\frac{\beta_2}{\hat{T}_1}\right)\tilde{H}_1\tau\right)}{pb_1\exp\left(\left(\alpha_1+\frac{\beta_1}{\hat{T}_1}\right)\tilde{H}_1\tau\right)}. \tag{D.9}$$

Hence, the mutant invasion fitness equals

$$\log\left(\frac{I_2(1)}{I_2(0)}\right)=\log\left(\frac{b_2}{b_1}\right)+\left[(\alpha_2-\alpha_1)+\frac{(\beta_2-\beta_1)}{\hat{T}_1}\right]\tilde{H}_1\tau. \tag{D.10}$$

Notice that the mutant invasion fitness does not depend directly on p, as imperfect transmission impacts every strain equally. However, $\hat{T}_1$ and $\tilde{H}_1$ may depend on p.

D.3. Seed versus vector transmission

To consider a trade-off between seed and vector transmission, we assume pollen transmission is constant ($\alpha_1=\alpha_2=\alpha$) and let

$$b_i=\overline{b_i}\,\exp(g(\beta_i)),\ i=1,2, \tag{D.11}$$

where $\overline{b_i}<b_H$ is the biologically feasible maximum number of seeds per infected plant. Also, $g(\beta)$ is negative and decreasing for all β, i.e., $g(\beta)<0$ and $g'(\beta)<0$. We define a new function as an invasion fitness proxy, sign-equivalent to the invasion fitness function (D.10):

$$s(\beta_1,\beta_2)=g(\beta_2)-g(\beta_1)+(\beta_2-\beta_1)E(\beta_1), \tag{D.12}$$

where $E(\beta)=\tau\tilde{H}(\beta)/\hat{T}(\beta)$, with $\tilde{H}(\beta_1)=\tilde{H}_1$ and $\hat{T}(\beta_1)=\hat{T}_1$ (the mean healthy host and total host equilibrium densities as a function of the resident trait β_1).

The selection gradient $G(\beta)$ is defined as the partial derivative of $s(\beta_1,\beta_2)$ with respect to its second argument, evaluated at $\beta_1=\beta_2=\beta$. An evolutionary singular point $\beta^{\star}$ is such that the selection gradient is zero:

$$G(\beta^{\star})=\frac{\partial s}{\partial\beta_2}(\beta^{\star},\beta^{\star})=g'(\beta^{\star})+E(\beta^{\star})=0. \tag{D.13}$$

Whether $\beta^{\star}$ is evolutionarily stable is determined by the sign of the second derivative of s with respect to β_2, evaluated at $\beta_1=\beta_2=\beta^{\star}$. Since

$$\frac{\partial^2 s}{\partial\beta_2^2}(\beta^{\star},\beta^{\star})=g''(\beta^{\star}), \tag{D.14}$$

whether the (log)-trade-off function g is concave or convex completely determines whether a singular point is evolutionarily stable or not, respectively.

In addition, whether $\beta^{\star}$ is evolutionarily attracting is determined by the sign of the derivative of the selection gradient with

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respect to β, evaluated at $\beta^\star$:

$$G'(\beta^\star) = g''(\beta^\star) + E'(\beta^\star). \tag{D.15}$$

A formula for $E(\beta)$ can be expressed in terms of $\hat{T}(\beta)$. It follows from definition (D.4) that

$$\begin{aligned}\tau\tilde{H}(\beta) &= \int_0^\tau H^\circ(s)\mathrm{d}s,\\ &= \hat{T}(\beta)\left[\frac{1}{\alpha\hat{T}(\beta)+\beta}\log\left(\frac{1}{H^\circ(0)}\frac{\hat{T}(\beta)}{1+\left(\frac{\hat{T}(\beta)}{H^\circ(0)}-1\right)\exp\left((\alpha\hat{T}(\beta)+\beta)\tau\right)}\right)+\tau\right],\\ &= \hat{T}(\beta)\left[\frac{1}{\alpha\hat{T}(\beta)+\beta}\log\left(\frac{H^\circ(\tau)}{H^\circ(0)}\right)+\tau\right].\end{aligned} \tag{D.16}$$

