

Spatiotemporal Model of Barley and Cereal Yellow Dwarf Virus Transmission Dynamics with Seasonality and Plant Competition

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Abstract Many generalist pathogens are influenced by the spatial distributions and relative abundances of susceptible host species. The spatial structure of host populations can influence patterns of infection incidence (or disease outbreaks), and the effects of a generalist pathogen on host community dynamics in a spatially heterogeneous community may differ from predictions derived via simple models. In this paper, we model the transmission of a generalist pathogen within a patch framework that incorporates the movement of vectors between discrete host patches to investigate the effects of local host community composition and vector movement rates on disease dynamics.

We use barley and cereal yellow dwarf viruses (B/CYDV), a suite of generalist, aphid-vectored pathogens of grasses, and their interactions with a range of host species as our case study. We examine whether B/CYDV can persist locally or in a patch framework across a range of host community configurations. We then deter-

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mine how pathogen-mediated interactions between perennial and annual competitors are altered at the local and regional scale when the host populations are spatially structured. We find that the spatial configuration of the patch system, host composition within patches, and patch connectivity affect not only the ability of the pathogen to invade a fragmented system, but also determine whether the pathogen facilitates the invasion of a non-native host species. Further, our results suggest that connectivity can interact with arrival time and host infection tolerance to determine the success or failure of establishment for newly arriving species.

Keywords BYDV · Competition · Age-structure · Patch model · Invasion · SI model · Disease

1 Introduction

Humans are converting and fragmenting landscapes on every continent, changing connectivity of habitats through effects including reduced patch size, creation of novel habitats, and altered movement rates among patches that affect a diversity of species. Pathogen movement and epidemics can depend intimately upon landscape connectivity patterns (Smith et al. 2002; Meentemeyer et al. 2008), which in turn, control epidemic propagation or fadeout (Jeger et al. 2007; Saramäki and Kaski 2005). Importantly, models including spatial heterogeneity can make qualitatively different predictions compared to models assuming homogeneous mixing (Holmes 1997; Hess et al. 2002; Keeling et al. 2004). In addition, many emerging pathogens infect multiple hosts, but most multi-host theory developed to date has focused on non-spatial models (Dobson 2004; Keesing et al. 2006; McCormack and Allen 2007; Power and Mitchell 2004; Holt and Pickering 1985; Begon et al. 1992; Bowers and Turner 1997; Holt and Dobson 2006). Thus, in spite of the importance of landscape connectivity for understanding spatial spread and persistence of disease in real communities, the body of spatially-explicit theory dealing with multi-host pathogens remains quite small (Ostfeld et al. 2008, Chap. 5; McElhany et al. 1995). As a result, the spatial dynamics of multi-species host-parasite assemblages are gaining increasing attention in both mathematics and ecology. In particular, metapopulation and patch models of disease are gaining impetus with the recognition that species live in increasingly fragmented landscapes (Hanski 1998; Arino 2009; Arino and van den Driessche 2006; Arino et al. 2005; Grenfell and Harwood 1997; McCallum and Dobson 2002; McCormack 2006), and that the heterogeneity of the landscape, as well as the demography and the epidemiology of multiple interacting species, determine spatial spread and persistence of the disease (Plantegenest et al. 2007).

The dynamics of generalist pathogens depend on many factors that influence their persistence and determine the manner in which disease spreads. Among these factors spatial dynamics are particularly important for plant pathogens because natural plant communities exist in spatially heterogeneous landscapes. The different host species, affected by a common generalist pathogen, are often distributed in patches

(Plantegenest et al. 2007). Another factor is cross-species transmission dynamics. Host species differ in their susceptibility to a disease and their competency in transmitting the disease to other hosts. Hence, the diversity and composition of a community can influence the pathogen load at both population and community levels (Knops et al. 1999; Mitchell et al. 2002; Power and Mitchell 2004). The mere presence of a host that is highly susceptible to a disease can lead to a local epidemic, while the presence of a host with a low reservoir competency can lead to a dilution effect where the overall disease prevalence is reduced (Keesing et al. 2006). Finally, if a generalist pathogen is vector-transmitted then host populations may also differ in their contact with and effect on the vector population (Power and Mitchell 2004).

Pathogens that are host generalists can also mediate the outcome of interspecific competition between host species. If the pathogen has differential effects on the fitness of the competing species, relative competitive strengths, and hence population outcomes can be altered (Borer et al. 2007). Theoretical and empirical investigations have shown that a generalist pathogen infecting multiple (competing) host species can influence species diversity and community structure (Power and Mitchell 2004; Keesing et al. 2006; Borer et al. 2007; Chase et al. 2002; Hatcher et al. 2006; Knops et al. 1999). Consequently, generalist pathogens can have a significant impact on endangered species, particularly in the presence of a species that acts as a reservoir for the pathogen (de Castro and Bolker 2005).

In this paper, we use barley and cereal yellow dwarf viruses (B/CYDV), a suite of aphid-vectored pathogens, and their interactions with a range of host species as our case study. Our goal is to construct and analyze a model that helps in determining the possibility of invasion of native species by exotic (i.e., non-native) species due to the presence of disease (B/CYDV) among the (competing) multiple species in a patch framework.

Here we develop a multi-patch framework to examine the influence of spatial heterogeneity, seasonality, and competition on disease dynamics and pathogen-mediated plant invasion. We begin by investigating the effects of local host community composition and vector movement on B/CYDV dynamics in which the landscape is divided into a system of discrete patches containing smaller local populations, with disease transmission occurring via vector movement between patches (Sect. 3). We then analyze a simplified two patch model in order to derive the B/CYDV system's basic reproduction number, which serves as a threshold for invasion into a susceptible host community. We also use the basic reproduction number to examine the sensitivity of spatial transmission dynamics to key epidemiological and biological parameters (Sect. 4). We then examine whether B/CYDV can persist locally or in a patch framework across a range of host community configurations. First, we adjust the number of patches connected by aphid migration and vary aphid migration rates between patches. We also modify community configurations to examine whether pathogen-mediated interactions and competitive outcome between perennial and annual competitors are altered at the local and regional scale when the host populations are spatially structured (Sect. 5).

2 The B/CYDV Empirical System

B/CYDV is one of the most economically important diseases of grain crops worldwide and infects over 100 grass species in both agricultural and natural systems (Irwin and Thresh 1990). Because it can be a devastating crop pathogen, the vast majority of the theoretical and empirical studies of B/CYDV have been focused on crop settings. Thus, the history of modeling of this pathogen is strongly focused on epidemiology in single host species (Kendall 1992; Fabre et al. 2006; Leclercq-Le Quillec et al. 2000; Zhang and Holt 2001). However, B/CYDV also infects many non-crop grass species. Exotic (i.e., non-native) annual grasses have been invading and displacing native perennial grasses in much of the western United States (Borer et al. 2007; Malmstrom et al. 2005b, 2005a). Recent theoretical and empirical work has demonstrated that B/CYDV may also play a critically important role in facilitating and maintaining natural grassland invasion (Malmstrom et al. 2005b; Borer et al. 2007). Borer et al. (2007) analyzed a non-spatial model of B/CYDV, which suggested that the virus could reverse the competitive outcome between perennial and annual host grasses, leading to the successful invasion by the competitively inferior annuals. However, continued existence of B/CYDV requires the persistence of the perennial grass in the community due to its role as a reservoir for infection between growing seasons. Hence, we incorporate patch structure in the non-spatial model considered in Borer et al. (2007) to examine the effect of spatial heterogeneity of the host species on disease dynamics and the possibility of invasion by exotic species.

