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Barley and cereal yellow dwarf viruses (B/CYDV) are a suite of aphid-vectored pathogens that affect diverse host communities, including economically important crops. Coinfection of a single host by multiple strains of B/CYDV can result in elevated virulence, incidence, and transmission rates. We develop a model for a single host, two pathogen strains, and n vector species, modeled by a system of nonlinear ordinary differential equations. A single parameter describes the degree of relatedness of the strains and the amount of cross-protection between them.

We compute the basic and type reproduction numbers for the model and analytically prove the (conditional) stability of the disease-free equilibrium. We demonstrate numerically that, although the basic reproduction number describes stability of the disease-free equilibrium, the type reproduction numbers better describe the individual behavior of each strain and the dynamics of coinfection. We then conduct a sensitivity analysis on the components of the endemic equilibrium for two different vector growth functions. Our results indicate that the disease transmission rates and the vector birth and mortality rates are the most influential parameters for the equilibrium prevalences of infection and coinfection.

Keywords: Barley yellow dwarf virus, coinfection, basic reproduction number, type reproduction numbers, sensitivity analysis

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A Vector–Host Model for Coinfection by Barley/Cereal Yellow Dwarf Viruses

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I understand that my project will become part of the permanent collection of Oregon State University, University Honors College. My signature below authorizes release of my project to any reader upon request.

Margaret-Rose W. Leung, Author

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1 Introduction

Barley and cereal yellow dwarf viruses (B/CYDV) are a suite of aphid-vectored pathogens. These viruses are known to cause disease in over 150 host species, including commercially valuable grasses and cereal crops such as barley, wheat, and oats [14,18,26]. Globally, they are some of the most prevalent and most economically important crop pathogens [4, 14, 21]. Infected crops have lowered yields, increased mortality, lowered seed production, yellowed foliage, and stunted growth [4, 8, 24]. Estimates for the yield losses caused by B/CYDV range from 11 to 33% and losses of 86% have been recorded [18]. In the wild, B/CYDV is able to reverse the competitive advantage of native Californian perennial grasses over exotic annuals [4].

Transmission of B/CYDV occurs solely between the vector and the host [24]. Infected aphids cannot directly infect other aphids, and infected grasses cannot transmit the disease to other grasses either horizontally or vertically through seed production. Therefore, in communities of annual hosts or in crop settings, B/CYDV cannot remain endemic unless a viral reservoir is available in the form of a perennial host [4, 25].

After initial infection, both hosts and vectors experience a latency period varying between 1 and 14 days, depending on temperature [21]. Host recoveries are generally rare and vectors remain infectious for life [21].

Research into this disease is frequently motivated by its role in crop production and so often only a single host species is examined. However, the community of pathogens that cause it is diverse and is recently becoming a topic of greater interest. Barley and cereal yellow dwarf viruses belong to distinct genera. Cross–protection occurs between BYDVs but not between CYDVs or between BYDVs and CYDVs [31].

Coinfection by both BYDV and CYDV can increase virulence, incidence, mortality, and disease transmission rates above those for either virus in isolation [20, 26]. Thus, cross–protection from infection by viruses from the same genus should theoretically lower the prevalence of coinfections, but field data has not yet supported this [26].

1.1 Mathematical Disease Modeling and Coinfection

Traditional models in mathematical epidemiology focus on a single pathogen affecting a single host community. The simplest example is the compartmental SI model, which divides the hosts into susceptible (S) and infected (I) types and describes the movement of the population between these classes using systems of nonlinear ordinary differential equations (ODEs). Standard variations on the SI model include the SIR model, which adds a recovered (R) or removed class into which infected individuals can move, and the SIS and SIRS models, which model reinfection by allowing infected or recovered hosts respectively to return to the susceptible class [9].

Disease transmission is the most important process in host-pathogen models [19]. In disease models, the *force of infection*, defined as the probability per unit time that a given susceptible individual becomes infected, is key in establishing threshold quantities such as the basic reproduction number [9] and type reproduction numbers [12] that determine the dynamics of infection. There are several approaches to modeling the transmission of disease that may or may not depend on host and vector densities/numbers. The two most common approaches are called density–dependent disease transmission (cf. Appendix A) and standard–incidence disease transmission. In this thesis we use density–dependent disease transmission, meaning the force of infection is proportional to the numbers of susceptible and infected hosts and vectors. This is due to the fact that our model is a generalization of the model by Seabloom et. al. [26] which assumes density–dependent disease transmission.

