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Highlights

* We use adaptive dynamics theory to study the evolution of plant-virus symbioses

* Ecological bistability in a discrete-time model with frequency-dependent transmission

* Darwinian extinction can occur under optimizing selection

* Evolutionary branching of parasitic and mutualistic viral symbioses

* Mutualism can outcompete parasitism in the long-run

The evolution of parasitic and mutualistic plant-virus symbioses through transmission-virulence trade-offs

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Running title: The evolution of mutualistic viral symbioses

Keywords: horizontal; vertical; seed; vector; adaptive dynamics; bi-stability

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Abstract

Virus-plant interactions range from parasitism to mutualism. Viruses have been shown to increase fecundity of infected plants in comparison with uninfected plants under certain environmental conditions. Increased fecundity of infected plants may benefit both the plant and the virus as seed transmission is one of the main virus transmission pathways, in addition to vector transmission. Trade-offs between vertical (seed) and horizontal (vector) transmission pathways may involve virulence, defined here as decreased fecundity in infected plants. To better understand plant-virus symbiosis evolution, we explore the ecological and evolutionary interplay of virus transmission modes when infection can lead to an increase in plant fecundity. We consider two possible trade-offs: vertical seed transmission vs infected plant fecundity, and horizontal vector transmission vs infected plant fecundity (virulence). Through mathematical models and numerical simulations, we show 1) that a trade-off between virulence and vertical transmission can lead to virus extinction during the course of evolution, 2) that evolutionary branching can occur with subsequent coexistence of mutualistic and parasitic virus strains, and 3) that mutualism can out-compete parasitism in the long-run. In passing, we show that ecological bi-stability is possible in a very simple discrete-time epidemic model. Possible extensions of this study include the evolution of conditional (environment-dependent) mutualism in plant viruses.

1 Introduction

Plant viruses exhibit the full symbiont spectrum and thus can have a range of effects on plants (Roossinck, 2011; Bao and Roossinck, 2013; Fraile and García-Arenal, 2016). Plant viruses can confer herbivore resistance (Gibbs, 1980), pathogen resistance (Shapiro et al., 2012), and drought tolerance (Xu et al., 2008; Davis et al., 2015). Differential effects of viruses on plants occur due to variation in environment and genetics of plants and viruses (Johansen et al., 1994, 1996; Domier et al., 2007, 2011; van Mölken and Stuefer, 2011; Davis et al., 2015; Hily et al., 2016). Some viruses have neutral or positive effects on plants by not affecting or increasing components of fitness, respectively (van Mölken and Stuefer, 2011; Davis et al., 2015; Hily et al., 2016). These recent works contradict decades of extensive research on plant viruses elucidating the negative effects of viruses in agronomic systems. Results from these previous works have led to the convention of virologists referring to viruses as pathogens. In light of recent findings, it is clear that plant viruses do not always lead to disease and therefore by definition are not always pathogens (Pagán et al., 2014; Fraile and García-Arenal, 2016).

Virus-plant interactions are obligate, symbiotic interactions that exist along a spectrum from parasitism to commensalism to mutualism. Parasitic associations occur when one species exists at a cost to the other, which follows the convention of virus-plant interactions. Commensalism occurs when one species profits from the interaction, but has no effect on the other species. The plant benefits the virus by promoting virus transmission. In the common bean (*Phaseolus vulgaris*) seed number and weight were not affected by *Phaseolus vulgaris* endornavirus 1 and 2 (R. A. Valverde pers. comm.). In a mutualistic relationship net effects are positive with enhanced survival and/or reproduction for both the plant and virus, thus as with all mutualisms the benefits outweigh the costs of the relationship. *Cucumber mosaic virus* (CMV) benefits *Arabidopsis thaliana* by increasing seed production in comparison to plants without virus though this effect

depends upon environmental conditions (Hily et al., 2016). CMV alters volatiles in *Solanum lycopersicum* making it more attractive to pollinators (Groen et al., 2016), which may enhance virus transmission by seed.

Plant viruses have evolved various modes of transmission resulting in genetic variation within and among virus species to interact with the genetic variation within and among plant species (Johansen et al., 1994, 1996; Domier et al., 2007, 2011). Some viruses are integrated into the plant genome and thus are persistent (Harper et al., 2002). Certain virus species can circulate within an insect vector or propagate within an insect vector resulting in persistent virus transmission to plants, while other vector-transmitted viruses are transferred in a semi-persistent to non-persistent manner (intermediate to short timeframe). Most viruses depend upon more than one mode of natural transmission by pollen, seed, and vector (reviewed in Hamelin et al. 2016) though having a suite of transmission modes can lead to trade-offs among modes of transmission.

Trade-offs between seed and vector transmission may occur when vector transmission is positively correlated with virulence, defined here as reduced fecundity in infected plants, as opposed to increased mortality in infected plants (Doumayrou et al., 2013). Serial passage of the *Barley stripe mosaic virus* in *Hordeum vulgare* through vectors resulted in an increase in vector transmission rate and virulence (reduced seed production), whereas serial passage through seed led to an increase in seed transmission and a decrease in virulence (increased seed production) (Stewart et al., 2005). Likewise, serial passage of *Cucumber mosaic virus* (CMV) by seed of *Arabidopsis thaliana* led to an increase in seed transmission rate, decline in CMV virulence (increased total seed weight) and reduction in virus accumulation (Pagán et al., 2014). A trade-off between virulence and vector transmission in a parasitic virus can lead to the emergence and coexistence of virulent vector-borne strains and less virulent, non-vector borne strains of virus (Hamelin et al., 2016). Futhermore, trade-offs between modes of transmission can result in the coexistence of different modes of virus transmission within a plant population that is

evolutionarily stable (Hamelin et al., 2016).

To better understand plant-virus symbiosis evolution, we explore the ecological and evolutionary interplay of virus transmission modes between seeds and vectors when infection can lead to an increase in plant fecundity, which was not addressed by (Hamelin et al., 2016). We consider two possible trade-offs: vertical seed transmission vs infected plant fecundity, and horizontal vector transmission vs infected plant fecundity (virulence). We use mathematical models and numerical simulations to address three questions: 1) Can a trade-off between virulence and vertical transmission lead to virus extinction in evolutionary time? 2) As a virus evolves, can evolutionary branching occur with subsequent coexistence of mutualistic and parasitic virus strains? 3) Can mutualism outcompete parastism in the long-run?

2 Ecological model

2.1 Discrete-time model

The model includes two methods for viral transmission to a host plant: (1) infected vectors and (2) infected seeds. A discrete-time model is formulated since each of the transmission events occur at different time periods during the year. Therefore, the year is divided into two periods, corresponding to vector and seed transmission, denoted as V and S, respectively:

$$t \underbrace{\rightarrow}_{V} t' \underbrace{\rightarrow}_{S} t+1\,.$$

During the time interval $[t,t']$, the newly developed plants are colonized by vectors. Virus transmission from the vector to the host plant occurs during this first time interval. During the second time interval $[t',t+1]$, seeds drop to the ground and those that survive, either uninfected or infected seeds, germinate and produce new uninfected or infected plants, respectively. We assume

there is no seed bank. At the beginning of the next year, $t+1$, seeds have germinated and produced new plants. The annual cycle repeats.

To keep the model simple, there are no explicit vector dynamics. The acquisition of the virus by non-viruliferous vectors, and inoculation of the host plant by viruliferous vectors are modeled implicitly. Only the dynamics of the host plant are modeled. Two variables account for the plant dynamics during each of these two stages. The two variables are H and I, the density of uninfected and infected plants, respectively. The total density of uninfected and infected plants is denoted as $T = H + I$. The plant dynamics are observed each year at time t, $t = 0, 1, 2, \ldots$ after seed transmission and before vector transmission.