The virus has a short latency period in both its host plants and the aphid vector; however, once infected, a vector is potentially infective for life and individual hosts typically do not recover from a B/CYDV infection. Host susceptibility to B/CYDV varies, with some species suffering increased mortality and reduced fecundity when infected and other species experiencing little change in their overall fitness (Irwin and Thresh 1990). Studies have also shown that the presence of highly competent reservoir species can increase the prevalence of B/CYDV in local host communities (Power and Mitchell 2004; Borer et al. 2007; Malmstrom et al. 2005b, 2005a). Host-aphid interactions also vary by host, with aphids showing preference for and experiencing higher fitness on certain host species (Lowry 2007; McElhany et al. 1995). The various host species often compete for resources and the presence of B/CYDV may alter their relative competitive abilities, leading to shifts in host community composition (Borer et al. 2007). Annual grasses may act to amplify the prevalence of B/CYDV because aphids feed preferentially on annuals and have higher growth rates when feeding on annuals (Borer et al. 2009). The transmission rates to and from aphids may also be higher for annual grasses (Cronin et al. 2010). While the effect of these host community differences have been investigated at the local level, their importance for regional patterns of B/CYDV spread and persistence have not been fully explored. Both local, within-field movements and long-distance dispersal by aphids are important for B/CYDV transmission (Irwin et al. 1988), and host-vector interactions at multiple spatial scales may influence local and regional disease dynamics.

3 Model Formulation

In California grasslands, the wet winter and spring growing seasons alternate with hot, dry summers, during which perennial grasses remain dormant and annuals persist only as seeds. We use a differential-difference modeling approach (Borer et al. 2007) to describe the punctuated seasonality of this system. During the growing season (of length τ), differential equations are the effective model, and the dormant summer season is described by difference equations $[(t_n + \tau) \text{ to } (t_n + 1 = t_{n+1})]$ where $t_n = n$ is time in years. Since time units are in years, $\tau < 1$. We use the following susceptible-infected model structure to summarize the multi-host-pathogen dynamics of the California grassland community.

Each of the vector and host sub-populations consists of susceptible (S) and infected (I) individuals. Vector population dynamics are modeled explicitly in order to represent infection dynamics within the aphid population as well as aphid migration between patches. Vector population dynamics and epidemiology during the growing (rainy) season are represented by a pair of differential equations for each subpopulation in a patch model. We use a simplified, reduced parameter Lotka–Volterra competition formulation (Case 1990), with a reduced competitive pressure from infected individuals (parameterized as ϵ), as described in Borer et al. (2007). Fecundity and biomass reduction are represented by the same parameter ϵ , because plant fecundity is largely a function of plant biomass and the empirical estimates were identical. We explicitly track susceptible perennial seedlings (p_S), infected perennial seedlings (p_I), susceptible perennial adults (P_S), infected perennial adults (P_I), susceptible annuals (a_S), and infected annuals (a_I). We also explicitly track the susceptible vectors (V_S), and infected vectors (V_I). Figure 1 shows the competitive structure between the

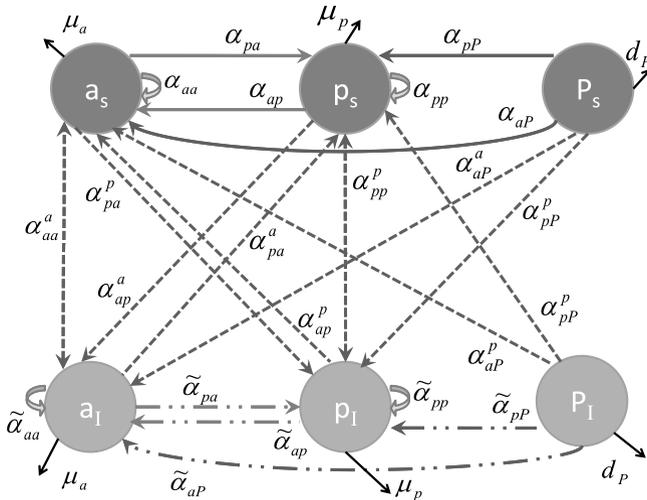


Fig. 1 Transfer diagram for the growing season depicting the population dynamics of hosts, and the competitive interactions between the susceptible and infected host plants. In the figure, the parameters α_{ij}^k , $\tilde{\alpha}_{ij}$ are defined as $\alpha_{ij}^k = \epsilon_k \alpha_{ij}$, and $\tilde{\alpha}_{ij} = \epsilon_j \epsilon_j \alpha_{ij}$ with $\epsilon_P = \epsilon_p$

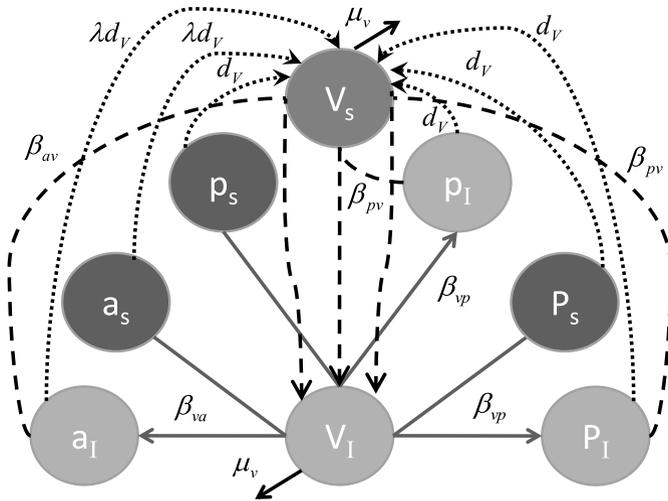


Fig. 2 Transfer diagram for the growing season depicting the population dynamics of vectors and the disease transmission between hosts and vectors

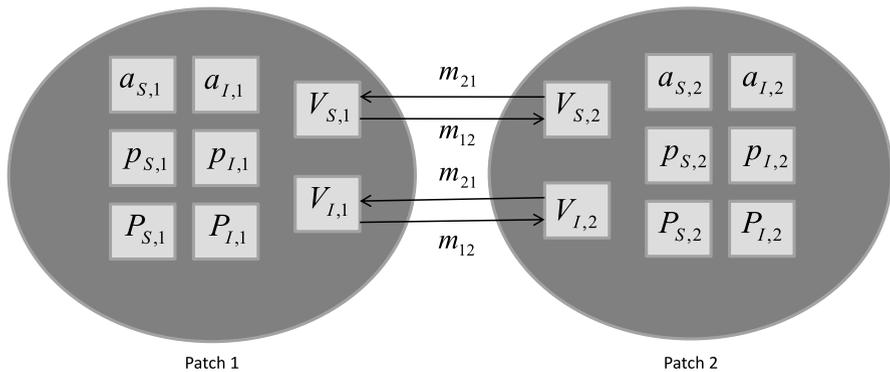


Fig. 3 Patch diagram for two patches, depicting the different compartments in each patch, and the movement of vectors between patches

susceptible and infected host compartments, while Fig. 2 shows the transmission of infection from vectors to hosts. Figure 3 depicts the movement of vectors between patches. Aphid population growth depends on the relative densities of annual and perennial hosts, because aphids show both a preference for annual grasses and higher performance (fecundity) on annual hosts (Borer et al. 2009).

During the dry season, it is assumed that all infected aphids die, while the uninfected aphids may either survive as a function of their density at the end of the growing season or they may recolonize from outside the patch system. These two possibilities correspond to different life history strategies employed by aphids; some species remain within the grassland at low abundances between growing seasons with uninfected offspring emerging at the beginning of the growing season, while others

migrate to an alternate host, typically in another habitat (e.g., *Rhopalosiphum padi* switches from grasses to a species of *Prunus* during the dry season).