At equilibrium,

$$H^\circ(0) = \frac{b_H H^\circ(\tau)}{1+\lambda\hat{T}(\beta)}. \tag{D.17}$$

Thus,

$$E(\beta) = \frac{\tau\tilde{H}(\beta)}{\hat{T}(\beta)} = \frac{1}{\alpha\hat{T}(\beta)+\beta}\log\left(\frac{1+\lambda\hat{T}(\beta)}{b_H}\right)+\tau. \tag{D.18}$$

However, the preceding formula for E still does not make condition (D.15) easily amenable to analysis. Therefore, the trade-off between seed and vector transmission is explored through numerical simulations.

D.4. Numerical simulations

Evolutionary computations in Fig. 2 were realized from the multi-strain model ((D.1) and (D.2)) using the following algorithm. The evolving phenotype β ranges from 0 to the biologically feasible maximum β_M. The interval $[0,\beta_M]$ is divided into a finite number of subintervals (here 100), each with length $\Delta\beta$. The evolutionary dynamics are governed by the following iteration scheme. The scheme is initiated with a given value of β equal to one of the endpoints of the subintervals. Next, the ecological equilibrium is computed from the multi-strain model (here after a fixed time horizon of 10,000 years), then a small mutation $\pm\Delta\beta$ occurs in β with equal likelihood of being smaller or larger than β. Time is advanced by one unit in evolutionary time (10,000 years) and β is changed to either $\beta+\Delta\beta$ or $\beta-\Delta\beta$. The evolutionary process continues with this new β value.

Simulations performed for $q=1-p\ll 1$ (slightly imperfect vertical transmission) showed similar results (including evolutionary branching) to the case $p=1$.

References

Abo, M., Alegbejo, M., Sy, A., Misari, S., 2000. An overview of the mode of transmission, host plants and methods of detection of *Rice yellow mottle virus*. J. Sustainable Agric. 17, 19–36.

Adams, D., Kuhn, C., et al., 1977. Seed transmission of *Peanut mottle virus* in peanuts. Phytopathology 67, 1126–1129.

Allen, D., Thottappilly, G., Rossel, H., 1982. *Cowpea mottle virus*: field resistance and seed transmission in virus-tolerant cowpea. Ann. Appl. Biol. 100, 331–336.

Altizer, S.M., Augustine, D.J., 1997. Interactions between frequency-dependent and vertical transmission in host–parasite systems. Proc. R. Soc. Biol. Sci. Ser. B 264, 807–814.

Alvarez, M., Campbell, R., et al., 1978. Transmission and distribution of *Squash mosaic virus* in seeds of cantaloupe. Phytopathology 68, 257–263.

Aramburu, J.L., Galipienso, L., Aparicio, F., Soler, S., Lopez, C., 2010. Mode of transmission of *Parietaria mottle virus*. J. Plant Pathol. 92, 679–684.

de Assis Filho, F.M., Sherwood, J.L., 2000. Evaluation of seed transmission of *Turnip yellow mosaic virus* and *Tobacco mosaic virus* in *Arabidopsis thaliana*. Virology 90, 1233–1238.

van Baalen, M., Sabelis, M.W., 1995. The dynamics of multiple infection and the evolution of virulence. Am. Nat. 146, 881–910.

Bacaër, N., 2011. A Short History of Mathematical Population Dynamics. Springer Science & Business Media.

Bernhauerová, V., Berec, L., 2015. Role of trade-off between sexual and vertical routes for evolution of pathogen transmission. Theor. Ecol. 8, 23–26.

Blackstock, J., 1978. *Lucerne transient streak* and *Lucerne latent*, two new viruses of lucerne. Crop Pasture Sci. 29, 291–304.