During the vector stage V, the Poisson distribution is used to model virus transmission between the vector and the host plant. Let Λ_V denote the parameter in the Poisson distribution: it is the average number of viruliferous vector visits per plant per year that result in subsequent inoculation of an uninfected plant. Horizontal transmission parameter β relates this number to the infection prevalence at the beginning of the vector stage. Virus transmission through vectors is assumed to depend on the frequency of infected plants, I/T, rather than on their density I (Ross, 1911; Hamelin et al., 2016). Then

$$\Lambda_V = \beta \frac{I(t)}{T(t)}$$

Hence, the probability of no successful virus transmission from vectors to a given host plant is $\exp(-\Lambda_V)$ and the probability of successful transmission is $1 - \exp(-\Lambda_V)$. Therefore, at time t', the model takes the form:

$$\begin{aligned} H(t') &= H(t)e^{-\Lambda_V} = H(t)\exp\left(-\beta\frac{I(t)}{T(t)}\right), \\ I(t') &= I(t) + H(t)[1 - e^{-\Lambda_V}] = I(t) + H(t)\left[1 - \exp\left(-\beta\frac{I(t)}{T(t)}\right)\right]. \end{aligned} \tag{1}$$

Notice that at low infected plant density ($I(t) \ll H(t) \approx T(t)$),

$$I(t') \approx I(t) + H(t)\beta \frac{I(t)}{T(t)} \approx I(t)(1+\beta)\,, \tag{2}$$

i.e., β is like a multiplication factor of infected plants associated with vector transmission.

For the second transmission stage S, we assume competition and overcrowding between neighboring plants reduces the number of seeds per plant (Watkinson and Harper, 1978; Pacala and Silander Jr, 1985). Density-dependent effects apply equally to uninfected and infected plants. Let b_H and b_I denote the effective number of seeds produced per uninfected or infected plant, respectively, at low plant density. We assume that the virus infects both the maternal plant and the seeds. Thus, only infected plants produce infected seeds. At low plant density, more than one effective seed is produced per uninfected plant,

$$b_H > 1. \tag{3}$$

The seeds that survive germinate into either uninfected or infected plants. If vertical transmission is full, 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.

We apply a well-known form for plant density-dependence due to de Wit (1960) (also known as Beverton-Holt density-dependence in animal populations). The model in the second stage is

$$\begin{aligned} H(t+1) &= \frac{b_H H(t') + q b_I I(t')}{1 + \lambda T(t')}, \\ I(t+1) &= \frac{p b_I I(t')}{1 + \lambda T(t')}, \end{aligned} \tag{4}$$

where $T(t) = H(t) + I(t)$ and λ describes density-dependent competition between plants.

The full vector-seed transmission model consists of the preceding models for the two stages

Table 1: Model parameters and variables.

Notation	Definition	Unit
t	time in years, $t = 0, 1, 2, \ldots$	time
$T(t)$	total plant density at time t	per area
$H(t)$	uninfected plant density at time t	per area
$I(t)$	infected plant density at time t	per area
b_H	effective number of seeds per uninfected plant	none
b_I	effective number of seeds per infected plant	none
$p = 1 - q$	infected seed transmission probability	none
β	vector transmission parameter	none
λ	plant competition parameter	area

V and S, equations (1)–(4). Combining these two pairs of difference equations, the model can be expressed as a first-order difference equation for uninfected and infected plants, i.e.,

$$\begin{aligned} H(t+1) &= \frac{b_H H(t) P(t) + q b_I \left(I(t) + H(t)\left(1 - P(t)\right)\right)}{1 + \lambda T(t)}, \\ I(t+1) &= \frac{p b_I \left(I(t) + H(t)\left(1 - P(t)\right)\right)}{1 + \lambda T(t)}, \end{aligned} \tag{5}$$

where

$$P(t) = \exp\left(-\beta \frac{I(t)}{T(t)}\right)$$

is the probability an uninfected plant escapes infection during year t. Table 1 lists all model variables and parameters with their definition.

2.2 Basic reproductive number

At virus-free equilibrium (VFE), the density of infected plants is zero and the density of uninfected plants is

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

The basic reproductive number for model (5) is computed from linearization of the difference equation for the infected host I about the VFE:

$$\mathscr{R}_0 = \frac{pb_I}{b_H}(1+\beta). \tag{6}$$

If the reproductive number is greater than one, then our annual plant model shows that these two transmission mechanisms may be able to maintain the virus within the host population. If viral transmission is purely vertical, limited only to seed transmission ($\beta = 0$), then $\mathscr{R}_0 > 1$ if and only if $pb_I > b_H$, which requires $b_I > b_H$. That is, this simple model shows that purely vertical transmission of a virus through the seed cannot maintain the virus in the host population unless infected plants have greater fecundity than uninfected plants (Fine, 1975; Hamelin et al., 2016). Note that the ratio b_I/b_H represents the extent to which host fecundity is reduced/increased by virus infection. If reduced, then the ratio is a measure of the virulence of the virus (virus-induced loss of fitness).

In the mathematical analysis (Appendix A), we focused on the case $p = 1$ (full vertical transmission), while simulations were additionally performed for $p < 1$ (partial vertical transmission; Figure 1). For the case $p = 1$, a second basic reproductive number for invasion of uninfected plants into an entirely infected plant population is derived. The equilibrium where the entire plant population is infected is referred to as the susceptible-free equilibrium (SFE). A new threshold value for the SFE is defined as

$$\overline{\mathscr{R}_0} = \frac{b_H}{b_I}\exp(-\beta).$$

If $\overline{\mathscr{R}_0} < 1$, then the SFE is stable and if $\overline{\mathscr{R}_0} > 1$ then the SFE is unstable (Appendix A.2). It appears that $p < 1$ is required for stable coexistence of both uninfected and infected plants to occur (Appendix A). Figure 1-B shows that for $b_I > 1$ and $p < 1$, the dynamics indeed converge

to an endemic equilibrium where uninfected and infected plants coexist.

2.3 Parameterization

The uninfected plant fecundity parameter b_H can be estimated from plant population dynamics. For instance, b_H ranges between 1.6 and 3.3 for the sand dune annual *Vulpia fasciculata* (Watkinson and Harper, 1978; Watkinson, 1980). By contrast, b_H is approximately 85 in Kherson oat (Montgomery, 1912; de Wit, 1960). Thus, b_H may range from 1 to 100, depending on the plant species considered. In this paper, infected plants may have greater fitness than uninfected plants, so b_I may range from 0 to 100 as well. Throughout the paper, we scale the plant densities by assuming a spatial unit such that $\lambda = 1$, without loss of generality.

In our model, β is a multiplication factor (Eq. 2) comparable to the basic reproductive number but restricted to the vector transmission period V (Eq. 6). Basic reproductive numbers are gaining increasing attention in the plant virus literature (Froissart et al., 2010; Péréfarres et al., 2014), yet few studies provide estimated values for this quantity. Reasonable values of β may range from 0 to 10 (Holt et al., 1997; Madden et al., 2000; Jeger et al., 2004), even though larger values might also be relevant (Escriu et al., 2003; Madden et al., 2007).

3 Evolutionary analysis

We follow an adaptive dynamics approach (Metz et al. 1992; Dieckmann and Law 1996; Geritz et al. 1998; Diekmann 2004). To address the evolution of mutualistic viral symbioses, the single-strain model (5) is first extended to n virus strains which differ in their abilities to be seed-transmitted (b_I, p) or vector-transmitted (β). We then consider a plant population infected with $n = 2$ virus strains, I_i, $i = 1,2$, which differ in their phenotypes. To simplify the notations, we drop the subscript I in b_I to replace it by the strain index i. Let $x_1 = (\beta_1, b_1, p_1)$ be the resident