The model considers age structure in the perennial grasses. Perennial adults are qualitatively different from annual grasses; they are competitively superior and less palatable to aphids (Borer et al. 2009). In contrast, first-year perennials are more similar to annuals in these characteristics. The model also examines both reduced fecundity and disease-induced dormant season mortality. See Table 1 for a description of model parameters. The subscript S represents susceptible and I represents infected individuals of a species and/or age class. The subscript j indicates the patch in which the individuals reside.

Host and vector growing season/continuous time equations for $t_n < t \leq t_n + \tau$: (a = annuals, p = 1st year perennials, P = adult perennials, V = vector)

$$\frac{dps_{j}}{dt} = -(\mu_p \eta_p + \beta_{vp} V_{I,j}) ps_{j}, \tag{1a}$$

$$\frac{dpl_{j}}{dt} = -\mu_p \eta_p \epsilon_p pl_{j} + \beta_{vp} V_{I,j} ps_{j}, \tag{1b}$$

$$\frac{dPs_{j}}{dt} = -\beta_{vp} V_{I,j} Ps_{j}, \tag{1c}$$

$$\frac{dPl_{j}}{dt} = \beta_{vp} V_{I,j} Ps_{j}, \tag{1d}$$

$$\frac{das_{j}}{dt} = -(\mu_a \eta_a + \beta_{va} V_{I,j}) as_{j}, \tag{1e}$$

$$\frac{dal_{j}}{dt} = -\mu_a \eta_a \epsilon_a al_{j} + \beta_{va} V_{I,j} as_{j}, \tag{1f}$$

$$\frac{dVs_{j}}{dt} = r(t) - (\beta_{av} a_{I,j} + \beta_{pv}(pl_{j} + Pl_{j})) Vs_{j} - \mu_v Vs_{j} + Ms_{j}, \tag{1g}$$

$$\frac{dVl_{j}}{dt} = (\beta_{av} a_{I,j} + \beta_{pv}(pl_{j} + Pl_{j})) Vs_{j} - \mu_v Vl_{j} + Ml_{j}. \tag{1h}$$

Dry season/discrete time for each patch, j , and for $t_n + \tau \rightarrow t_{n+1}$:

$$ps_{j}(t_{n+1}) = b_P (ps_{j}(t_n + \tau) + \epsilon_p pl_{j}(t_n + \tau)), \tag{2a}$$

$$pl_{j}(t_{n+1}) = 0, \tag{2b}$$

$$Ps_{j}(t_{n+1}) = \sigma_{Ps} (ps_{j}(t_n + \tau) + Ps_{j}(t_n + \tau)), \tag{2c}$$

$$Pl_{j}(t_{n+1}) = \sigma_{Pl} (pl_{j}(t_n + \tau) + Pl_{j}(t_n + \tau)), \tag{2d}$$

$$as_{j}(t_{n+1}) = b_A (as_{j}(t_n + \tau) + \epsilon_a al_{j}(t_n + \tau)), \tag{2e}$$

$$al_{j}(t_{n+1}) = 0, \tag{2f}$$

$$Vs_{j}(t_{n+1}) = C, \tag{2g}$$

$$Vl_{j}(t_{n+1}) = 0. \tag{2h}$$

Plant competition terms:

$$\eta_a = 1 + \alpha_{aa}(a_{S,j} + \epsilon_a a_{I,j}) + \alpha_{ap}(p_{S,j} + \epsilon_p p_{I,j}) + \alpha_{aP}(P_{S,j} + \epsilon_P P_{I,j}), \quad (3a)$$

$$\eta_p = 1 + \alpha_{pp}(p_{S,j} + \epsilon_p p_{I,j}) + \alpha_{pa}(a_{S,j} + \epsilon_a a_{I,j}) + \alpha_{pP}(P_{S,j} + \epsilon_P P_{I,j}). \quad (3b)$$

Vector growth term:

$$r(t) = d_V(\lambda(a_{S,j} + a_{I,j}) + (p_{S,j} + p_{I,j}) + (P_{S,j} + P_{I,j})). \quad (4)$$

Vector migration terms where $Y \in \{S, I\}$:

$$M_{Y,j} = - \sum_{k=1, k \neq j}^N m_{jk} V_{Y,j} + \sum_{k=1, k \neq j}^N m_{kj} V_{Y,k}, \quad (5)$$

where N is the total number of patches. We remark here that the parameter m_{jk} denotes the migration rate from patch j to patch k and not from patch k to j , as is assumed in some other papers.

We note that B/CYDV requires an aphid vector for transmission from plant to plant and cannot be spread via seeds. We use a Lotka–Volterra competition framework because of the nature of the data available for parameter estimation. In addition, host density makes a sensible common currency for competition and disease for a systemic virus (Borer et al. 2007). MATLAB was used to numerically simulate the outcome of reciprocal invasion experiments with susceptible and infected perennials and annuals as both residents and invaders and to test the sensitivity of our results to the estimated vital rate values.

3.1 Estimation of Model Parameter Values from Field Data

A great deal of information about aphid reproductive rates, host composition of grasslands, transmission competence of hosts for certain B/CYDV serotypes, and population dynamic effects of infection on different host species is available for model parameterization (Borer et al. 2007). We have amassed data on 20 different native and exotic, annual, and perennial grass US West Coast species (Cronin et al. 2010; Borer et al. 2009; Welsh, Borer, and Mitchell, unpubl. data). The species for which we have many experimental and observational estimates represent some of the most important native and invasive grasses in the system. In addition, species we have examined span the range of characteristics from extremely widespread to extremely restricted West Coast ranges as well as locally abundant and always rare within communities. Although B/CYDV infection has been documented in at least 33 native and 80 exotic grasses in California (Malmstrom 1998), we focus on two common grasses, *Elymus glaucus*, a native perennial, and *Bromus hordeaceus*, an exotic annual, because these species have among the broadest ranges of West Coast grassland species. They have been the focus of our own multi-year B/CYDV monitoring and, among the native perennials, the best-quality published prevalence data are available for *E. glaucus* (Malmstrom et al. 2005b). Although we parameterize the model for these two species, for our numerical simulations (see parameter values in Table 1), we

Table 1 Description of model parameters and values used for initial model analysis and simulation. Values were estimated from empirical work in California grasslands. Discrete and continuous units are year⁻¹ (except ϵ and λ which are unitless), and competition parameters are individual⁻¹