Blanc, S., Uzest, M., Drucker, M., 2011. New research horizons in vector-transmission of plant viruses. Curr. Opin. Microbiol. 14, 483–491.

Boldin, B., Kisdi, É, 2015. Evolutionary suicide through a non-catastrophic bifurcation: adaptive dynamics of pathogens with frequency-dependent transmission. J. Math. Biol., 1–24.

Boots, M., Bowers, R.G., 1999. Three mechanisms of host resistance to microparasites—avoidance, recovery and tolerance—show different evolutionary dynamics. J. Theor. Biol. 201, 13–23.

Boots, M., Sasaki, A., 2003. Parasite evolution and extinctions. Ecol. Lett. 6, 176–182.

Bos, L., Huttinga, H., Maat, D., 1980. *Spinach latent virus*, a new *Ilarvirus* seed-borne in *Spinacia oleracea*. Neth. J. Plant Pathol. 86, 79–98.

van den Bosch, F., Fraaije, B.A., van den Berg, F., Shaw, M.W., 2010. Evolutionary bistability in pathogen transmission mode. Proc. R. Soc. Biol. Sci. Ser. B 277, 1735–1742.

Bragard, C., Caciagli, P., Lemaire, O., Lopez-Moya, J., MacFarlane, S., Peters, D., Susi, P., Torrance, L., 2013. Status and prospects of plant virus control through interference with vector transmission. Annu. Rev. Phytopathol. 51, 177–201.

Bristow, P., Martin, R., 1999. Transmission and the role of honeybees in field spread of *Blueberry shock ilarvirus*, a pollen-borne virus of highbush blueberry. Phytopathology 89, 124–130.

Brown, L., Brown, J., Tsai, J., et al., 1990. *Lettuce infectious yellows virus*. Plant Pathol. Circ. (Gainesville).

Busenberg, S., Cooke K. 1993. Vertically transmitted diseases, models and dynamics. Biomathematics, vol. 23, Springer-Verlag, Berlin.

Card, S., Pearson, M., Clover, G., 2007. Plant pathogens transmitted by pollen. Austral. Plant Pathol. 36, 455–461.

Carroll, T.W., 1972. Seed transmissibility of two strains of *Barley stripe mosaic virus*. Virology 48, 323–336.

Carroll, T.W., Mayhew, D.E., 1976. Anther and pollen infection in relation to the pollen and seed transmissibility of two strains of *Barley stripe mosaic virus* in barley. Can. J. Bot. 54, 1604–1621.

Chare, E.R., Holmes, E.C., 2006. A phylogenetic survey of recombination frequency in plant RNA viruses. Arch. Virol. 151, 933–946.

Cockbain, A., Bowen, R., Vorra-Urai, S., 1976. Seed transmission of *Broad bean stain virus* and *Echtes Ackerbohnenmosaik-Virus* in field beans (*Viciafaba*). Ann. Appl. Biol. 84, 321–332.

Codoñer, F.F., Elena, S.F., 2008. The promiscuous evolutionary history of the family *Bromoviridae*. J. Gen. Virol. 89, 1739–1747.

Córdoba-Sellés, M.d.C., García-Rández, A., Alfaro-Fernández, A., Jordá-Gutiérrez, C., 2007. Seed transmission of *Pepino mosaic virus* and efficacy of tomato seed disinfection treatments. Plant Dis. 91, 1250–1254.

Desbiez, C., Lecoq, H., 1997. *Zucchini yellow mosaic virus*. Plant Pathol. 46, 809–829.

Dieckmann, U., Law, R., 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. J. Math. Biol. 34, 579–612.

Diekmann, O., 2004. A beginners guide to adaptive dynamics, vol. 63. Banach Center Publ., pp. 47–86.

Dinant, S., Lot, H., 1992. *Lettuce mosaic virus*. Plant Pathol. 41, 528–542.