None of these models incorporate more than a single type of infection. However,

in nature, hosts are exposed to hundreds of pathogens and field studies have demonstrated that multiple infections (coinfection) are a relatively common occurrence and most hosts support multiple parasites [16, 27]. Coinfection refers to the simultaneous infection of a single host by multiple pathogens or pathogen strains [26]. The classical theory of competitive exclusion states that no two species can indefinitely share the same ecological niche [1]. However, the significance of inter-pathogen interactions is recently becoming more apparent [1, 16, 30]. Pathogen interactions can increase host mortality [16] or transmission rates; for example, by lowering host resistance and allowing coinfected hosts to serve as superspreaders [26]. Conversely, infected hosts may have increased resistance due to a more active immune system or cross-protection conferred against related pathogens [26].

Coinfection may have consequences for entire host ecosystems. Several recent studies have explored its implications and conditions under which it is sustainable.

Alizon [2] considered the invasion of a mutant viral strain into a stable system with an endemic strain already present. In most cases, only a single strain persists; however, if cross-protection reduces the overall virulence (the net pathogen-induced host mortality) below the virulence of each coinfecting strain, then coexistence between two pathogen strains can occur as a stable state.

Allen et al. [3] examined an SI model for a single host affected by two pathogens: hantavirus and arenavirus. Both are transmitted horizontally via standard–incidence [9, 19]; arenavirus may also be transmitted vertically. The authors determine that if cross-protection is not complete and both viruses may simultaneously infect a single host, then stable pathogen coexistence and coinfection is possible.

1.2 Applications

The dynamics and epidemiology of barley and cereal yellow dwarf viruses has been the topic of several modeling efforts. Borer et al. [4] constructed a nonspatial B/CYDV

model consisting of a continuous–time rainy season and discrete time dry season. Perennial grasses were divided between first-year seedlings and adult plants. The aphid population was not explicitly modeled; instead, it was assumed to depend entirely on the infected plant biomass. B/CYDV was assumed to reduce fecundity and increase dry season mortality of the perennials, which persist over the summer in a dormant stage. Using this model, the authors were able to show that the presence of B/CYDV allowed invasive annual grasses to replace native perennial grasses.

Moore et al. [21] examined the effect of spatial dynamics on the spread of B/CYDV by adding a network of connected patches. As in Borer et al. [4], both annual and perennial grasses were included, and a differential-difference approach was used to capture varying dynamics in the growing and dormant seasons. A mathematical analysis was conducted for the two-patch case, including a derivation of the basic reproduction number and a sensitivity analysis of this number with respect to the model parameters. Numerical simulations were used to explore both the two-patch case and a larger network of 20 linearly arranged patches. Based on this analysis, the authors conclude that the patches' spatial configuration, host composition, and connectivity determine the pathogen's invasive ability and its ability to facilitate invasion of the system by exotic annual grasses [21], as predicted by Borer et al. [4].

Although we focus our investigation on B/CYDV, the methods are relevant to many other vector-borne diseases, such as the whitefly-transmitted African cassava mosaic virus [15] and the mosquito-transmitted dengue virus.

There are four strains of dengue virus; infection by one confers immunity against that particular strain but only temporary cross-immunity to the others [11]. Katri [17] examined several dengue models, beginning with an SIR-type model for a single strain of dengue combined with an SI model for the vector. Although the vector population was assumed constant, it had explicit age structure. Seasonal, spatial, and multi-strain variations on this model were also considered; in particular, the latter included

two strains of dengue. In this model, coinfection was impossible due to the assumption of complete cross-protection while infected, but recovered hosts were susceptible to the strain by which they were not infected. In order to prevent a multi-strain epidemic, controlling susceptibility to secondary infection was found to be critical.

Feng and Velasco-Hernandez [11] also analyzed a two-strain dengue model, but assumed variably sized host and vector populations. Recovered hosts experienced a temporary period of incomplete cross-immunity before becoming fully susceptible. In this model, competitive exclusion occurred, and the pathogen coexistence equilibrium, with both viruses persisting, was unstable.