Symbol	Description	Value	Reference
<i>Discrete time parameters</i>			
b_P	Perennial birth rate	45.0	Borer et al. (2007)
b_A	Annual birth rate	200	Seabloom et al. (2003)
σ_{P_S}	Healthy perennial (adult) survival rate	0.88	Malmstrom et al. (2005a)
σ_{P_I}	Infected perennial (adult) survival rate	0.77	Malmstrom et al. (2005a)
ϵ_p	Fractional reduction in biomass and fecundity of infected perennials	0.5	Malmstrom et al. (2005a)
ϵ_a	Fractional reduction in biomass and fecundity of infected annuals	0.11	Malmstrom et al. (2005a)
τ	Growing season length	20 weeks	Borer et al. (2007)
C	Number of aphids at beginning of growing season	100	
<i>Continuous time parameters</i>			
β_{vp}	Aphid to perennial transmission rate	0.2	
β_{va}	Aphid to annual transmission rate	0.4	
β_{pv}	Perennial to aphid transmission rate	0.02	Borer et al. (2007)
β_{av}	Annual to aphid transmission rate	0.04	Borer et al. (2007)
λ	Vector preference and performance (aphids per annual/aphids per perennial)	1.5	Malmstrom et al. (2005b)
μ_a	Annual death rate	1	Borer et al. (2007)
μ_p	Seedling perennial death rate	0.5	Borer et al. (2007)
μ_V	Aphid death rate	10	
d_V	Aphid fecundity rate	13.2	
m_{jk}	Aphid migration rate from patch j to patch k	1×10^{-5}	see text
<i>Competition parameters</i>			
α_{pp}	Competition between first-year perennials	1.3×10^{-3}	Borer et al. (2007)
α_{pa}	The effect on first-year perennials by annuals	6.8×10^{-4}	Borer et al. (2007)
α_{pP}	The effect on first-year perennials by adult perennials	0.7	Borer et al. (2007)
α_{aa}	Competition between annuals	1.1×10^{-3}	Borer et al. (2007)
α_{ap}	Effect on annuals of first-year perennials	3.4×10^{-7}	Borer et al. (2007)
α_{aP}	Effect on annuals of adult perennials	0.7	Borer et al. (2007)

also conduct sensitivity analyses by varying the different epidemiological parameters within the range exhibited by other grass species in field and laboratory studies. This allows us to examine how B/CYDV may control competitive outcomes depending on the composition of the host community.

For the estimation of the dispersal coefficient, we note that movement involves leaving, moving between, and arriving in patches and is notoriously complicated to estimate on very small animals, such as aphids. Therefore, we use simulations to

examine the effect of different aphid migration rates on B/CYDV transmission in a patch framework.

4 Analysis of a Two Patch Model

In order to better understand the dynamics of the full model that includes spatial heterogeneity, seasonality, competition, and disease dynamics, we consider the growing season dynamics in one and two patches. In particular, we find the basic reproduction number for the growing season dynamics in an isolated population and in two patches under an additional assumption about adult perennial death rates. Since the dry season dynamics decrease the number of infected organisms through the death of all infected annuals and infected aphids and the reduced survival of infected perennials, the basic reproduction number for the growing season will give us a good approximation of the initial spread of the virus.

4.1 Computation of the Basic Reproduction Number, R_0

In order for the basic reproduction number to be defined, we assume that there is a small death rate for perennial adults, d_P , during the growing season. Omitting a growing season death rate for adult perennials was a simplifying assumption of the non-spatial model in Borer et al. (2007). Based on numerical sensitivity analysis we choose appropriate values of d_P that do not significantly change the outcome of the model (see Fig. 4). This assumption changes only (1c) and (1d) to the following:

$$\frac{dP_{S,j}}{dt} = -\beta_{vp}V_{I,j}P_{S,j} - d_P P_{S,j}, \quad (6)$$

$$\frac{dP_{I,j}}{dt} = \beta_{vp}V_{I,j}P_{S,j} - d_P P_{I,j}. \quad (7)$$

We chose $d_P = 0.1$ so that, neglecting the dry season, the average lifespan of a perennial is 10 years.

With this additional assumption, we will use the next generation matrix method (van den Driessche and Watmough 2002) to determine the basic reproduction number for the one-patch case, or an isolated population. Let $X = (p_S, P_S, a_S, V_S, p_I, P_I, a_I, V_I)^T$. Then we can rewrite system (1a)–(1h) in the form

$$\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X), \quad (8)$$

where $\mathcal{F}(X)$ represents a vector function for the new infectious cases and $\mathcal{V}(X)$ contains all other dynamics. We compute the Jacobian of \mathcal{F} and \mathcal{V} and evaluate these at the disease free equilibrium (DFE), $E^* = (p_S^*, P_S^*, a_S^*, V_S^*, 0, 0, 0, 0)$. Let F and V be the matrices defined by

$$F = \left[\frac{\partial \mathcal{F}_i}{\partial x_j}(E^*) \right]; \quad V = \left[\frac{\partial \mathcal{V}_i}{\partial x_j}(E^*) \right], \quad (9)$$

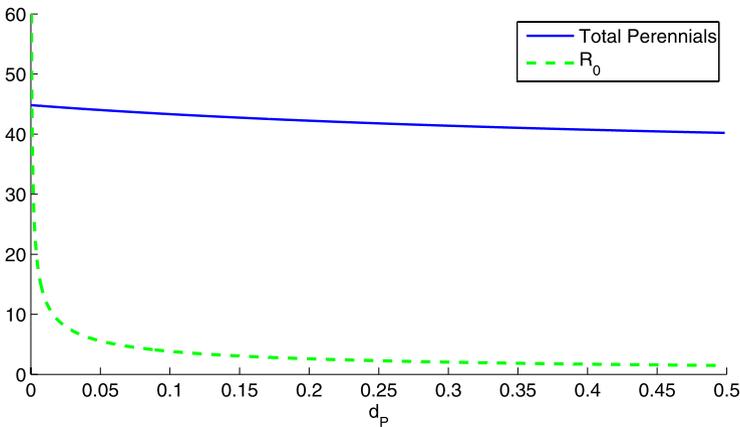


Fig. 4 We plot d_p versus R_0 (dotted line) and d_p versus total adult perennial equilibrium values (solid line) in the disease-free scenario for one patch. In this case, regardless of d_p , the disease-free equilibrium value for annuals is 0

where $5 \leq i, j \leq 8$ and x_j is the j th component of the vector X defined in (8). Computing these matrices we have

$$F = \begin{bmatrix} 0 & 0 & 0 & \beta_{vp}P_S^* \\ 0 & 0 & 0 & \beta_{vp}P_S^* \\ 0 & 0 & 0 & \beta_{va}a_S^* \\ \beta_{pv}V_S^* & \beta_{pv}V_S^* & \beta_{av}V_S^* & 0 \end{bmatrix}, \tag{10}$$

and

$$V = \begin{bmatrix} \epsilon_p \mu_p \eta_p(E^*) & 0 & 0 & 0 \\ 0 & d_p & 0 & 0 \\ 0 & 0 & \epsilon_a \mu_a \eta_a(E^*) & 0 \\ 0 & 0 & 0 & \mu_v \end{bmatrix}. \tag{11}$$

The basic reproduction number, $R_{0,1}$, is given as

$$R_{0,1} = \rho(FV^{-1}), \tag{12}$$

where $\rho(A)$ is the spectral radius of the matrix A . The spectral radius of the matrix FV^{-1} is given by the formula

$$R_{0,1} = \sqrt{R^{V_p}(R^p + R^p) + R^{V_a}R^a}, \tag{13}$$

where

$$R^p = \frac{\beta_{vp}P_S^*}{\epsilon_p \mu_p \eta_p(E^*)}, \tag{14a}$$

$$R^p = \frac{\beta_{vp}P_S^*}{d_p}, \tag{14b}$$

$$R^a = \frac{\beta_{va}a_S^*}{\epsilon_a\mu_a\eta_a(E^*)}, \quad (14c)$$

$$R^{V_p} = \frac{\beta_{pv}V_S^*}{\mu_V}, \quad (14d)$$

$$R^{V_a} = \frac{\beta_{av}V_S^*}{\mu_V}. \quad (14e)$$

For the baseline parameter values chosen, simulations indicate that in the disease free scenario perennials and annuals do not co-exist. When the disease free patch is perennial-only $R^a = 0$, and when the patch is annual-only then $R^p = R^P = 0$. The parameter $R_{0,1}$ is proportional in both cases to the transmission terms and the equilibrium populations of both vector and plants while varying inversely with vector and plant death rates. For seedling perennials and annuals, these death rates depend in part upon the reduction of biomass due to infection.