Domier, L.L., Hobbs, H.A., McCoppin, N.K., Bowen, C.R., Steinlage, T.A., Chang, S., Wang, Y., Hartman, G.L., 2011. Multiple loci condition seed transmission of *Soybean mosaic virus* (SMV) and SMV-induced seed coat mottling in soybean. Phytopathology 101, 750–756.

Domier, L.L., Steinlage, T.A., Hobbs, H.A., Wang, Y., Herrera-Rodriguez, G., Haudenshield, J.S., McCoppin, N.K., Hartman, G.L., 2007. Similarities in seed and aphid transmission among *Soybean mosaic virus* isolates. Plant Dis. 91, 546–550.

Escriu, F., Perry, K.L., García-Arenal, F., 2000. Transmissibility of cucumber mosaic virus by aphis gossypii correlates with viral accumulation and is affected by the presence of its satellite rna. Phytopathology 90, 1068–1072.

Evans, I., Zettler, F., et al., 1970. Aphid and mechanical transmission properties of *Bean yellow mosaic virus* isolates. Phytopathology 60, 1170–1174.

Fabre, F., Rousseau, E., Mailleret, L., Moury, B., 2012. Durable strategies to deploy plant resistance in agricultural landscapes. New Phytol. 193, 1064–1075.

Fabre, F., Rousseau, E., Mailleret, L., Moury, B., 2015. Epidemiological and evolutionary management of plant resistance: optimizing the deployment of cultivar mixtures in time and space in agricultural landscapes. Evol. Appl.

Faeth, S.H., Hadeler, K.P., Thieme, H.R., 2007. An apparent paradox of horizontal and vertical disease transmission. J. Biol. Dyn. 1, 45–62.

Fajinmi, A., Fajinmi, O., 2010. Incidence of *Okra mosaic virus* at different growth stages of okra plants (*Abelmoschus esculentus* (L.) *Moench*) under tropical condition. J. Gen. Mol. Virol. 2, 28–31.

Ferdy, J.-B., Godelle, B., 2005. Diversification of transmission modes and the evolution of mutualism. Am. Nat. 166, 613–627.

Ferris, M.A., Castello, J.D., Sinclair, W.A., et al., 1989. Effects of virus and mycoplasma like organism infection on green and white ash. Phytopathology 79, 579–583.

Fine, P., 1975. Vectors and vertical transmission: an epidemiological perspective. Ann. N. Y. Acad. Sci. 266, 173–194.

Ford, R., 1966. Recovery of *Pea streak virus* from pea seed parts and its transmission by immature seed. Phytopathology 56, 858.

Fortass, M., Bos, L., 1992. *Broad bean mottle virus* in Morocco; variability, interaction with food legume species, and seed transmission in faba bean, pea, and chickpea. Neth. J. Plant Pathol. 98, 329–342.

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Friess, N., Maillet, J., 1996. Influence of *Cucumber mosaic virus* infection on the intraspecific competitive ability and fitness of purslane (*Portulaca oleracea*). New Phytol. 132, 103–111.

Friess, N., Maillet, J., 1997. Influence of *Cucumber mosaic* virus infection on the competitive ability and reproduction of chickweed (*Stellaria media*). New Phytol. 135, 667–674.

Froissart, R., Doumayrou, J., Vuillaume, F., Alizon, S., Michalakis, Y., 2010. The virulence-transmission trade-off in vector-borne plant viruses: a review of (non-) existing studies. Philos. Trans. R. Soc. Lond. B Biol. Sci. 365, 1907–1918.

Garrett, K., Dendy, S., Frank, E., Rouse, M., Travers, S., 2006. Climate change effects on plant disease: genomes to ecosystems. Annu. Rev. Phytopathol. 44, 489–509.

Garrett, K., Zúñiga, L., Roncal, E., Forbes, G., Mundt, C., Su, Z., Nelson, R., 2009. Intraspecific functional diversity in hosts and its effect on disease risk across a climatic gradient. Ecol. Appl. 19, 1868–1883.