1.3 Objectives

We are interested in examining the dynamics of coinfection by B/CYDV: the conditions under which it is possible and how its prevalence varies with respect to the parameters governing the host, vector, and pathogen dynamics.

We focus our analysis on a single host species. Since the economic importance of B/CYDV is primarily due to its role as a crop pathogen, it is reasonable to examine a homogeneous host community. However, within a single population, multiple species of aphid vectors may be active and transmit B/CYDV. In California alone, at least seven vector species are known to transmit B/CYDV [26].

1.4 Methods

Our proposed model is closely related to the model of Seabloom et al. [26], who consider two pathogen strains, two vector species, and a single host. The host is divided into susceptible, coinfected, and two singly-infected compartments. Coinfection in the vector is not considered and the vector population is not tracked explicitly. The population of each vector species infected with each strain is assumed to be at its quasiequilibrium value [13] (also called the pseudoequilibrium, see Appendix B), as-

suming a constant total vector population. The model is fitted to B/CYDV data and explored computationally, showing that coinfection is most prevalent with generalist vectors and weak cross-protection and coinfection-induced mortality.

Our paper differs from that of Seabloom et al. [26] in the following respects. First, our model allows varying numbers of vector species, using a structure similar to that of Ackleh and Allen [1], who examined competitive exclusion in an SIR model for j pathogen strains directly transmitted to a host population via density–dependent disease transmission. However, in their model, complete cross-immunity was assumed, prohibiting coinfection.

Second, we explicitly model vector populations rather than assuming fixed total vector populations and using the quasiequilibrium argument to reduce the model to four host equations. We formulate our initial model with general functions representing host and vector birth rates.

The outline of this paper is as follows. In Section 2, we introduce the model for n vector species, including suggested birth functions for the host and vector. This system is then rescaled to model the proportions of each population that is infected. The case of $n = 1$ vector species, assuming a constant vector growth rate, is examined in Section 3, including both the disease-free and coinfected equilibria. We compute the basic and type reproduction numbers of the pathogen strains and conduct a sensitivity analysis of the coinfected equilibrium. We also provide simulations depicting the system dynamics and disease prevalances for varying parameters and initial conditions. An alternative logistic-type vector birth rate is examined in Section 4, including a second sensitivity analysis of the coinfected equilibrium. Section 5 discusses conclusions of the analysis and future work. Finally, we provide a glossary of terms in Appendix A and relate the explicit vector equations in our model to the implicit equations used by Seabloom et al. [26] in Appendix B.

2 Vector–Host Model with Multiple Vectors

Our model is based on that developed by Seabloom et al. [26] and includes a single host species, n vector species, and two strains of BYDV. We consider coinfection in the host, but not the vector, and model each vector population explicitly.

We divide the host species between four classes, representing susceptible (S), infected by strain A (I_A), infected by strain B (I_B), and coinfected (I_{AB}) populations. We denote the total population N, where $N(t) := S(t) + I_A(t) + I_B(t) + I_{AB}(t)$, with t denoting time. This gives the system of nonlinear ODEs shown below.

Hosts:

$$\frac{dS}{dt} = g(S, I_A, I_B, I_{AB}) - \beta_H S \sum_{i=1}^{n} (V_{i,A} + V_{i,B}) - \mu_0 S, \tag{1a}$$

$$\frac{dI_A}{dt} = \beta_H \sum_{i=1}^{n} (S V_{i,A} - \psi I_A V_{i,B}) - (\mu_0 + \mu_1) I_A, \tag{1b}$$

$$\frac{dI_B}{dt} = \beta_H \sum_{i=1}^{n} (S V_{i,B} - \psi I_B V_{i,A}) - (\mu_0 + \mu_1) I_B, \tag{1c}$$

$$\frac{dI_{AB}}{dt} = \beta_H \psi \sum_{i=1}^{n} (I_A V_{i,B} + I_B V_{i,A}) - (\mu_0 + \mu_1 + \psi\mu_2) I_{AB}. \tag{1d}$$

Each vector species i, for $i = 1, \ldots, n$, is divided into three classes: susceptible ($V_{i,S}$), infected by strain A ($V_{i,A}$), and infected by strain B ($V_{i,B}$).