Next, we compute the basic reproduction number for the growing season in two patches. Let $E_1^* = (p_{S,1}^*, P_{S,1}^*, a_{S,1}^*, V_{S,1}^*, 0, 0, 0, 0)$, $E_2^* = (p_{S,2}^*, P_{S,2}^*, a_{S,2}^*, V_{S,2}^*, 0, 0, 0, 0)$ be the disease-free equilibrium for patch 1 and patch 2, respectively. Then, using the next generation method (Arino et al. 2005; Arino 2009),

$$F = \begin{bmatrix} F_1 & \mathbf{0} \\ \mathbf{0} & F_2 \end{bmatrix}, \quad (15)$$

where $\mathbf{0}$ denotes a 4×4 matrix of all zeros, and for $i = 1, 2$, the 4×4 matrices, F_1, F_2 are defined as

$$F_i = \begin{bmatrix} 0 & 0 & 0 & \beta_{vp}P_{S,i}^* \\ 0 & 0 & 0 & \beta_{vp}P_{S,i}^* \\ 0 & 0 & 0 & \beta_{va}a_{S,i}^* \\ \beta_{pv}V_{S,i}^* & \beta_{pv}V_{S,i}^* & \beta_{av}V_{S,i}^* & 0 \end{bmatrix}. \quad (16)$$

We also define the matrix

$$V = \begin{bmatrix} V_1 & M_{12} \\ M_{21} & V_2 \end{bmatrix}, \quad (17)$$

where for $i, j = 1, 2, i \neq j$,

$$V_i = \begin{bmatrix} \epsilon_p\mu_p\eta_p(E_i^*) & 0 & 0 & 0 \\ 0 & d_p & 0 & 0 \\ 0 & 0 & \epsilon_a\mu_a\eta_a(E_i^*) & 0 \\ 0 & 0 & 0 & \mu_V + m_{ji} \end{bmatrix}, \quad (18)$$

and

$$M_{ij} = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -m_{ij} \end{bmatrix}. \quad (19)$$

The basic reproduction number for the whole system, R_0^C , where $R_{0,i}$ is the basic reproduction number for patch i alone and assuming that migration is symmetric so

that $m_{12} = m_{21} = m$, is

$$R_0^C = \frac{1}{\sqrt{2}} \sqrt{R^* + (R_{0,1}^2 + R_{0,2}^2)(R_{\mu_V} + R_m)}, \tag{20}$$

where

$$R_{\mu_V} = \frac{\mu_V}{2m + \mu_V}, \tag{21}$$

$$R_m = \frac{m}{2m + \mu_V}, \tag{22}$$

and where

$$R^* = \sqrt{R_{\mu_V}(R_{0,1}^2 - R_{0,2}^2)^2 + R_m^2(R_{0,1}^2 + R_{0,2}^2)^2}. \tag{23}$$

We remark that if both patches are identical and inhabited by the same single species, then $R_{0,2} = R_{0,1}$. In addition, if migration of vectors between patches is very small so R_m is negligible and R_{μ_V} is close to one, then $R_0^C \approx R_{0,1}$. Thus, the basic reproduction number for the system simply becomes the basic reproduction number for the individual identical patches.

4.2 Sensitivity Analysis of R_0 to Parameters

In order to better understand why the native perennials are susceptible to invasion by non-native annuals in the presence of disease, we found the relative importance of all parameters to the initial spread of B/CYDV using R_0 for one and two patches. The initial successful spread of B/CYDV depends on R_0 for the growing season, while the endemic co-existence equilibrium values indicate long term persistence of the virus and thus long term co-existence of annuals and perennials. We therefore computed the sensitivity indices of R_0 to the parameters of the model in order to understand what factors are most important in disease prevalence and exotic invasion.

The sensitivity index $\frac{dR_0}{d\xi}$ is a linear estimate of the number of unit change in R_0 as a result of a unit change in the parameter ξ . Such sensitivity indices depend on the physical units of state variables and parameters, and hence we cannot compare different sensitivity indices. To make comparison feasible, and make the sensitivity analysis independent of the units of the model, we use normalized sensitivity indices as defined below.

Definition 4.1 A normalized sensitivity index for the state variable R_0 , with respect to the parameter ξ , denoted as $\psi_{R_0}^\xi$, is the ratio of relative change in R_0 , to the relative change in the parameter ξ , and is defined as (Saltelli et al. 2004; Chitnis et al. 2008):

$$\psi_{R_0}^\xi = \frac{\partial R_0}{\partial \xi} \cdot \frac{\xi}{R_0}. \tag{24}$$

The coefficient $\psi_{R_0}^\xi$, represents a linear estimate of the percentage change in the state variable R_0 caused by a one percent change in the parameter ξ .

Table 2 Normalized sensitivity indices for R_0 . Note that these rates are in 1/year units. The other parameters used are total time = 100, $\tau = 40/100$, $b_A = 200$, $b_P = 45$, $\mu_V = 10$, $\mu_a = 1$, $\mu_p = 0.5$, $d_V = 13.2$, $d_P = 0.1$, $\alpha_{aa} = 1.1 \times 10^{-3}$, $\alpha_{ap} = 3.4 \times 10^{-7}$, $\alpha_{aP} = 0.7$, $\alpha_{pp} = 1.3 \times 10^{-3}$, $\alpha_{pa} = 6.8 \times 10^{-4}$, $\alpha_{pP} = 0.7$, $\lambda = 1.5$, $\epsilon_a = 0.11$, $\epsilon_p = 0.5$, $\beta_{av} = 0.04$, $\beta_{pv} = 0.02$, $\beta_{vp} = 0.2$, $\beta_{va} = 0.4$, $\sigma_{P_S} = 0.88$, $\sigma_{P_I} = 0.77$, $m_{12} = m_{21} = 0.6$, 10 initial annuals for the annual-only, and 4 seedling perennials and 10 adult perennials for the perennial-only patch

Parameter	Annual-only patch	Perennial-only patch	One of each
R_0	343.09	4.8131	310.5801
Sensitivity Indices	$R_{0,1}$	$R_{0,2}$	R_0^C
ϵ_a	-4.5455	0	-4.4961
β_{av}	0.5	0	0.4946
β_{va}	0.5	0	0.4946
μ_V	-0.5	-0.5	-0.4696
μ_a	-0.5	0	-0.4946
α_{aa}	-0.3346	0	-0.3308
β_{pv}	0	0.5	1.2457×10^{-5}
β_{vp}	0	0.5	1.2457×10^{-5}
d_P	0	-0.4986	-1.2423×10^{-5}
ϵ_p	0	-0.0014	-3.4077×10^{-8}
μ_p	0	-0.0014	-3.4077×10^{-8}
α_{pP}	0	-0.0013	-3.2787×10^{-8}
α_{pp}	0	-4.4660×10^{-7}	-1.1127×10^{-11}
m_{12}, m_{21}	0	0	-0.0250

Since we have an analytic expression for R_0 for one and two patches, we can explicitly compute the sensitivity indices for R_0 with respect to all the parameters in our model. We evaluate the sensitivity indices at the baseline parameters (see Table 1) for our two patch model. Many of the sensitivity indices depend on the disease-free equilibrium population sizes of perennials and annuals (see the Appendix for an example), so changing demographic parameters will affect the indices.