Geritz, S.A., van der Meijden, E., Metz, J.A., 1999. Evolutionary dynamics of seed size and seedling competitive ability. Theoret. Popul. Biol. 55, 324–343.

Geritz, S.A.H., Mesze, G., Metz, J.A.J., et al., 1998. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. Evol. Ecol. 12, 35–57.

Getz, W.M., Pickering, J., 1983. Epidemic models: thresholds and population regulation. Am. Nat. 121, 892–898.

Gibbs, A., Ohshima, K., 2010. Potyviruses and the digital revolution. Annu. Rev. Phytopathol. 48, 205–223.

Giesler, L.J., Ghabrial, S.A., Hunt, T.E., Hill, J.H., 2002. *Bean pod mottle virus*: a threat to us soybean production. Plant Dis. 86, 1280–1289.

Gonsalves, D., 1998. Control of *Papaya ringspot virus* in papaya: a case study. Annu. Rev. Phytopathol. 36, 415–437.

Gray, S.M., Banerjee, N., 1999. Mechanisms of arthropod transmission of plant and animal viruses. Microbiol. Mol. Biol. Rev. 63, 128–148.

Guerri, J., Pina, J., Vives, M., Navarro, L., Moreno, P., 2004. Seed transmission of *Citrus leaf botch virus*: implications in quarantine and certification programs. Plant Dis. 88 906–906.

Gulya, T., Shiel, P., Freeman, T., Jordan, R., Isakeit, T., Berger, P., 2002. Host range and characterization of *Sunflower mosaic virus*. Phytopathology 92, 694–702.

Gyllenberg, M., Parvinen, K., 2001. Necessary and sufficient conditions for evolutionary suicide. Bull. Math. Biol. 63, 981–993.

Gyllenberg, M., Service, R., 2011. Necessary and sufficient conditions for the existence of an optimisation principle in evolution. J. Math. Biol. 62, 359–369.

Hampton, R., 1963. Seed transmission of *White clover mosaic* and *Clover yellow mosaic* viruses in red clover. Phytopathology 53, 1139.

Harris, K.F., Maramorosch, K., et al., 1980. Vectors of Plant Pathogens. Academic Press, Inc..

Hemmati, K., McLean, D., et al., 1977. Gamete-seed transmission of *Alfalfa mosaic virus* and its effect on seed germination and yield in alfalfa plants. Phytopathology 67, 576–579.

Hull, R., 2014. Plant Virology, fifth ed. Elsevier, New York.

Jansen, V., Mulder, G., 1999. Evolving biodiversity. Ecol. Lett. 2, 379–386.

Jeger, M., Seal, S., Van den Bosch, F., 2006. Evolutionary epidemiology of plant virus disease. Adv. Virus Res. 67, 163–203.

Jensen, S., Wysong, D., Ball, E., Higley, P., et al., 1991. Seed transmission of *Maize chlorotic mottle virus*. Plant Dis. 75, 497–498.

Jeyanandarajah, P., Brunt, A.A., 1993. The natural occurrence, transmission, properties and possible affinities of *Cowpea mild mottle virus*. J. Phytopathol. 137, 148–156.

Johansen, E., Edwards, M.C., Hampton, R.O., 1994. Seed transmission of viruses: current perspectives. Annu. Rev. Phytopathol. 32, 363–386.

Johansen, I., Dougherty, W., Keller, K., Wang, D., Hampton, R., 1996. Multiple viral determinants affect seed transmission of *Pea seedborne mosaic virus* in *Pisum sativum*. J. Gen. Virol. 77, 3149–3154.

Jones, R., 1982. Tests for transmission of four potato viruses through potato true seed. Ann. Appl. Biol. 100, 315–320.

Jossey, S., Hobbs, H.A., Domier, L.L., 2013. Role of *Soybean mosaic virus*-encoded proteins in seed and aphid transmission in soybean. Phytopathology 103, 941–948.