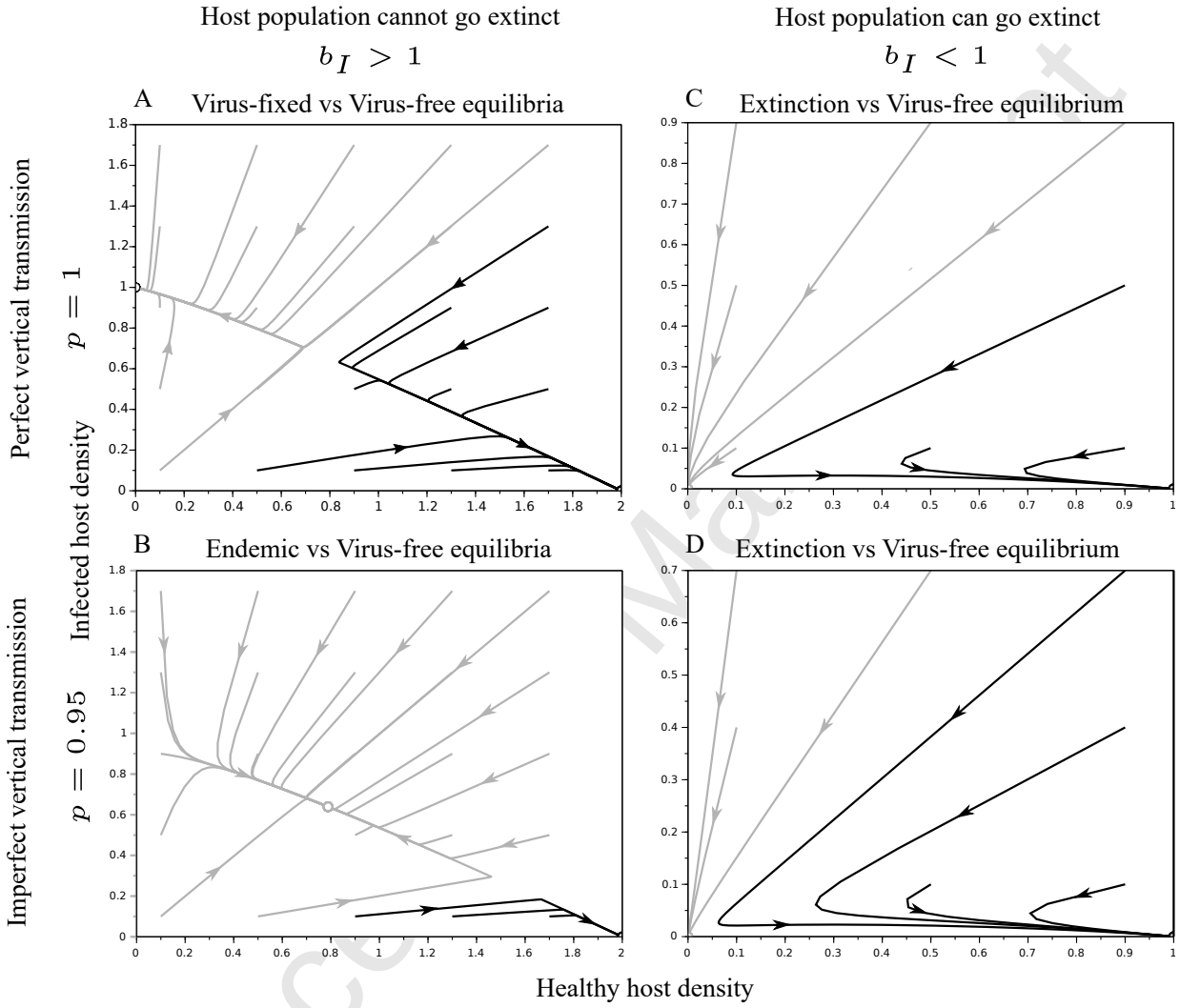

Figure 1: Virus and plant host population dynamics in the phase plane (H,I). Each panel shows a set of possible orbits. Ecological bi-stability occurs for these parameter values ($\mathscr{R}_0 < 1$ and, for $p = 1$, $\overline{\mathscr{R}_0} < 1$). Depending on initial conditions, the dynamics converge to the virus-free equilibrium (black curves) or to an alternative equilibrium (grey curves): (A) virus fixation in the plant population, (B) coexistence of uninfected and infected plants, (C-D) complete extinction of the plant host population. Parameter values: (A-B) $b_H = 3$, $b_I = 2$, $\lambda = 1$, (A) $\beta = 0.45$, $p = 1$, (B) $\beta = 0.57$, $p = 0.95$, (C-D) $b_H = 2$, $b_I = 0.5$, $\lambda = 1$ (C) $\beta = 2$, $p = 1$, (D) $\beta = 2$, $p = 0.95$.

phenotype and let $x_2 = (\beta_2, b_2, p_2)$ be the mutant phenotype. We assume the mutant initially represents a relatively small subpopulation as compared to the resident. That is, $I_2 \ll I_1$.

3.1 Multi-strain dynamics

A natural extension of the single-strain model (5) to n virus strains, I_i, $i = 1, \ldots, n$, with traits (β_i, b_i, p_i) is

$$\begin{aligned} H(t+1) &= \frac{b_H H(t)P(t) + \sum_{k=1}^{n}(1-p_k)b_k\left(I_k(t) + H(t)\left(1-P(t)\right)\frac{\beta_k I_k(t)}{\sum_{j=1}^{n}\beta_j I_j(t)}\right)}{1+\lambda T(t)}, \\ I_i(t+1) &= \frac{p_i b_i\left(I_i(t) + H(t)\left(1-P(t)\right)\frac{\beta_i I_i(t)}{\sum_{j=1}^{n}\beta_j I_j(t)}\right)}{1+\lambda T(t)}, \end{aligned} \tag{7}$$

where $T(t) = H(t) + \sum_{j=1}^{n} I_j(t)$. The probability uninfected plants escape vector infection becomes

$$P(t) = \exp\left(-\sum_{j=1}^{n}\beta_j \frac{I_j(t)}{T(t)}\right), \tag{8}$$

whereas the expression $(1-P(t))$ is the probability of vector infection from some strain (Hamelin et al., 2011).

3.2 Evolutionary invasion analysis

Following Metz et al. (1992), we are interested in testing whether the mutant can invade. In particular, if

$$\lim_{t\to\infty}\frac{1}{t}\log\left(\frac{I_2(t)}{I_2(0)}\right) < 0, \tag{9}$$

the mutant cannot invade the resident. For simplicity, we assume that the resident population with phenotype x_1 is at ecological equilibrium, i.e., $I_1(0) \approx i(x_1) = i_1 > 0$ and $H(0) = h(x_1) = h_1 > 0$. Thus, the resident population is at an equilibrium corresponding to coexistence of uninfected and

infected plants. We therefore define an evolutionary invasion condition as

$$\log\left(\frac{I_2(1)}{I_2(0)}\right) > 0. \tag{10}$$

From the assumptions $I_2 \ll I_1$ and the resident population at ecological equilibrium, it follows from model (7) with $n = 2$ strains that the evolutionary invasion condition (10) can be expressed as

$$\frac{I_2(1)}{I_2(0)} \approx \frac{p_2 b_2 \left(1 + h_1 (1 - P_1) \frac{\beta_2}{\beta_1 i_1}\right)}{1 + \lambda (h_1 + i_1)} > 0, \tag{11}$$

with

$$P_1 = \exp\left(-\beta_1 \frac{i_1}{h_1 + i_1}\right),$$

where P_1 is the probability that uninfected plants escape vector infection at the ecological equilibrium corresponding to the resident phenotype x_1. Using the fact that the resident population I_1 is at ecological equilibrium,

$$\frac{I_1(1)}{I_1(0)} \approx \frac{p_1 b_1 \left(1 + h_1 (1 - P_1) \frac{\beta_1}{\beta_1 i_1}\right)}{1 + \lambda (h_1 + i_1)} = 1,$$

simplifies the evolutionary invasion condition to

$$\frac{p_2 b_2 \left(1 + h_1 (1 - P_1) \frac{\beta_2}{\beta_1 i_1}\right)}{p_1 b_1 \left(1 + h_1 (1 - P_1) \frac{\beta_1}{\beta_1 i_1}\right)} > 1. \tag{12}$$

Let F_1 be the number of vector-borne infections per year relative to the force of infection of the resident population, i.e.,

$$F_1 = \frac{h_1 (1 - P_1)}{\beta_1 i_1}. \tag{13}$$

The evolutionary invasion condition (12) can equivalently be expressed as

$$\underbrace{(p_2 b_2 - p_1 b_1)}_{\substack{\text{seed-only}\\ \text{transmission}}} + \underbrace{(\beta_2 p_2 b_2 - \beta_1 p_1 b_1) F_1}_{\substack{\text{vector-seed}\\ \text{transmission}}} > 0\,. \tag{14}$$

The expression on the left side of (14) is an invasion fitness proxy function, $s(x_1, x_2)$, sign-equivalent to the invasion fitness function in (10). The dynamics of $s(x_1, x_2)$ as a function of the mutant phenotype x_2 determine the evolutionary trajectories.

In this paper, virulence is defined as the negative impact of the virus on host fitness, i.e., b_H/b_I. The remainder of the analysis is restricted to the case of bipartite transmission-virulence trade-offs with negative correlations between b_I and p (vertical transmission), and b_I and β (horizontal transmission).