The results of this sensitivity analysis are tabulated in Table 2. Based on these results we make the following observations. The sensitivity indices for two perennial (or annual) patches (not shown in Table 2) with very small migration rates are essentially the same as those for one perennial (or annual) patch (see Table 2). For the annual-only patches, $R_0 = R_{0,1}$ is most sensitive to ϵ_a , the fractional reduction in fecundity of infected annuals. If ϵ_a is increased by 10%, then R_0 is decreased by 45.46%. If the transmission rates between aphids and annuals, β_{av} or β_{va} , increase or decrease by 10% then R_0 increases or decreases by 5%. The sensitivity indices with respect to μ_V and μ_a , the death rate of aphids and annuals, are constant at -0.5 so that if μ_V or μ_a are increased by 10% then R_0 decreases by 5%. R_0 is just slightly less sensitive to α_{aa} , the competition coefficient between annuals, than it is to μ_V and μ_a . For this case, the sensitivity indices for α_{pa} and α_{Pa} are always zero since $p_S^* = P_S^* = 0$. In summary, the initial spread of B/CYDV in annual-only patches is most sensitive to the fractional reduction in fecundity of infected annuals, the disease

transmission rates between aphids and annuals, the death rates of aphids and annuals, and the competitive effect of annuals on each other, in the given order of importance.

In perennial-only patches, R_0 increases by 5% when either β_{pv} or β_{vp} increase by 10%. R_0 decreases by 5% when μ_V increases by 10%. R_0 also decreases by 4.98% when d_P , the growing season death rate of adult perennials, increases by 10%. Neither ϵ_p , μ_p , α_{pP} , nor α_{pp} have much effect on the value of R_0 (see Table 2). For this case, the sensitivity index for α_{ap} is always zero since $a_S^* = 0$. So, the initial spread of B/CYDV in perennials is most sensitive to the adult perennial death rate, disease transmission rates, and aphid death rate for perennial-only patches.

Next, we consider two patches that are not identical. When one patch is annual and one is perennial, the annual patch dynamics dominate the initial spread of the virus. R_0^C is most sensitive to ϵ_a as with the annual-only patches. In fact, if ϵ_a is increased by 1%, R_0^C decreases by 4.5%. The rest of the parameters related to annual plants are relatively less important (see Table 2) while the parameters related to perennials have little effect on the value of R_0^C . In Table 2, the migration rate used is rather high. When the migration rate is lower, the perennials are even less significant to the initial spread of disease in the two patch system. The dominant role of annuals in initial spread of B/CYDV is due to their higher density, higher transmission rates, and aphid preference for and increased fecundity on the annual species we are considering. Ultimate persistence of the virus, however, depends almost entirely on the perennials since disease is not maintained in annuals during the dry season and there is no vertical transmission.

4.3 Numerical Simulations for the Two Patch Model

For our baseline values, if disease is present, the exotic and native species will be able to co-exist, in part due to a basic reproduction number for perennial-only patches greater than one. It may be important for conservation and restoration design to consider the case when a patch of perennials would not support the virus alone but faces invasion by an exotic and competent reservoir for B/CYDV.

We performed numerical simulations for two patches, one perennial only, and one annual only patch. Perennial disease transmission rates are kept small enough so that a perennial-only patch will have a basic reproduction number less than one ($R_{0,1} < 1$) when there is no migration of vectors between patches. The nearby annual-only patch has baseline annual-aphid transmission rates resulting in a high basic reproduction number, $R_{0,2}$, for that patch. Since we are concerned primarily with the presence of disease in the perennial patch, we consider the sensitivity of $R_{0,1}$ to aphid migration rates. However, (20) indicates that the basic reproduction number for the whole system is proportional to $R_{0,1}$ and $R_{0,2}$ so when $R_{0,1}$ increases, R_0^C increases as well. When migration of vectors is symmetric between the perennial patch and the annual patch, the perennial patch basic reproduction number, $R_{0,1}$ increases with the increase of migration and quickly moves above one. When vectors migrate only from the annual to the perennial patch, $R_{0,1}$ for the perennial patch increases even more quickly and is slightly more sensitive to the migration rates. For both of these cases, $R_{0,1}$ increases as the migration rates of the vectors increase. Thus, movement of vectors between the annual and perennial patches results in persistence of the pathogen

in perennials that would not otherwise occur. When vectors migrate only from perennial to annual patch, however, the perennial patch basic reproduction number $R_{0,1}$ decreases slightly as migration increases while the basic reproduction number for the annual patch remains virtually unchanged. These results confirm that the disease dynamics of the annual patches dominate the initial spread and success of the virus and migration behavior of vectors can in fact change the disease dynamics of a perennial-only patch.

5 Large Scale Numerical Simulations

For our larger scale numerical simulations, we examined the full spatial model with 20 patches arranged linearly, as shown in Fig. 5. Initial simulations with this model were conducted using within-patch transmission rates high enough for annual grasses to increase in abundance in infected mixed-host patches (see Table 1 for initial parameter values). Under a global connectivity scenario (identical aphid migration rates between each patch), every patch becomes infected with B/CYDV, even when aphid migration rates are very low ($\leq 10^{-12} \text{ yr}^{-1}$). Under this scenario, B/CYDV prevalence is so high in annual-only, perennial-only, and mixed-host patches (even when migration rates are very low) that increasing the migration rate does not have a large effect on prevalence. In addition, varying the proportion of patches occupied by perennial grasses does not significantly affect B/CYDV persistence or prevalence; prevalence

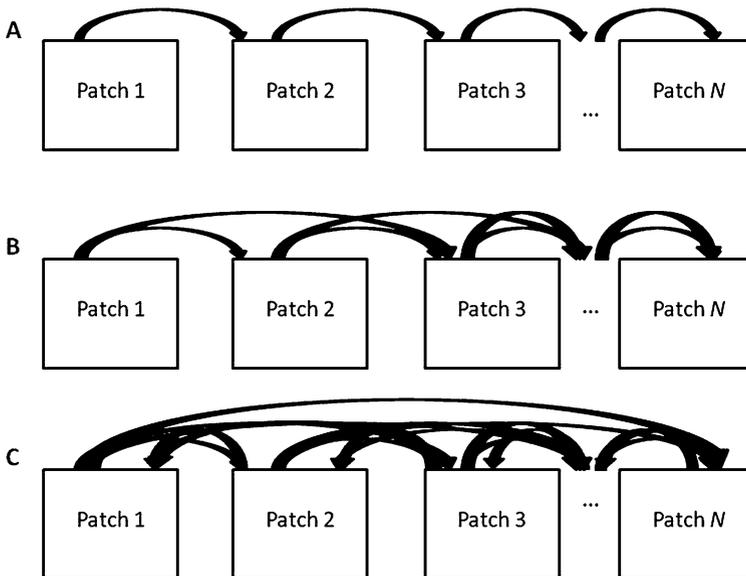


Fig. 5 Linear arrangement of 20 patches used in numerical simulations. Three scenarios included are: (a) aphid migration is unidirectional and to the next patch only, (b) aphid migration is unidirectional and to the two nearest patches, and (c) aphids migration occurs between every patch at the same rate (global connectivity scenario)