Keeling, M.J., Rohani, P., 2008. Modeling Infectious Diseases in Humans and Animals. Princeton University Press.

Kenten, R., Cockbain, A., Woods, R., 1980. *Crimson clover latent virus*—a newly recognised seed-borne virus infecting crimson clover (*Trigolium incarnatum*). Ann. Appl. Biol. 96, 79–85.

Kisdi, E., Geritz, S.A., 2003. On the coexistence of perennial plants by the competition-colonization trade-off. Am. Nat. 161, 350–354.

Konate, G., Neya, B., 1996. Rapid detection of *Cowpea aphid-borne mosaic virus* in cowpea seeds. Ann. Appl. Biol. 129, 261–266.

Laney, A.G., Avanzato, M.V., Tzanetakis, I.E., 2012. High incidence of seed transmission of *Papaya ringspot virus* and *Watermelon mosaic virus*, two viruses newly identified in *Robinia pseudoacacia*. Eur. J. Plant Pathol. 134, 227–230.

Lapidot, M., Guenoune-Gelbart, D., Leibman, D., Holdengreber, V., Davidovitz, M., Machbash, Z., Klieman-Shoval, S., Cohen, S.S., Gal-On, A., 2010. *Pelargonium zonate spot virus* is transmitted vertically via seed and pollen in tomato. Phytopathology 100, 798–804.

Larsen, R.C., Miklas, P.N., Druffel, K.L., Wyatt, S.D., 2005. Nl-3 k strain is a stable and naturally occurring interspecific recombinant derived from *Bean common mosaic necrosis virus* and *Bean common mosaic virus*. Phytopathology 95, 1037–1042.

Li, L., Wang, X., Zhou, G., 2007. Analyses of maize embryo invasion by *Sugarcane mosaic virus*. Plant Sci. 172, 131–138.

Lipsitch, M., Siller, S., Nowak, M.A., 1996. The evolution of virulence in pathogens with vertical and horizontal transmission. Evolution 50, 1729–1741.

Lively, C.M., Clay, K., Wade, M.J., Fuqua, C., 2005. Competitive co-existence of vertically and horizontally transmitted parasites. Evol. Ecol. Res. 7, 1183.

Mahgoub, H.A., Wipf-Scheibel, C., Delécolle, B., Pitrat, M., Dafalla, G., Lecoq, H., 1997. *Melon rugose mosaic virus*: characterization of an isolate from sudan and seed transmission in melon. Plant Dis. 81, 656–660.

Mailleret, L., Lemesle, V., 2009. A note on semi-discrete modelling in the life sciences. Phil. Trans. R. Soc. A 367, 4779–4799.

Makkouk, K., Kumari, S., Bos, L., 1990. *Broad bean wilt virus*: host range, purification, serology, transmission characteristics, and occurrence in faba bean in West Asia and North Africa. Neth. J. Plant Pathol. 96, 291–300.

Martin, R.R., Zhou, J., Tzanetakis, I.E., 2010. *Blueberry latent virus*: an amalgam of the *Partitiviridae* and *Totiviridae*. Virus Res. 155, 175–180.

May, R.M., Anderson, R.M., McLean, A.R., 1988. Possible demographic consequences of HIV/AIDS epidemics. I. Assuming HIV infection always leads to AIDS. Math. Biosci. 90, 475–505.

McKirdy, S., Jones, R., Latham, L., Coutts, B., 2000. *Bean yellow mosaic potyvirus* infection of alternative annual pasture, forage, and cool season crop legumes: susceptibility, sensitivity, and seed transmission. Crop Pasture Sci. 51, 325–346.

Metz, J., Mylius, S., Diekmann, O., 2008. When does evolution optimize? Evol. Ecol. Res. 10, 629–654.

Metz, J.A.J., Nisbet, R., Geritz, S., 1992. How should we define fitness for general ecological scenarios? Trends Ecol. Evol. 7, 198–202.