3.3 Trade-off between vertical transmission and virulence

To consider a trade-off between seed transmission and virulence, we assume vector transmission is constant, $\beta_i = \beta$, $i = 1, 2$, then the invasion condition (14) reads

$$(p_2 b_2 - p_1 b_1)(1 + \beta F_1) > 0\,.$$

Since $F_1 \geq 0$, the preceding inequality is equivalent to

$$p_2 b_2 - p_1 b_1 > 0\,.$$

Next, assume there is a trade-off between virulence and seed transmission, i.e., $p_i = g(b_i)$, $i = 1, 2$, with $g'(b_i) < 0$. Then the invasion fitness proxy function depends only on b_1 and b_2. That

is,

$$s(b_1,b_2) = g(b_2)b_2 - g(b_1)b_1\,. \tag{15}$$

The dynamics of $s(b_1,b_2)$ as a function of b_2 determine the evolutionary trajectory. In this case, b evolves so as to maximize the product $g(b)b$ (Gyllenberg et al., 2011). This result was confirmed by numerical simulations (Figure 2 A-B; Appendix B). However, it may be that the value of b that maximizes $g(b)b$ is such that $b \leq 1$ (Figure 2 C-D). In this case, evolution drives the virus population to extinction (see also Figure 1). Such a phenomenon has recently been found to occur in a similar but continuous-time model with frequency-dependent horizontal transmission (Boldin and Kisdi, 2016). Darwinian extinction under optimizing selection can also occur through a catastrophic bifurcation (Parvinen and Dieckmann, 2013).

3.4 Trade-off between horizontal transmission and virulence

The trade-off between vector transmission and virulence yields a different evolutionary outcome than the trade-off between seed transmission and virulence. Assume seed transmission is constant, $p_i = p > 0$, $i = 1,2$. The invasion condition (14) is equivalent to

$$(b_2 - b_1) + (\beta_2 b_2 - \beta_1 b_1)\,F_1 > 0\,.$$

Let $\beta_i = f(b_i)$, $i = 1,2$, with $f'(b_i) < 0$. Then an invasion fitness proxy function is

$$s(b_1,b_2) = (b_2 - b_1) + (f(b_2)b_2 - b_1 f(b_1))\,F_1(b_1). \tag{16}$$

In this case, there may exist an evolutionary singular point, $b^\star$, if the selection gradient is zero,

$$G(b^\star) = \frac{\partial s}{\partial b_2}(b^\star,b^\star) = 1 + \left(f(b^\star) + b^\star f'(b^\star)\right) F_1(b^\star) = 0\,. \tag{17}$$

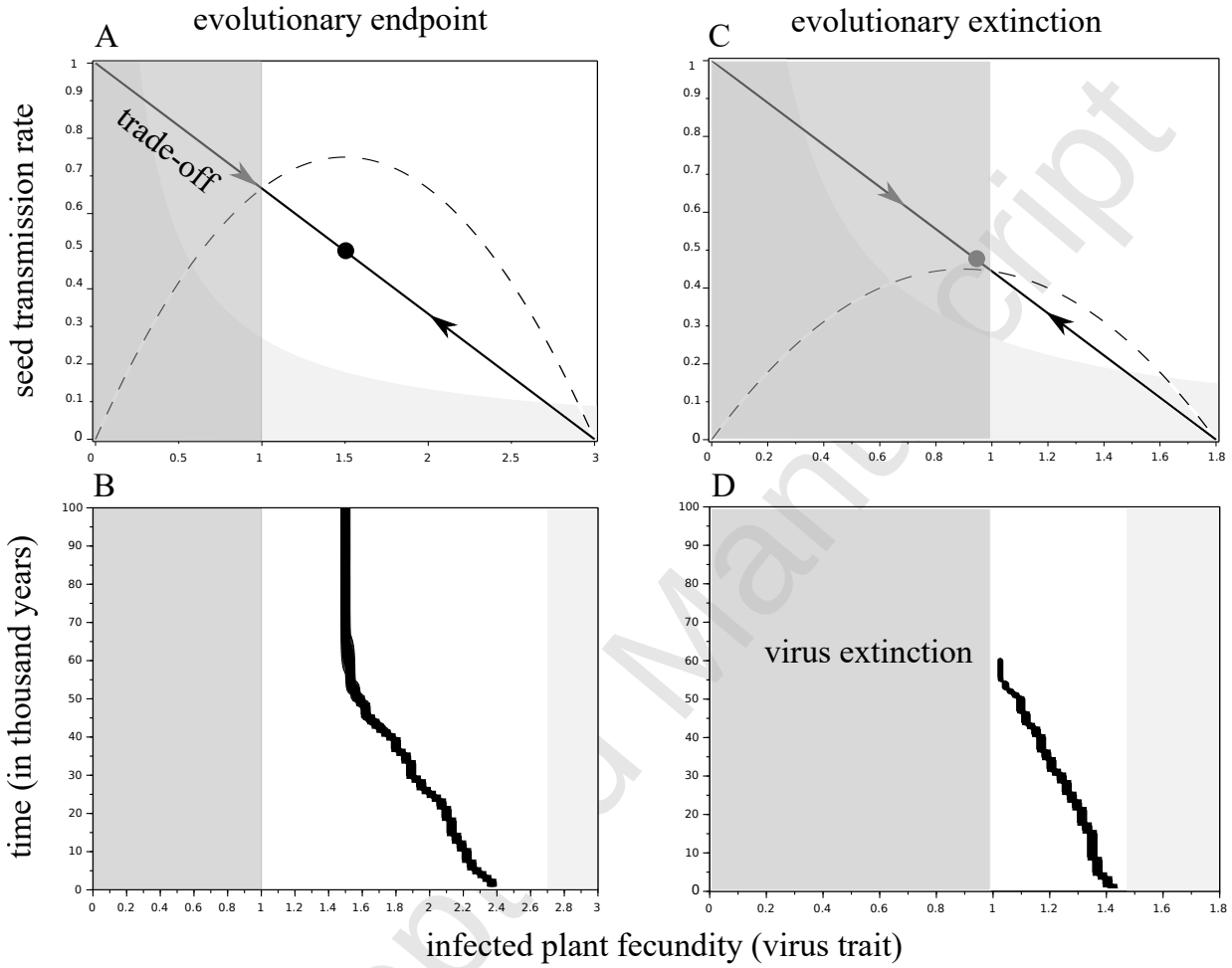

Figure 2: Evolutionary dynamics along a trade-off between infected plant fecundity (b_I) and seed transmission rate (p). The straight lines correspond to linear trade-off functions, i.e., $p = g(b_I) = 1 - b_I/B$, with (A-B) $B = 3$, (C-D) $B = 1.8$. The dashed curves correspond to the associated functions $g(b_I)b_I$. The dots denote trait values maximizing $g(b_I)b_I$. The arrows denote the direction of evolution. The light gray regions correspond to $\mathscr{R}_0 \leq 1$ (virus unable to invade). The darker gray regions correspond to $b_I \leq 1$, which leads to virus extinction (Fig. 1). The thick curves correspond to numerical simulations of the evolutionary dynamics (Appendix B): (B) starting from $b_I \approx 2.4$, evolution selects for decreasing b_I values until reaching an evolutionary endpoint ($b_I = 1.5$) corresponding to the maximum of $g(b_I)b_I$, (D) starting from $b_I \approx 1.4$, evolution selects for decreasing b_I values until reaching $b_I = 1$ where the virus population goes extinct. Other parameter values: $b_H = 3$, $\lambda = 1$, $\beta = 10$.

Whether $b^\star$ is evolutionarily stable is determined by the sign of the second derivative of s with respect to b_2, evaluated at $b_1 = b_2 = b^\star$. The stability condition is

$$\frac{\partial^2 s}{\partial b_2^2}(b^\star, b^\star) = \left(2f'(b^\star) + b^\star f''(b^\star)\right) F_1(b^\star) < 0\,. \tag{18}$$

Since $F_1(b^\star) > 0$ and $f'(b^\star) < 0$, $b^\star$, if it exists, is evolutionarily stable for concave or linear trade-off functions ($f''(b^\star) \leq 0$). For convex trade-off functions ($f''(b^\star) > 0$), $b^\star$ may be unstable.

The singular point $b^\star$ is evolutionarily attractive if the derivative of the selection gradient G in (17) at $b^\star$ is negative, i.e.,

$$G'(b^\star) = (f(b^\star) + bf'(b^\star))F_1'(b^\star) + (2f'(b^\star) + bf''(b^\star))F_1(b^\star) < 0\,. \tag{19}$$

Unfortunately, we have no explicit expression of F_1, which makes conditions (18) and (19) intractable to analysis. Therefore, the trade-off between virulence and vector transmission is explored through numerical simulations.