Both strains have the same transmission rate (β_H) from vector to host. However, the cross-protection factor ($\psi \in [0, 1]$) may reduce or prevent transmission of the second strain to singly infected hosts. For similar viral strains, $\psi \to 0$, resulting in high levels of cross-protection. For very different strains, $\psi \to 1$, resulting in little or no cross-protection (though for some diseases like dengue ψ may be greater than 1).

Hosts have a birth function g and experience a base mortality rate (μ_0). Infection by either strain results in the same additive increase to mortality (μ_1). Coinfected

hosts experience an additional additive mortality rate (μ_2), which is scaled by the cross-protection factor ψ. Thus, coinfection by highly similar viral strains causes little additional mortality, whereas coinfection by highly distinct strains causes greater additional mortality. We assume $\mu_0 \geq \mu_1 \geq \mu_2 \geq 0$.

We assume disease transmission is density dependent and model it using density–dependent disease transmission, rather than standard–incidence disease transmission. This is primarily because our model is based on that proposed by Seabloom et al. [26] which also uses density– dependent disease transmission, and this type of transmission has been used to model BYDV previously [4].

The total population size of the host is then described by

$$\frac{dN}{dt} = g(S, I_A, I_B, I_{AB}) - \mu_0 N - \mu_1(N - S) - \psi\mu_2 I_{AB}. \tag{2}$$

The lifespan of the aphid is approximately one month [26]; this is assumed sufficiently short such that the prevalence and effect of coinfection in the vector is negligible. We also assume that any additional mortality due to infection is negligible, due to the aphids' short lifespan and to the stronger regulatory effect of predators and seasonality [26].

We let r denote the vector birth function, which gives the following system of equations for each vector species.

Vectors:

$$\frac{dV_{i,S}}{dt} = r(V_{i,S}, V_{i,A}, V_{i,B}) - \beta_{V_{i,A}} V_{i,S}(I_A + I_{AB}) - \beta_{V_{i,B}} V_{i,S}(I_B + I_{AB}) - \mu_V V_{i,S}, \tag{3a}$$

$$\frac{dV_{i,A}}{dt} = \beta_{V_{i,A}} V_{i,S}(I_A + I_{AB}) - \mu_V V_{i,A}, \tag{3b}$$

$$\frac{dV_{i,B}}{dt} = \beta_{V_{i,B}} V_{i,S}(I_B + I_{AB}) - \mu_V V_{i,B}. \tag{3c}$$

The infection rates of each strain ($\beta_{V_{i,A}}$, $\beta_{V_{i,B}}$) may be different for each vector species.

However, we assume here that single infections of each type and their further coinfections are equally transmissible.

We denote the total vector population $N_{V,i}$, where $N_{V,i} = V_{i,S} + V_{i,A} + V_{i,B}$. This population size is described by the equation

$$\frac{dN_{V,i}}{dt} = r(V_{i,S}, V_{i,A}, V_{i,B}) - \mu_V N_{V,i}, \tag{4}$$

so by choosing r to be a function of the total population $N_{V,i}$ only, infection does not affect the vital rates of the vector species.

For the case $n = 1$, the birth and death dynamics of the model are shown in Figure 1 and the model's disease dynamics are shown in Figure 2. With additional vector species ($n \geq 2$), the host would experience separate rates of infection corresponding to each vector. The remaining dynamics would be unchanged in both diagrams.

2.1 Suggested Birth Functions

We use the following birth rates for hosts and vectors respectively:

$$g(S, I_A, I_B, I_{AB}) = \left[b_0 S + b_1(I_A + I_B) + b_2 I_{AB}\right]\left(1 - \frac{N}{\theta}\right), \tag{5}$$

$$r(V_{i,S}, V_{i,A}, V_{i,B}) = b_V N_{V,i}, \tag{6}$$

where $b_0 \geq b_1 \geq b_2 \geq 0$. Thus, uninfected hosts reproduce at rate b_0, hosts infected by one strain reproduce at a reduced rate b_1, and coinfected hosts reproduce at a rate reduced further to b_2. We define the maximum growth rate of the susceptible host population to be $r_0 := b_0 - \mu_0 > 0$. In Section 4, we consider a logistic growth rate for the vector population.