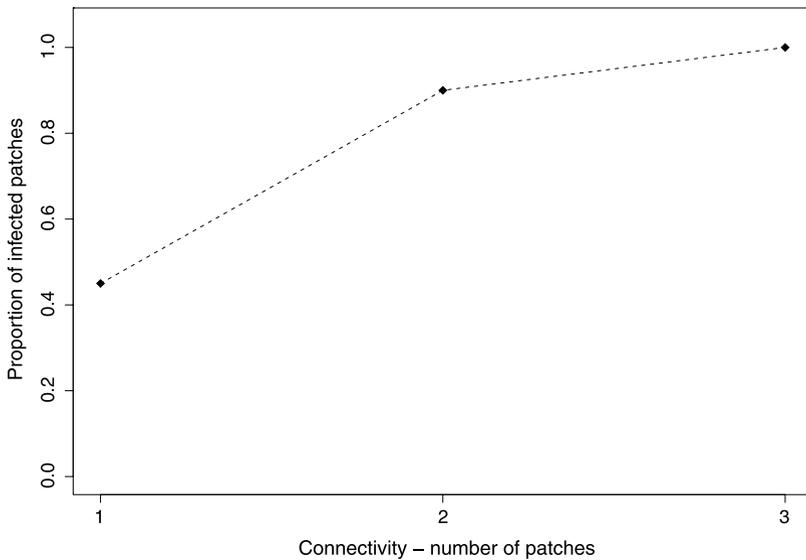


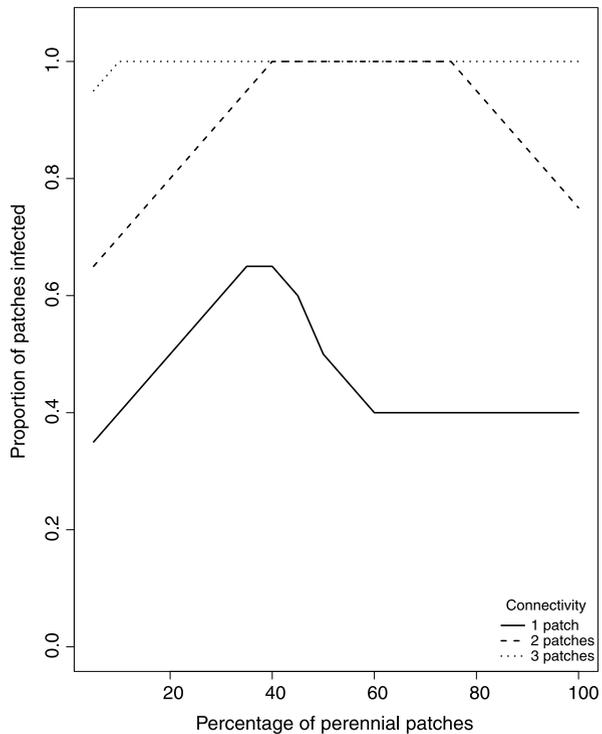
Fig. 6 Proportion of 20 patches that are infected with B/CYDV as a function of the number of neighboring patches that are connected via aphid migration (1, 2, or 3). Figure shows simulation results for scenario where patches contain either all perennial or all annual patches. *Points* represent the mean proportion of patches infected when the percentage of patches contains perennial grasses is varied from 5% to 100%

in each perennial patch remains constant and prevalence in annual patches is 100% by the end of each growing season.

When aphid migration is not global, increasing patch connectivity leads to an increase in the number of infected patches when the simulations are allowed to run to long-term equilibrium (Fig. 6). Patch connectivity is determined by the number of neighboring patches an aphid can reach when it emigrates from a patch (Fig. 5). Connectivity is unlikely to be global because aphid dispersal between patches is limited by distance and landscape heterogeneity. An increase in patch connectivity could be the result of either a decrease in the distance between patches or an increase in the distance that individual aphids can travel. As the number of infected patches increases, B/CYDV prevalence at the regional level increases. However, patch-level prevalence depends only on the distance of the patch from the initial source of infection and not the regional-level prevalence.

When each patch is connected with only one or two neighboring patches (in a linear chain), increasing the proportion of perennial-only patches leads to a linear increase in the proportion of infected patches (assuming that all perennial patches are initially infected). However, if we start with B/CYDV in a single source patch, the relationship between the proportion of perennial-occupied patches and B/CYDV prevalence is nonlinear over intermediate timeframes (Fig. 7). Initial increases in the proportion of perennial patches increase global B/CYDV prevalence by increasing the number of infected patches; but above a threshold, increasing the number of perennial patches leads to a decline in the number of infected patches over the first

Fig. 7 The proportion of infected patches as a function of the percentage of patches containing perennial grasses when aphids can migrate to the nearest 1, 2, or 3 patches

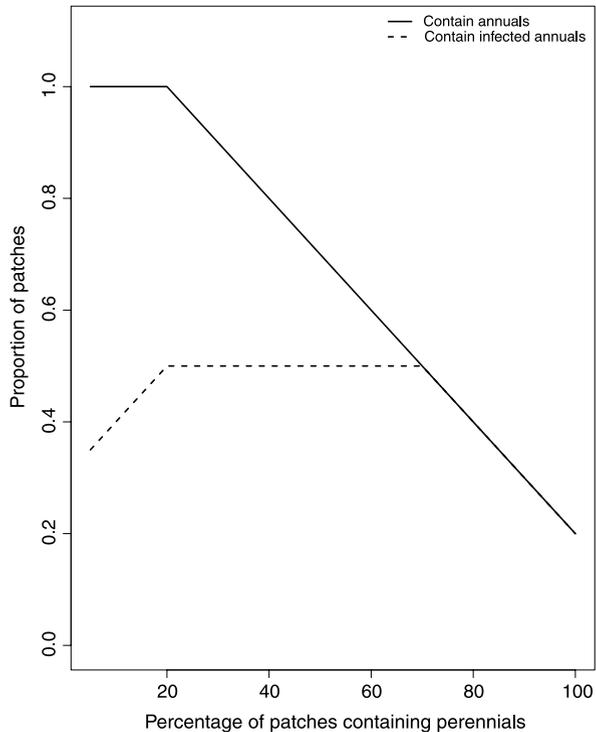


50 years of the simulation. Although when the simulation is allowed to run to equilibrium, all perennial patches eventually become infected, it can take 50+ years for B/CYDV to reach the furthest patches from the initial source of infection. This occurs because in mixed host patches further away from the initial source of infection the perennial grasses out-compete annual grasses before B/CYDV can arrive in the patch. In this case, increasing the proportion of perennial grasses leads to a decline in the regional level presence of annual grasses (Fig. 8). The lower transmission rates to and from perennial grasses reduces the number of patches to which B/CYDV can travel within the growing season.

Figure 8 also shows that the ability of annuals to co-exist with perennials depends on the distance of the patch from initial source of infection as well as the proportion of perennials in the patch system. Only patches within 3 jumps of the initial source of infection receive infected aphid immigrants early enough in the growing season for annuals to overcome their competitive inferiority in the absence of the pathogen. However, simulations of the invasion by annuals in perennial only patches reveal that annuals can invade perennial patches once B/CYDV has become established in the patch. Thus only perennial patches that remain disease-free because they are not connected to any other patches via migration, or are only distantly connected to other perennially infected patches, will remain resistant to invasion from annual grasses.

B/CYDV prevalence at the patch and regional levels is higher when there are mixed patches containing both host species rather than patches containing either all annual or all perennial grasses. The higher transmission rates associated with annual

Fig. 8 Proportion of 20 initial patches that contain annual grasses at equilibrium as a function of the number of patches containing perennial grasses in the presence of B/CYDV. Each patch initially contains either annual grasses only, or a mixture of annuals and perennials. B/CYDV is initially present in a single mixed host patch and subsequently spreads via aphid migration



grasses lead to higher prevalence levels in juvenile perennial grasses in mixed patches compared to perennial-only patches (85% versus 60% at equilibrium). In addition, the average abundances of both annual grasses and perennial grasses are reduced in mixed patches. Perennial biomass is reduced by 4.5% and 20.5% in adults and juveniles respectively, and annual biomass can be reduced by up to 30% depending on prevalence.