To perform the numerical computations, we considered the trade-off form:

$$\beta = f(b) = \beta_{\max} \exp(-k(b - b_{\min}))\,.$$

This exponential form is convex and its curvature increases with k ($f''(b) = k^2 f(b) > 0$). Also, this exponential form allows us to check the stability of a singular point as in this special case, the stability condition (18) becomes:

$$\frac{\partial^2 s}{\partial b_2^2}(b^\star, b^\star) = (2 - kb^\star) f'(b^\star) F_1(b^\star) < 0\,.$$

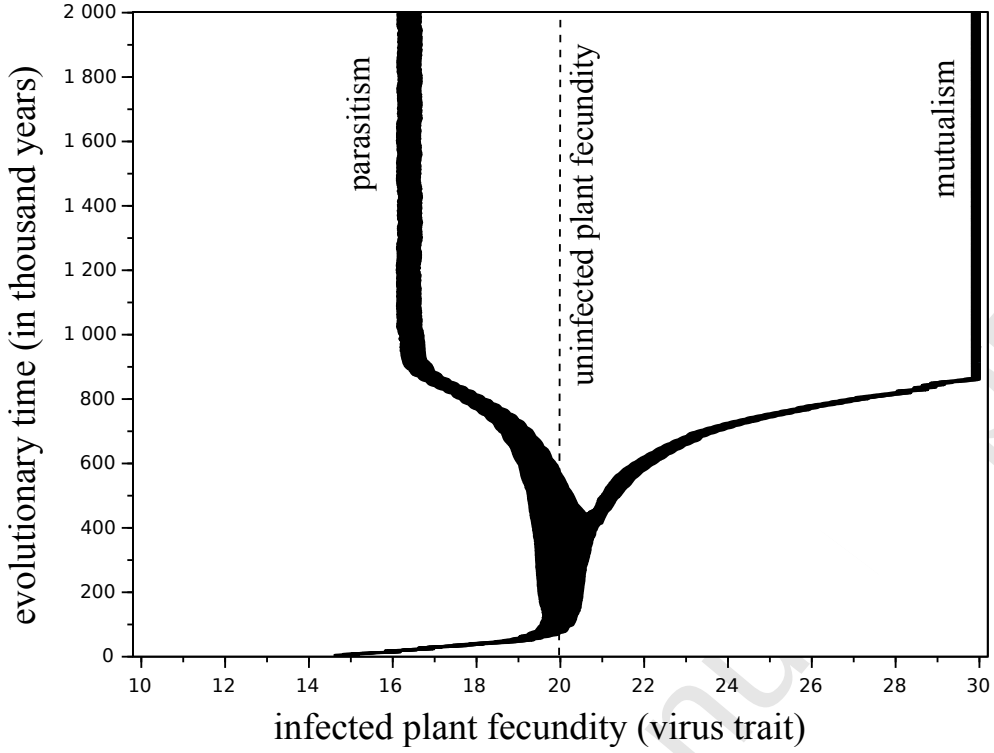

Figure 3: Evolutionary branching of parasitic and mutualistic viral symbioses and their long-run coexistence. We assumed a trade-off between transmission and virulence of the form: $\beta = f(b) = \beta_{\max} \exp(-k(b - b_{\min}))$. Parameter values: $b_H = 20$, $\lambda = 1$, $b_{\min} = 10$, $b_{\max} = 30$, $p = 0.5$, $\beta_{\max} = 10$, $k = 0.1$.

Since $f'(b^\star) < 0$, the evolutionary stability of a singular point $b^\star$ requires

$$2 - kb^\star > 0\,.$$

For the parameter set corresponding to Figures 3 and 4, including $b_H = 20$ and $k = 0.1$, the critical value (indeterminate stability) is $b_c = 2/k = 20$. In our simulations, $b^\star$ seems to be slightly above b_c, thus branching occurs after a relatively long period of apparent stability. Extensive numerical simulations indicate that evolutionary branching is the rule rather than the exception in this model. However the fact that $b^\star$ approximately coincides with both b_c and b_H is a coincidence used for illustrative purposes only. For instance, for $b_H = 15$ and the other parameters unchanged, $b^\star \approx 21$ is clearly greater than $b_c = 20$ and $b_H = 15$ (not shown).

Figures 3 and 4 show that it is possible for a mutualistic symbiosis to evolve ($b_I/b_H > 1$)

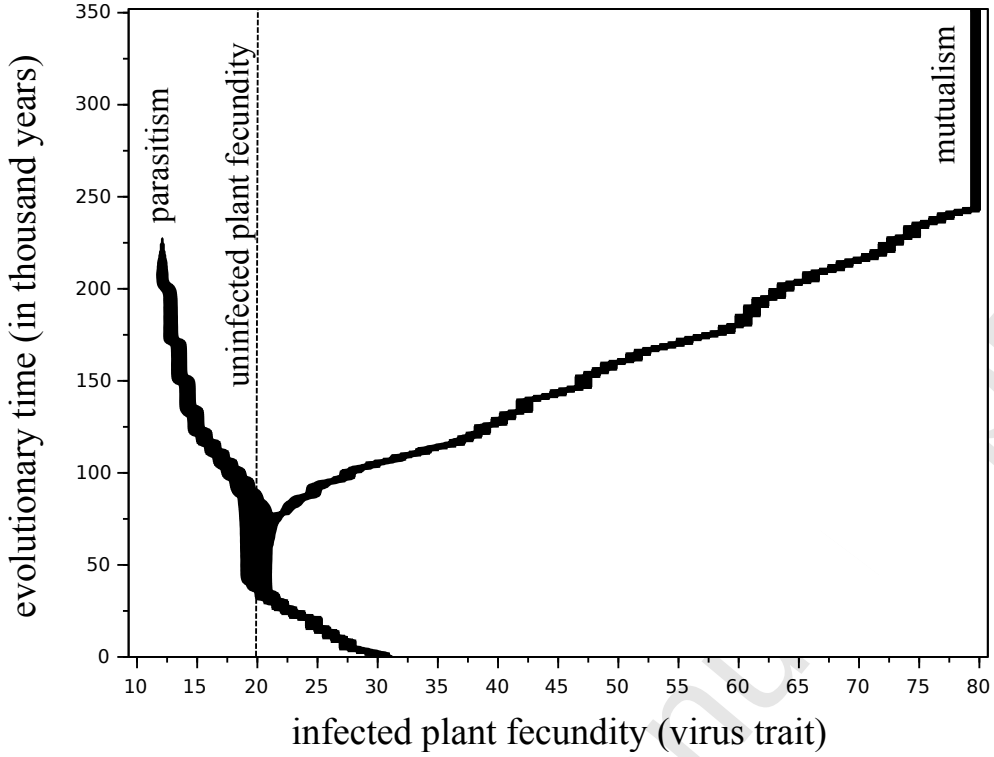

Figure 4: Evolutionary branching of parasitic and mutualistic viral symbioses and the eventual exclusion of parasitism by mutualism. We assumed a trade-off between transmission and virulence of the form: $\beta = f(b) = \beta_{\max} \exp(-k(b - b_{\min}))$. Parameter values: $b_H = 20$, $\lambda = 1$, $b_{\min} = 10$, $b_{\max} = 80$, $p = 0.5$, $\beta_{\max} = 10$, $k = 0.1$.

(or not) from a parasitic symbiosis ($b_I/b_H < 1$) (Fig. 3), or conversely for a parasitic symbiosis to evolve (or not) from an initial mutualistic symbiosis (Fig. 4). Starting from a monomorphic virus population, evolutionary dynamics may converge towards commensalism and split into two branches: parasitism and mutualism ($b_I/b_H < 1$ and $b_I/b_H > 1$, respectively). The evolutionary outcome depends on the biologically feasible maximum plant host fecundity value: if it is large then mutualism may exclude parasitism in the long-run (Fig. 4), otherwise both parasitic and mutualistic variants may coexist in the long-run (Fig. 3).

4 Discussion

4.1 Findings

4.1.1 Ecological model

The discrete-time ecological model of an annual plant virus we developed included two modes of transmission: vector and seed. Key parameters include vector transmissibility β, uninfected and infected plant fecundities b_H and b_I, resp., and seed transmissibility $p \leq 1$. We can summarize our findings in terms of these parameters and the basic reproductive number $\mathscr{R}_0$ that defines a threshold for successful invasion of infected plants. The main conclusions concern the type of virus–plant interaction, coexistence of infected and uninfected plants, and ecological bistability.

First, if there is only seed transmission, i.e., $\beta = 0$, then $\mathscr{R}_0 = pb_I/b_H$ indicating that purely vertical transmission through seed cannot maintain the virus in the host population unless the plant–virus symbiosis is mutualistic ($b_I > b_H$). If, however, vector transmission is included with seed transmission ($\beta > 0$) then a parasitic virus ($b_I < b_H$) may be maintained in the host population.