In the absence of disease, the host population grows logistically towards its carrying capacity. Since the host's birth and death rates are modeled separately, this

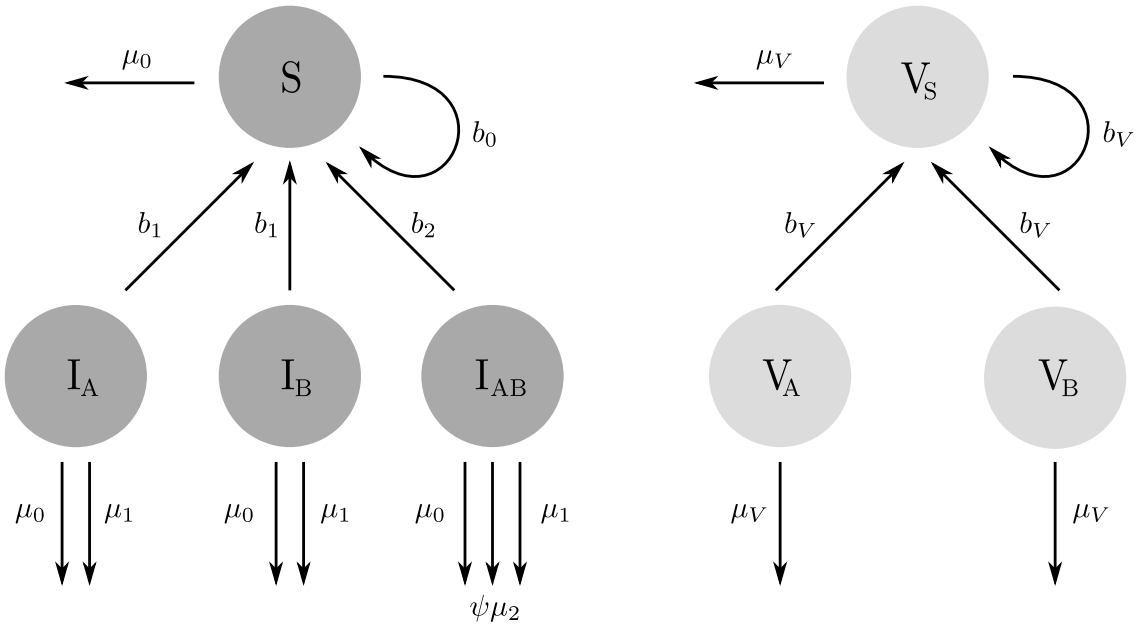

Figure 1: Diagram of the birth and death dynamics of the system (1) and (3). Left: host. Right: each vector species.