Thus, we can conclude from our numerical simulations that if invasion by annuals grasses occurs in a patch system where B/CYDV has already established in perennial grasses then annuals will be able to invade all infected patches. However, if the introduction of B/CYDV and annual grasses occurs simultaneously, then the success of the annuals will depend on where within the patch system they invade relative to the introduction of B/CYDV. While annuals will be able to invade patches close to the initial site of infection, they will fail to invade patches further from the infection source because they will be out-competed by perennials before B/CYDV becomes prevalent enough in the patch to counter their competitive inferiority. However, these perennial patches will become susceptible to annual invasion over time as they reach an equilibrium-level of B/CYDV prevalence.

On the west coast of the US, fragmented grassland habitat is arranged along both a north-south and an elevational gradient. Although grasslands in California are dominated by invasive annuals, grasslands in Oregon and at higher elevations tend to remain perennial-dominated. Our sensitivity analysis showed that R_0 is more sensitive to aphid migration rates when transmission rates are lower than predicted for Cali-

ifornia grasslands. This suggests that a combination of lower B/CYDV transmission rates in perennial-dominated populations and a shorter growing season could prevent annuals from establishing in these populations at a higher latitude or elevation. The simulation results also show that if B/CYDV were initially introduced into a population in Southern California, annuals would be out-competed at the northern end of the range before the virus became prevalent enough in those populations. The results of the sensitivity analysis suggest that these northern perennial populations could then remain resistant to invasion by annuals if transmission rates were low enough to prevent annual reintroduction.

6 Discussion and Conclusions

The landscape-scale composition and configuration of host communities, along with vector movement patterns among patches, are essential determinants of pathogen spread and prevalence in fragmented landscapes (Ostfeld et al. 2005; Plantegenest et al. 2007). Pathogen spread depends on host composition (e.g., presence of reservoirs, probability of transmission) and vector density and dispersal, all of which can vary among patches in a complex landscape. Management can increase connectivity, elevating transmission of multi-host pathogens, as is the case with fire suppression increasing connectivity among hosts susceptible to sudden oak death (*Phytophthora ramorum*; see Meentemeyer et al. (2008)). Landscape-scale host composition also influences the dynamics of many multi-host pathogens, including spread of sudden oak death in California oaks (Condeso and Meentemeyer 2007), Lyme disease prevalence (Buskirk and Ostfeld 1998; Allan et al. 2003; Brownstein et al. 2005), West Nile virus dynamics (Allan et al. 2009), hantavirus prevalence in rodents (Langlois et al. 2001), and the spread of foot and mouth disease (Keeling et al. 2001). Here, we have shown that the spatial configuration of the patch system, host composition within patches, and patch connectivity affect not only the ability of B/CYDV to invade a fragmented system, but also determine whether the pathogen facilitates the invasion of a non-native host species. Below we discuss these three factors in the context of the analysis conducted for our focal B/CYDV system, and we make broad observations and conclusions that could apply to other similar systems.

The spatial structure of host populations can influence the spread of infectious disease, as well as the spatial pattern of disease prevalence. Here, we have shown that the spatial configuration of the host community can interact with the timing of pathogen and invasive host arrival to determine the ability of the pathogen to invade local populations and influence competition between annual and perennial grass species. In our numerical simulations, long term pathogen persistence and prevalence depended on the abundance of perennial grasses in a patch system, with increasing perennial patch occupancy generally leading to an increase in B/CYDV prevalence at the regional level because perennial patches serve as a long-term pathogen reservoirs, whereas annual-only patches do not maintain the pathogen between growing seasons. However, high proportions of perennial patches can slow the spread of B/CYDV during the growing season because both aphid fecundity and transmission rates are lower for

perennial grasses than annuals. Thus, mixed species patches or mixtures of patches with differing host composition tend to have the highest prevalence rates because of the balance among pathogen residence time, pathogen transmission probability, and vector fecundity.

The viral-induced reduction in annual host fecundity was the most important factor controlling the successful initial spread of B/CYDV in mixed populations of annuals and perennials and in perennial-only populations that were connected to annual populations via aphid dispersal. Because annual hosts are superior to perennials for vector fecundity in this system, the suppressive effect of infection on annuals and the amplification via vector density interact to control the rate of pathogen spatial spread. The baseline value for B/CYDV's effect on annual fecundity was based on observations of the exotic annual *Bromus hordeaceus* in California grasslands. However, the impact of the virus on host fecundity will vary by host species and also be mediated by environmental conditions (Seabloom et al. 2009). Therefore, in our focal system, B/CYDV prevalence levels in perennial populations will depend on both the identity of nearby annual grasses as well as the environmental conditions experienced by the annuals. If the reduction in annual fecundity is higher than we estimated, annual grasses may not be able to invade perennial populations. More generally, host composition can feed back to control pathogen spread rates in a patchy system.

Spatial connectivity can control both dispersal rates and local species densities. In animal communities, both hosts and vectors can move among patches; however, in our focal community, among-patch host movement is negligible, whereas vector dispersal is key. Landscape-scale host composition can interact with vector dispersal to control disease spread and epidemics, as in our case study. Similarly, bean dwarf mosaic virus, a whitefly-transmitted virus that infects both soybeans and common bean plants causes severe disease in the latter. In Argentina, increased soybean acreage shifted the landscape-scale host composition, leading to the emergence of bean dwarf mosaic virus and threatening local common bean production (McCormack 2006). In multi-host communities where hosts also move among patches, this will add further complexity that warrants future exploration. In mixed-host communities, hosts vary in infection tolerance and probability of transmission; our results suggest that this variation can interact with patch connectivity to affect pathogen persistence and prevalence. This has important implications for both conservation and understanding species invasions. For example, increased patch connectivity, a common management scheme for endangered species, can lead to increased pathogen transmission and prevalence (Hess 1994, 1996). Our results suggest that this is especially true for mixed-host communities. If hosts differ strongly in their pathogen tolerance, a less tolerant species of concern could be driven to extinction in a highly connected landscape (Holt et al. 2003), particularly by a vector-transmitted disease (de Castro and Bolker 2005).

Species invasions can shape the composition of communities, species co-existence, and bio-diversity in fragmented patch systems, and pathogens have been implicated in species invasions in the B/CYDV system and others. For example, in the United Kingdom, the invasive grey squirrel and an introduced parapox virus are causing declines in the native red squirrel population through resource competition and pathogen-mediated apparent competition (Rushton et al. 2000;

Tompkins et al. 2003). In the B/CYDV system, co-existence of annual and perennial grasses requires B/CYDV to reduce the competitive advantage of perennial grasses (Borer et al. 2007). Our current results modify this non-spatial understanding. In the context of a fragmented patch system, the ability of annuals to invade depends on the timing of invasion with respect to the introduction of disease, the spatial locations where these invasions happen, and the composition and configuration of the patch system. Thus, our current results suggest that connectivity can interact with arrival time and host infection tolerance to determine the success or failure of an invasion.

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Appendix

We computed the sensitivity indices analytically before evaluating them numerically. Note that p_S^* , P_S^* , V_S^* are the equilibrium values of susceptible seedling perennials, adult perennials, and vectors for the growing season dynamics. These equilibrium values are computed numerically by running the numerical simulation for 100 years and using the values for each group at the end of the growing season. Following is one normalized sensitivity index as an example:

$$\psi_{R_{0,1}}^{\beta_{pv}} = \frac{\beta_{pv}}{R_{0,1}} \frac{1}{2R_{0,1}} \frac{V_S^*}{\mu_V} \left(\frac{\beta_{vp} P_S^*}{\epsilon_p \mu_p (1 + \alpha_{pp} P_S^* + \alpha_{pP} P_S^*)} + \frac{\beta_{vp} P_S^*}{d_P} \right). \quad (25)$$

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