Second, we checked conditions for the coexistence of uninfected and infected plants in specific models. In the case of full vertical transmission ($p = 1$), there is a susceptible-free equilibrium corresponding to virus fixation. It is stable if the threshold $\overline{\mathscr{R}_0}$ for successful invasion of uninfected plants is below one. However, numerical simulations indicate that a stable coexistence state between uninfected and infected plants does not exist for full vertical transmission. Instead, simulations suggest that stable coexistence requires partial seed transmission ($p < 1$).

Third, we have found bistability in this model. That is, the dynamic behavior and the long-term solutions in particular depend on the initial conditions. There are three different types of bistability.

(i) There is bistability between the virus-free and susceptible-free equilibria, i.e., either infected or uninfected plants go extinct but not both. This occurs if $p = 1$, $b_I > 1$, $\mathscr{R}_0 < 1$ and $\overline{\mathscr{R}_0} < 1$. It is remarkable because the virus can infect the entire plant population even though $\mathscr{R}_0 < 1$. However, virus fixation in this case requires that healthy host plants have not reached their carrying capacity and the initial density of infected plants is sufficiently large, see the example in Figure 1A.

(ii) There is bistability between an endemic coexistence equilibrium and the virus-free equilibrium, i.e., either both uninfected and infected plants coexist or infected plants go extinct. This has been observed for $p < 1$, $b_I > 1$, and $\mathscr{R}_0 < 1$. The virus persists in the population in coexistence with uninfected plants, provided the latter are away from the uninfected carrying capacity state and the density of infected plants is sufficiently large, see the example in Figure 1B. That is, the infection can establish itself in the host population even though $\mathscr{R}_0 < 1$.

(iii) There is bistability between the virus-free equilibrium and extinction, i.e., either the virus infects all plants or drives the entire plant population to extinction. This has been observed for both full and partial vertical transmission, $\mathscr{R}_0 < 1$, $\overline{\mathscr{R}_0} < 1$ and $b_I < 1$. The latter condition means that infected plants cannot persist on their own. If the virus is introduced in sufficiently large density of plants that have not reached their uninfected carrying capacity state, the virus drives the entire plant population extinct, see the examples in Figure 1C,D. Disease-induced host extinction is well-known to occur in time-continuous models with frequency-dependent horizontal transmission for the case $\mathscr{R}_0 > 1$ (e.g. Getz and Pickering, 1983; Busenberg and van den Driessche, 1990), as virus transmission is ongoing even when the population density is close to zero. In discrete-time models, host extinction caused by disease-related mortality seems to have been less investigated (but see Franke and Yakubu,

2008, who also consider $\mathscr{R}_0 > 1$). Here, we have shown that disease-induced host extinction can occur even if $\mathscr{R}_0 < 1$.

The occurrence of ecological bistability in an epidemiological model as simple as the one considered here is remarkable for three reasons. First, infection can persist in the population even if $\mathscr{R}_0 < 1$. This can be particularly important if control measures to combat virus infections are aimed at reducing the basic reproduction number below one, because this will not be sufficient and a higher level of control will be necessary. The reason for this apparent 'failure' of the basic reproduction number is its derivation from the assumption that the system is at virus-free equilibrium. However, this of course not always the case, and one may even argue that this assumption rarely holds true considering the plethora of perturbations in variable and stochastic environments. That is, if the densities of infected and uninfected plants are far from this equilibrium, the basic reproduction number does not apply anymore and may grossly underestimate the possibility of virus invasion. In particular, we have shown that if the density of infected plants is high or the density of uninfected plants low, the virus is likely to invade the population or even drive it extinct even if $\mathscr{R}_0 < 1$. Similar observations have been made in epidemiological models with backward bifurcations (e.g. Dushoff et al., 1998). In fact, numerical simulations (not shown here) suggest that our model exhibits a backward bifurcation as well.

Second, short-term dynamics can become particularly important if the system is bistable. Figure 5A shows the long-term total plant density as a function of vector transmissibility β. For an intermediate parameter range ($0.29 < \beta < 0.33$) there is bistability between the susceptible-free and virus-free equilibrium. However, if we consider the plant densities after short-term (Fig. 5B), they show a range of values between the two equilibrium values. This is because the system dynamics becomes very slow for some initial plant densities such that they take very long to approach the equilibrium (there is an unstable coexistence state which slows down the

dynamics in its vicinity; cf. Appendix A.1). Transients are therefore important if the system is bistable, as they 'diversify' the values taken by the plant densities. Moreover, due to this effect, the bistability region has effectively 'expanded' to neighboring parameter regions.

Third, there is no bistability in similar (and even more general) continuous-time models possible (Zhou and Hethcote, 1994). The simplest model with frequency-dependent transmission that we know of and leads to bistability is of SEI type, i.e., has an extra compartment of latent infections (Gao et al., 1995). In this model, bistability is possible for complete disease-induced sterilization of the host population (Gao et al., 1995, Sect. 5), i.e., in terms of our model parameters $b_I = 0$. Considering that a latent infection compartment introduces a form of time delay in the disease and host reproduction dynamics, it may not be too surprising that our discrete-time SI model and the continuous-time SEI model show similar behavior.

4.1.2 Evolutionary analysis

The ecological model was used to explore the evolution of the plant-virus symbiosis (parasitic or mutualistic). The main conclusions from the evolutionary analysis are summarized below:

(i) Vertical (seed) transmission (p) versus virulence (defined as b_H/b_I): evolution maximizes the product pb_I, i.e., maximizes transmission relative to virulence. Interestingly, such a trade-off can lead to virus extinction in evolutionary time.

(ii) Horizontal (vector) transmission (β) versus virulence: evolutionary branching and the subsequent coexistence of parasitic and mutualistic symbioses is possible, as well as the extinction of the parasitic branch.

In the evolutionary simulations of vector transmission versus virulence, we assumed a simple exponential trade-off function. Its convex shape allows for richer evolutionary dynamics than linear or concave trade-off forms. Consideration of other trade-off shapes (e.g. linear) indicated