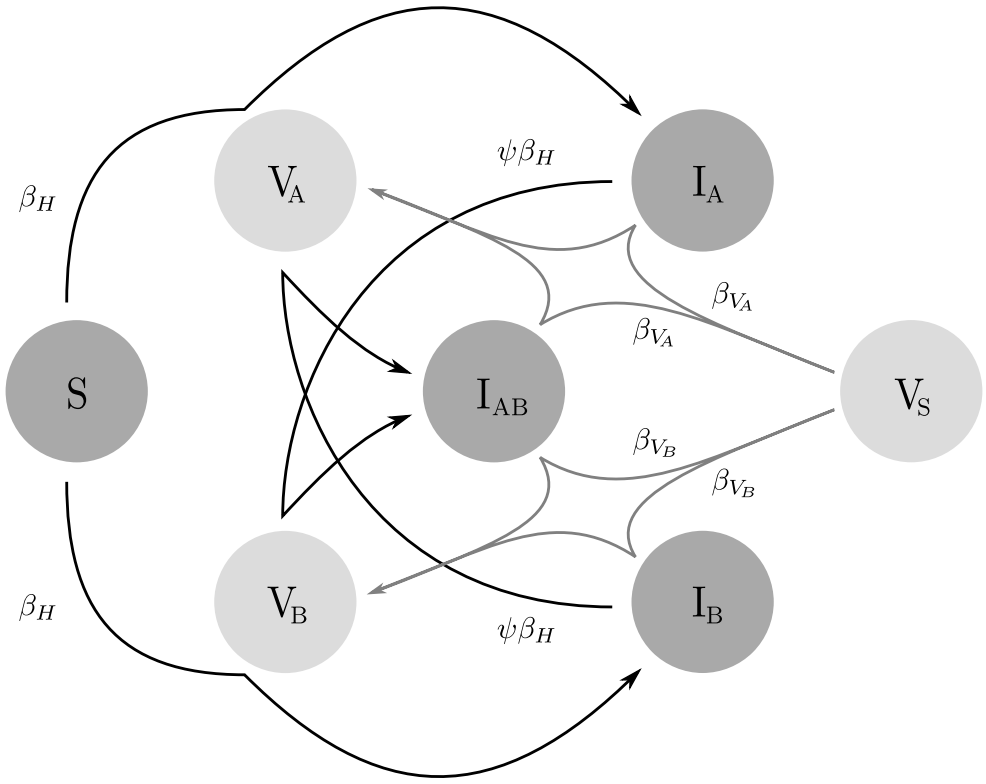

Figure 2: Diagram of the infective dynamics of the system (1) and (3). Dark arrows indicate new host infections; light arrows indicate new vector infections.

carrying capacity does not appear explicitly. Rather, the function (5) depends on the *population pressure* (θ), the population at which host births cease. This is defined in terms of the carrying capacity K as

$$\theta := \frac{b_0}{r_0} K, \tag{7}$$

so $K = \frac{r_0}{b_0}\theta < \theta$.

By using the function (6), we assume that infection does not alter the vector's reproductive ability, and so regardless of the presence of disease, the vector population grows exponentially with growth rate $r_V := b_V - \mu_V \geq 0$, so (4) becomes

$$\frac{dN_{V,i}}{dt} = r_V N_{V,i}, \ i = 1, 2, \ldots, n.$$

A summary of the model's variables and units is given in Table 1. The parameters, including those from the suggested growth functions (5) and (6), are listed in Table 2.

2.2 Proportions Model

For analysis, it is simpler to rewrite the system (1) and (3) in terms of the total host and vector populations and the infected proportions of each. Note that since

$$S(t) = N(t) - I_A(t) - I_B(t) - I_{AB}(t)$$

and

$$V_{i,S}(t) = N_{V,i}(t) - V_{i,A}(t) - V_{i,B}(t)$$

for all $t \geq 0$ and $i = 1, \ldots, n$, the susceptible populations are uniquely determined by the infected and total populations and need not be modeled explicitly. Thus, in our system, we omit the equations for susceptible hosts (1a) and vectors (3a) and substi-

Variable	Description
Host	
N	Total host population ($N := S + I_A + I_B + I_{AB}$)
S	Susceptible host population
I_A	Population of hosts infected by strain A
I_B	Population of hosts infected by strain B
I_{AB}	Population of hosts coinfected by strains A and B
Vector	
$N_{V,i}$	Total population of vector i ($N_{V,i} := V_{i,S} + V_{i,A} + V_{i,B}$)
$V_{i,S}$	Susceptible population of vector i
$V_{i,A}$	Population of vector i infected by strain A
$V_{i,B}$	Population of vector i infected by strain B

Table 1: List of variables and the populations they represent.

Parameter	Description	Range	Default
Host			
r_0	Growth rate of susceptible hosts ($r_0 := b_0 - \mu_0$)	$[0, \infty)$	44.87
b_0	Birth rate of susceptible hosts	$(0, \infty)$	45
b_1	Birth rate of singly-infected hosts	$[0, b_0]$	22.5
b_2	Birth rate of coinfected hosts	$[0, b_1]$	11.25
μ_0	Natural host mortality rate	$(0, b_0]$	0.13
μ_1	Additional mortality for singly-infected hosts	$[0, \mu_0]$	0.13
μ_2	(Max) Additional mortality for coinfected hosts	$[0, \mu_1]$	0.13
θ	Host population pressure	$(0, \infty)$	100
K	Host carrying capacity ($K := \frac{r_0}{b_0}\theta$)	$(0, \infty)$	99.71
Vector			
r_V	Vector growth rate ($r_V := b_V - \mu_V$)	$[0, \infty)$	0
b_V	Vector birth rate	$(0, \infty)$	12
μ_V	Vector mortality rate	$(0, b_V]$	12
Pathogen			
β_H	Vector-to-host transmission rate	$[0, 1]$	0.04
$\beta_{V_{i,A}}$	Host-to-vector-i transmission rate of strain A	$[0, 1]$	0.04
$\beta_{V_{i,B}}$	Host-to-vector-i transmission rate of strain B	$[0, 1]$	0.04
ψ	Pathogen taxa similarity	$[0, 1]$	0.5

Table 2: List of parameters, their ranges, and their default values, taken from Seabloom et al. [26].

tute those for total host (2) and vector populations (4) respectively.