Michalakis, Y., Olivieri, I., Renaud, F., Raymond, M., 1992. Pleiotropic action of parasites: how to be good for the host. Trends Ecol. Evol. 7, 59–62.

Mikel, M., D'Arcy, C.J., Ford, R., 1984. Seed transmission of *Maize dwarf mosaic virus* in sweet corn. J. Phytopathol. 110, 185–191.

Mink, G.I., 1993. Pollen- and seed-transmitted viruses and viroids. Annu. Rev. Phytopathol. 31, 375–402.

Morales, F., Castano, M., 1987. Seed transmission characteristics of selected bean common mosaic virus strains in differential bean cultivars. Plant Dis. 71, 51–53.

Ndiaye, M., Bashir, M., Keller, K., Hampton, R., et al., 1993. Cowpea viruses in senegal, West Africa: identification, distribution, seed transmission and sources of genetic resistance. Plant Dis. 77, 999–1003.

Ng, J., Perry, K.L., 1999. Stability of the aphid transmission phenotype in *Cucumber mosaic virus*. Plant Pathol. 48, 388–394.

Ng, J.C., Falk, B.W., 2006. Virus-vector interactions mediating nonpersistent and semipersistent transmission of plant viruses. Annu. Rev. Phytopathol. 44, 183–212.

Ng, J.C., Perry, K.L., 2004. Transmission of plant viruses by aphid vectors. Mol. Plant Pathol. 5, 505–511.

O'Keefe, K.J., 2005. The evolution of virulence in pathogens with frequency-dependent transmission. J. Theor. Biol. 233, 55–64.

O'Keefe, K.J., Antonovics, J., 2002. Playing by different rules: the evolution of virulence in sterilizing pathogens. Am. Nat. 159, 597–605.

Oxelfelt, P., 1976. Biological and physicochemical characteristics of three strains of *Red clover mottle virus*. Virology 74, 73–80.

Pagán, I., Montes, N., Milgroom, M.G., García-Arenal, F., 2014. Vertical transmission selects for reduced virulence in a plant virus and for increased resistance in the host. PLoS Pathog. 10, e1004293.

Parvinen, K., 2005. Evolutionary suicide. Acta Biotheor. 53, 241–264.

Parvinen, K., Dieckmann, U., 2013. Self-extinction through optimizing selection. J. Theor. Biol. 333, 1–9.

Paulsen, A., Niblett, C., 1977. Purification and properties of *Foxtail mosaic virus*. Phytopathology 67 346-1.

Pirone, T.P., Blanc, S., 1996. Helper-dependent vector transmission of plant viruses. Annu. Rev. Phytopathol. 34, 227–247.

Porta, C., Spall, V.E., Findlay, K.C., Gergerich, R.C., Farrance, C.E., Lomonossoff, G.P., 2003. *Cowpea mosaic virus-based chimaeras*: effects of inserted peptides on the phenotype, host range, and transmissibility of the modified viruses. Virology 310, 50–63.

Power, A.G., 2000. Insect transmission of plant viruses: a constraint on virus variability. Curr. Opin. Plant Biol. 3, 336–340.

Prendeville, H.R., Tenhumberg, B., Pilson, D., 2014. Effects of virus on plant fecundity and population dynamics. New Phytol. 202, 1346–1356.

Remold, S.K., 2002. Unapparent virus infection and host fitness in three weedy grass species. J. Ecol. 90, 967–977.

Revers, F., García, J.A., 2015. Molecular biology of potyviruses. Adv. Virus Res. 92, 101–109.

Roossinck, M.J., 2011. The good viruses: viral mutualistic symbioses. Nat. Rev. Microbiol. 9, 99–108.

Roossinck, M.J., 2015. A new look at plant viruses and their potential beneficial roles in crops. Mol. Plant Pathol. 16, 331–333.