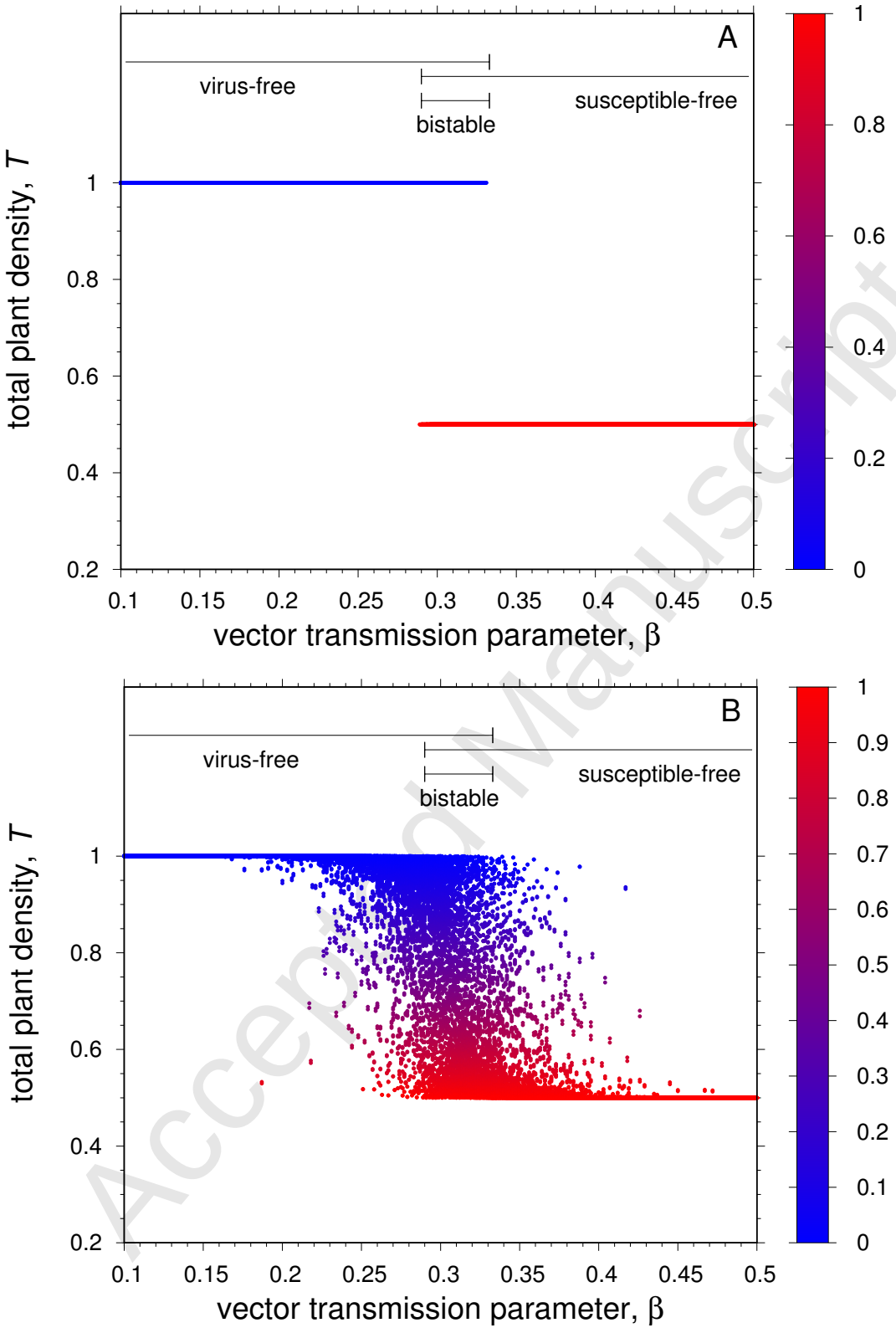

Figure 5: Total plant population density as a function of vector transmission parameter β. (A) Long-term dynamics, approximated after 10,000 years, (B) short-term dynamics after 100 years. The threshold criteria $\overline{\mathscr{R}_0} = 1$ and $\mathscr{R}_0 = 1$ correspond to $\beta \approx 0.33$ and $\beta \approx 0.29$, respectively. In between these parameter values, the system tends to either the virus-free equilibrium with $T = (b_H - 1)/\lambda = 1$ or the susceptible-free equilibrium with $T = (b_I - 1)/\lambda = 0.5$, depending on initial conditions. The color coding indicates the infection prevalence. For each value of β, 100 initial conditions were drawn from a pseudo-uniform random distribution. Parameter values: $b_H = 2$, $b_I = 1.5$, $\lambda = 1$, $p = 1$.

that other outcomes are theoretically possible, such as directional selection and convergence to a stable monomorphic evolutionary endpoint (as expected from the mathematical analysis). However, we never observed parasitism excluding mutualism after evolutionary branching occurred. This might be because seed production is a necessary condition for virus year-to-year persistence in our annual plant model.

4.2 Limits and prospects

In this study, we focused on unconditional mutualism, i.e., when infected plant fecundity is always greater than uninfected plant fecundity. However, conditional mutualism occurs when infected plants have lower fecundity than uninfected plants under favorable conditions, and higher fecundity than uninfected plants under unfavorable conditions such as water stress (Hily et al., 2016). Our model may be extended to address the evolution of conditional mutualism. A possibility would be to consider that b_H is a random variable that can take two values $b_H^{\min}$ and $b_H^{\max}$, corresponding to unfavorable and favorable conditions, respectively, with mean $\bar{b}_H$. One may then let $b_I = \bar{b}_H + c(b_H - \bar{b}_H) - v$, where v (for virulence) is the possible loss of fecundity due to infection, and $c \in [0,1]$ is a coefficient buffering the variations of fecundity in infected plants, subject to selection (if $c = 0$, the variance is zero). For instance, $c = 0$ implies infected plants have constant fecundity regardless of environmental variability. Whether and how such conditional mutualism would evolve is left for future research.

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A Additional analyses

A.1 Full vertical transmission

Focusing on the case $p = 1$ (full vertical transmission), model (5) reads:

$$\begin{aligned} H(t+1) &= \frac{b_H H(t) P(t)}{1+\lambda T(t)}, \\ I(t+1) &= \frac{b_I \left(I(t) + H(t)\left(1-P(t)\right)\right)}{1+\lambda T(t)}. \end{aligned} \tag{20}$$

If $b_I > 1$, there exists a "susceptible-free" equilibrium (SFE) which is found by setting $H = 0$ and solving for I. The SFE value for I is

$$\bar{I} = \frac{b_I - 1}{\lambda}.$$

Linearizing the difference equation for the uninfected host H about the SFE, we obtain the basic reproductive number of an uninfected host introduced into a fully infected population:

$$\overline{\mathscr{R}_0} = \frac{b_H}{b_I} \exp\left(-\beta\right).$$

The notation $\overline{\mathscr{R}_0}$ stands for the dual of $\mathscr{R}_0$ (Hamelin et al., 2016). If $\overline{\mathscr{R}_0} > 1$ then the SFE is

unstable. If both $\mathscr{R}_0 > 1$ and $\overline{\mathscr{R}_0} > 1$, then infected and uninfected plants can invade each other when rare, so coexistence of uninfected and infected plants is protected (Kisdi and Geritz, 2003).

It appears that there is no stable coexistence equilibrium with both uninfected and infected plants. The ecologically relevant results are summarized for $b_I > 1$ and $b_I < 1$.

In the case $b_I > 1$, there exist both a VFE and a SFE. The two reproductive numbers equal

$$\mathscr{R}_0 = \frac{b_I}{b_H}(1+\beta) \quad \text{and} \quad \overline{\mathscr{R}_0} = \frac{b_H}{b_I}\exp(-\beta)\,,$$

respectively. It follows that

$$\mathscr{R}_0\overline{\mathscr{R}_0} < 1\,.$$

Therefore, $\mathscr{R}_0 > 1$ and $\overline{\mathscr{R}_0} > 1$ is impossible; coexistence of uninfected and infected plants is not protected. Moreover, in Section A.1.1 it is shown that there exists an endemic equilibrium (EE) such that $H, I > 0$ if and only if $\mathscr{R}_0 < 1$ and $\overline{\mathscr{R}_0} < 1$ but it does not appear to be stable. In addition, it is shown that if $\overline{\mathscr{R}_0} < 1$, then the SFE is locally stable. Both reproductive numbers less than 1 leads to ecological bi-stability. The three ecologically relevant cases are summarized below (see also Figure 6).

1: If $\mathscr{R}_0 < 1$ and $\overline{\mathscr{R}_0} > 1$, then the VFE is globally stable.

2: If $\mathscr{R}_0 < 1$ and $\overline{\mathscr{R}_0} < 1$, then there is bi-stability of the VFE and the SFE (either infected or uninfected plants go extinct but not both).

3: If $\mathscr{R}_0 > 1$ and $\overline{\mathscr{R}_0} < 1$, then the SFE is globally stable.

For the case $b_I < 1$, there is no SFE, only the VFE. The numerical results indicate that there are only two ecologically relevant cases.

4: If $\overline{\mathscr{R}_0} < 1$, then there is bi-stability of the VFE and the extinction "equilibrium" (either

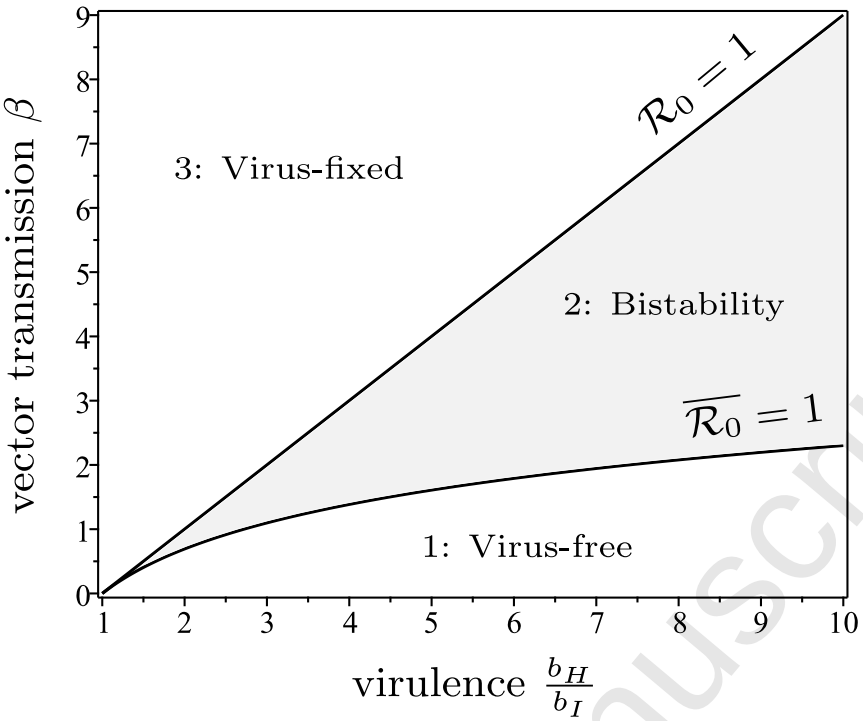

Figure 6: The three ecologically relevant cases for $p = 1$ and $b_I > 1$, with $\mathscr{R}_0 = (b_I/b_H)(1+\beta)$ and $\overline{\mathscr{R}_0} = (b_H/b_I)\exp(-\beta)$ (see text). The range of β values for which ecological bistability occurs increases with b_H/b_I (virulence).

infected plants go extinct or there is complete population extinction).