Next, we rewrite the components of the system as proportions of the total populations by introducing new variables defined as

$$s := \frac{S}{N}, \qquad i_A := \frac{I_A}{N}, \qquad i_B := \frac{I_B}{N}, \quad i_{AB} := \frac{I_{AB}}{N},$$
$$v_{i,S} := \frac{V_{i,S}}{N_{V,i}}, \quad v_{i,A} := \frac{V_{i,A}}{N_{V,i}}, \quad v_{i,B} := \frac{V_{i,B}}{N_{V,i}}.$$

Thus, the proportions of susceptible hosts and vectors can be rewritten in terms of the infected proportions as

$$s = 1 - i_A - i_B - i_{AB} \quad \text{and} \quad v_{i,S} = 1 - v_{i,A} - v_{i,B}. \tag{8}$$

We also define the host's scaled maximum birth rate as

$$p := s + \frac{b_1}{b_0}(i_A + i_B) + \frac{b_2}{b_0}\, i_{AB}. \tag{9}$$

We note that $0 \leq p \leq 1$, and in the absence of disease, $p = 1$.

In order to rescale the model, we must rewrite the system in terms of these new variables and quantities. For $x = A, B, AB$ corresponding to the viral strain(s) infecting the host, the chain rule yields

$$\frac{di_x}{dt} = \frac{1}{N}\frac{dI_x}{dt} - \frac{1}{N}\frac{I_x}{N}\frac{dN}{dt} = \frac{1}{N}\frac{dI_x}{dt} - \frac{i_x}{N}\frac{dN}{dt}.$$

Similarly, for $y = A, B$ corresponding to the viral strain infecting the vector,

$$\frac{dv_{i,y}}{dt} = \frac{1}{N_{V,i}}\frac{dV_{i,y}}{dt} - \frac{1}{N_{V,i}}\frac{V_{i,y}}{N_{V,i}}\frac{dN_{V,i}}{dt} = \frac{1}{N_{V,i}}\frac{dV_{i,y}}{dt} - \frac{v_{i,y}}{N_{V,i}}\frac{dN_{V,i}}{dt}.$$

Using these formulas, the system in proportions form is as follows.

Proportions Model:

$$\frac{dN}{dt} = N\left[b_0\left(1-\frac{N}{\theta}\right)p - \mu_0 - \mu_1(1-s) - \psi\mu_2 i_{AB}\right], \tag{10a}$$

$$\frac{di_A}{dt} = \beta_H \sum_{i=1}^{n} N_{V,i}\left(s v_{i,A} - \psi i_A v_{i,B}\right) - i_A\left[b_0\left(1-\frac{N}{\theta}\right)p + \mu_1 s - \psi\mu_2 i_{AB}\right], \tag{10b}$$

$$\frac{di_B}{dt} = \beta_H \sum_{i=1}^{n} N_{V,i}\left(s v_{i,B} - \psi i_B v_{i,A}\right) - i_B\left[b_0\left(1-\frac{N}{\theta}\right)p + \mu_1 s - \psi\mu_2 i_{AB}\right], \tag{10c}$$

$$\frac{di_{AB}}{dt} = \beta_H\psi \sum_{i=1}^{n} N_{V,i}(i_A v_{i,B} + i_B v_{i,A}) - i_{AB}\left[b_0\left(1-\frac{N}{\theta}\right)p + \mu_1 s + \psi\mu_2(1-i_{AB})\right], \tag{10d}$$

$$\frac{dN_{V,i}}{dt} = r_V N_{V,i}, \tag{10e}$$

$$\frac{dv_{i,A}}{dt} = \beta_{V_{i,A}} N v_{i,S}(i_A + i_{AB}) - b_V v_{i,A}, \tag{10f}$$

$$\frac{dv_{i,B}}{dt} = \beta_{V_{i,B}} N v_{i,S}(i_B + i_{AB}) - b_V v_{i,B}. \tag{10g}$$

2.3 Host Population Bounds

We demonstrate that the total host population N is asymptotically bounded by using the comparison argument presented in Feng and Hinson [10]. Consider two initial value problems

$$u_i' = f_i(t, u_i), \text{ in } (0,T],\ u_i(0) = u_{i,0},\ i = 1,2. \tag{11}$$

where the functions f_i, $i = 1,2$ are continuous in $[0,T]\times\mathbb{R}$. Then we have the comparison result below.