Ross, R., 1911. The Prevention of Malaria. Murray, London, UK.

Ryder, E., et al., 1964. Transmission of common *Lettuce mosaic virus* through the gametes of the lettuce plant. Plant Dis. Report. 48 522-3.

Sacristán, S., Díaz, M., Fraile, A., García-Arenal, F., 2011. Contact transmission of *Tobacco mosaic virus*: a quantitative analysis of parameters relevant for virus evolution. J. Virol. 85, 4974–4981.

Sastry, K.S., 2013. Seed-Borne Plant Virus Diseases. Springer, New York.

Scott, S., Zimmerman, M., 2006. The complete sequence of the genome of *Humulus japonicus latent virus*. Arch. Virol. 151, 1683–1687.

Smith, C.C., Fretwell, S.D., 1974. The optimal balance between size and number of offspring. Am. Nat., 499–506.

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Stewart, A.D., Logsdon, J.M., Kelley, S.E., 2005. An empirical study of the evolution of virulence under both horizontal and vertical transmission. Evolution 59, 730–739.
Stuart, G.W., Moffett, P.K., Bozarth, R.F., 2006. A comprehensive open reading frame phylogenetic analysis of isometric positive strand ssRNA plant viruses. Arch. Virol. 151, 1159–1177.
Thompson, J.R., Kamath, N., Perry, K.L., 2014. An evolutionary analysis of the *Secoviridae* family of viruses. PLoS One 9, e106305.
Thrall, P., Boere, A., Uyenoyama, M., 1995. Frequency-dependent disease transmission and the dynamics of the *Silene-Ustilago* host–pathogen system. Am. Nat. 145, 43–62.
Thrall, P.H., Antonovics, J., 1997. Polymorphism in sexual versus non-sexual disease transmission. Proc. R. Soc. Biol. Sci. Ser. B 264, 581–587.
Thrall, P.H., Antonovics, J., Hall, D.W., 1993. Host and pathogen coexistence in sexually transmitted and vector-borne diseases characterized by frequency-dependent disease transmission. Am. Nat. 142, 543–552.
Thrall, P.H., Antonovics, J., Wilson, W.G., 1998. Allocation to sexual versus nonsexual disease transmission. Am. Nat. 151, 29–45.
Troutman, J., Bailey, W., Thomas, C., et al., 1967. Seed transmission of *Peanut stunt virus*. Phytopathology 57, 1280–1281.
Turina, M., Ciuffo, M., Lenzi, R., Rostagno, L., Mela, L., Derin, E., Palmano, S., 2006. Characterization of four viral species belonging to the family Potyviridae isolated from *Ranunculus asiaticus*. Phytopathology 96, 560–566.
Uyemoto, J., Grogan, R., et al., 1977. Southern bean mosaic virus: evidence for seed transmission in bean embryos. Phytopathology 67, 1190–1196.
Wang, D., Maule, A.J., 1992. Early embryo invasion as a determinant in pea of the seed transmission of *Pea seed-borne mosaic virus*. J. Gen. Virol. 73, 1615–1620.
de Wit, C.T., 1960. On competition. Versl. Landbouwk. Onderz. 66.
Wroth, J., Jones, R., 1992. *Subterranean clover mottle virus* infection of subterranean clover: widespread occurrence in pastures and effects on productivity. Crop Pasture Sci. 43, 1597–1608.
Xie, W.S., Antoniw, J.F., White, R.F., Jolliffe, T.H., 1994. Effects of beet cryptic virus infection on sugar beet in field trials. Ann. Appl. Biol. 124, 451–459.
Xu, P., Chen, F., Mannas, J.P., Feldman, T., Sumner, L.W., Roossinck, M.J., 2008. Virus infection improves drought tolerance. New Phytol. 180, 911–921.
Yang, Y., Kim, K.S., Anderson, E.J., 1997. Seed transmission of *Cucumber mosaic virus* in spinach. Phytopathology 87, 924–931.

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