5: If $\overline{\mathscr{R}_0} > 1$, then the VFE is globally stable.

Simulations performed for $q = 1 - p \ll 1$ (slightly partial vertical transmission) showed similar results to the case $p = 1$ with the exception that the SFE becomes an endemic equilibrium (for which we have no explicit expression). Therefore, coexistence of uninfected and infected plants is possible in this model.

A.1.1 Existence conditions of an endemic equilibrium

Model (5) has the following form:

$$\begin{aligned} H(t+1) &= \frac{b_H H(t)\exp\left(-\frac{\beta I(t)}{T(t)}\right) + qb_I\left[I(t)+H(t)\left[1-\exp\left(-\frac{\beta I(t)}{T(t)}\right)\right]\right]}{1+\lambda T(t)} \\ I(t+1) &= \frac{pb_I\left[I(t)+H(t)\left[1-\exp\left(-\frac{\beta I(t)}{T(t)}\right)\right]\right]}{1+\lambda T(t)}. \end{aligned}$$

We focus on the case $p = 1-q = 1$ (full vertical transmission). Let

$$H^* = \frac{H}{\bar{H}}, I^* = \frac{I}{\bar{H}}.$$

The dimensionless model (asterisk notation has been dropped) simplifies to

$$H(t+1) = \frac{b_H H(t)\exp\left(-\frac{\beta I(t)}{H(t)+I(t)}\right)}{1+(b_H-1)(H(t)+I(t))} \tag{21}$$

$$I(t+1) = \frac{b_I\left[I(t)+H(t)\left[1-\exp\left(-\frac{\beta I(t)}{H(t)+I(t)}\right)\right]\right]}{1+(b_H-1)(H(t)+I(t))}. \tag{22}$$

There exist at most three equilibria:

$$(1,0),\ \left(0,\frac{b_I-1}{b_H-1}\right),\ \text{and}\ (h,i).$$

Consider the proportions $\hat{i} = i/(h+i)$ and $\hat{h} = h/(h+i)$. Then $\hat{i}+\hat{h} = 1$. If $b_I \geq 1$, then the total plant population is bounded below by a positive constant (Appendix A.2). If the population does not go extinct, then existence of a unique $\hat{i}$, $0 < \hat{i} < 1$ implies existence of a unique (h,i). We derive conditions for existence of a unique $\hat{i}$, $0 < \hat{i} < 1$. Using the notation h, i and $\hat{i}$ in Eq. (21), it follows that $1+(b_H-1)(h+i) = b_H e^{-\beta\hat{i}}$. Substituting this latter expression into Eq. (22), we

obtain an implicit expression for $\hat{i}$:

$$\hat{i} = \frac{b_I(1-(1-\hat{i})e^{-\beta\hat{i}})}{b_H e^{-\beta\hat{i}}}$$

which can be expressed as

$$\hat{i}\left(\frac{b_H}{b_I}-1\right)+1 = e^{\beta\hat{i}}. \tag{23}$$

The two curves $f_1(\hat{i}) = \hat{i}\left(\frac{b_H}{b_I}-1\right)+1$ and $f_2(\hat{i}) = e^{\beta\hat{i}}$ intersect at $\hat{i}=0$ so that a unique positive solution exists $\hat{i}$, $0<\hat{i}<1$, if and only if the following conditions hold:

$$\frac{b_H}{e^{\beta}} < b_I < \frac{b_H}{1+\beta}$$

522 ($f_1'(0) > f_2'(0)$ and $f_1(1) < f_2(1)$). The left side of the inequality is equivalent to $\overline{\mathscr{R}_0} < 1$ and the right side is equivalent to $\mathscr{R}_0 < 1$.

524 A.2 Plant population is bounded

For model (1)–(4), it is shown that the total plant population is bounded and if the average number 526 of seeds per infected plant is greater than one, $b_I > 1$, then the plant population always persists.

The total plant population, $T(t) = H(t) + I(t)$ is bounded below by zero; $H(t)$ and $I(t)$ are 528 nonnegative. In addition, we show that the total population is bounded above and for the case $b_I > 1$, the total population is bounded below by a positive constant. The total plant population 530 satisfies the inequality

$$T(t+1) \le \frac{b_H T(t)}{1+\lambda T(t)} = f_H(T(t)),$$

since $b_H > b_I$. Comparing the solution $T(t)$ with the solution of the difference equation, $x(t+$ 532 $1) = f_H(x(t))$, where $x(0) = T(0) > 0$, it follows that $T(1) \le f_H(T(0)) = f_H(x(0)) = x(1)$.

Since $f_H(x)$ is monotone increasing for $x \in [0,\infty)$, $f_H(T(1)) \leq f_H(x(1))$, leads to $T(2) \leq x(2)$ and in general, from induction it follows that $T(t) \leq x(t)$ for $t \in \{0,1,2,3,\ldots\}$. The fact that $x(t)$ approaches $\bar{H} = (b_H - 1)/\lambda$ monotonically implies $T(t) \leq \max\{T(0), \bar{H}\}$.

A similar argument applies in the case $b_I > 1$ to show that the total plant population is bounded below by a positive constant, e.g., uniform persistence. The inequality $b_H > b_I$ leads to the reverse inequality for the total plant population:

$$T(t+1) \geq \frac{b_I T(t)}{1+\lambda T(t)} = f_I(T(t)).$$

Comparing the solution of $T(t)$ with the solution of $y(t+1) = f_I(y(t))$, $T(0) = y(0)$, leads to $T(t) \geq y(t)$ for $t \in \{0,1,2,\ldots\}$. Since $b_I > 1$, the solution $y(t)$ converges monotonically to $\bar{I} = (b_I - 1)/\lambda > 0$ which implies $T(t) \geq \min\{T(0), \bar{I}\}$.

A.3 Absence of vector transmission

Focusing on the case $\beta = 0$ (no vector transmission), model (5) reads:

$$\begin{aligned} H(t+1) &= \frac{b_H H(t) + (1-p) b_I I(t)}{1+\lambda T(t)}, \\ I(t+1) &= \frac{p b_I I(t)}{1+\lambda T(t)}, \end{aligned} \tag{24}$$

where $T(t) = H(t) + I(t)$. There exist at most three equilibria:

$$(0,0), \quad \left(\frac{b_H - 1}{\lambda}, 0\right), \quad \left(h = \frac{b_I(1-p)(1-pb_I)}{\lambda(b_H - b_I)}, i = \frac{(pb_I - b_H)(1-pb_I)}{\lambda(b_H - b_I)}\right).$$

If $b_H > b_I$, then $h > 0$ implies $pb_I < 1$, and $i > 0$ thus implies $pb_I > b_H$ which is impossible since $b_H > 1$. If $b_I > b_H$ then $h > 0$ implies $pb_I > 1$ and $i > 0$ thus implies $pb_I > b_H > 1$. Therefore, the endemic equilibrium (h,i) existence requires $pb_I > b_H$.

B Evolutionary simulations

Evolutionary computations in Figures 2, 3 and 4 were realized from the multi-strain model (7) using the following algorithm. The evolving phenotype b ranges from $b_{\min}$ to $b_{\max}$, the biologically feasible minimum and maximum plant host fecundity values. The interval $[b_{\min}, b_{\max}]$ is divided into a finite number of subintervals (here 100), each with length Δb. The evolutionary dynamics are governed by the following iteration scheme. The scheme is initiated with a given value of b 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 $1,000$ years), then a small mutation $\pm\Delta b$ occurs in b with equal likelihood of being smaller or larger than b. Time is advanced by one unit in evolutionary time ($1,000$ years) and b is changed to either $b+\Delta b$ or $b-\Delta b$. The evolutionary process continues with this new b value.