Lemma 1 (Comparison Argument)**.** *Let* $\frac{\partial f_i}{\partial u}$, $i = 1,2$ *be continuous in* $[0,T]\times\mathbb{R}$. *If* $f_1(t,u) \le f_2(t,u)$ *in* $(0,T]\times\mathbb{R}$ *and* $u_{1,0} \le u_{2,0}$, *then the corresponding solutions* u_1 *and* u_2 *of* (11) *satisfy* $u_1(t) \le u_2(t)$ *on* $[0,T]$.

Based on Lemma 1, we have the following bounds on the host population.

Theorem 1 (Host Population Bounds)**.** *The host population for the proportions model* (10) *satisfies the bounds*

$$\left[\left(\frac{1}{N(0)}+\frac{b_0}{\mu\theta}\right)e^{\mu t}-\frac{b_0}{\mu}\right]^{-1}\leq N(t)\leq\left[\left(\frac{1}{N(0)}-\frac{1}{K}\right)e^{-r_0 t}+\frac{1}{K}\right]^{-1}, \tag{12}$$

where $\mu=\mu_0+\mu_1+\mu_2$.

Proof. Since the quantities $p,\ s,\ \psi$ and i_{AB} are all positive and bounded above by 1,

$$-b_0\frac{N^2}{\theta}-\mu N\leq\frac{dN}{dt}\leq r_0 N\left(1-\frac{N}{K}\right).$$

The comparison argument Lemma 1 gives us the upper and lower bounds on $N(t)$. □

By Theorem 1, the host population satisfies the bounds

$$0\leq\liminf_{t\to\infty}N(t)\leq\limsup_{t\to\infty}N(t)\leq K.$$

This proves the boundedness of the total population.

3 Analysis of the Single Vector Model with Constant Vector Growth

We begin our analysis by examining the most basic case of a single vector species. To simplify notation, we omit the species subscripts i from the vector variables and parameters in (10), giving the single–host, single–vector, two–pathogen strain model

$$\frac{dN}{dt} = N\left[b_0\left(1-\frac{N}{\theta}\right)p - \mu_0 - \mu_1(1-s) - \psi\mu_2 i_{AB}\right], \tag{13a}$$

$$\frac{di_A}{dt} = \beta_H N_V\left(sv_A - \psi i_A v_B\right) - i_A\left[b_0\left(1-\frac{N}{\theta}\right)p + \mu_1 s - \psi\mu_2 i_{AB}\right], \tag{13b}$$

$$\frac{di_B}{dt} = \beta_H N_V\left(sv_B - \psi i_B v_A\right) - i_B\left[b_0\left(1-\frac{N}{\theta}\right)p + \mu_1 s - \psi\mu_2 i_{AB}\right], \tag{13c}$$

$$\frac{di_{AB}}{dt} = \beta_H \psi N_V(i_A v_B + i_B v_A) - i_{AB}\left[b_0\left(1-\frac{N}{\theta}\right)p + \mu_1 s + \psi\mu_2(1-i_{AB})\right], \tag{13d}$$

$$\frac{dN_V}{dt} = r_V N_V, \tag{13e}$$

$$\frac{dv_A}{dt} = \beta_{V_A} N v_S(i_A + i_{AB}) - b_V v_A, \tag{13f}$$

$$\frac{dv_B}{dt} = \beta_{V_B} N v_S(i_B + i_{AB}) - b_V v_B. \tag{13g}$$

Let $\mathbf{x} = (N, i_A, i_B, i_{AB}, N_V, v_A, v_B)^T$ be the vector of host and vector populations and population proportions and define $f(\mathbf{x}) = \frac{d\mathbf{x}}{dt}$ by the system (13). Define the region

$$D := \left\{ \begin{pmatrix} N \\ i_A \\ i_B \\ i_{AB} \\ N_V \\ v_A \\ v_B \end{pmatrix} \in \mathbb{R}^7_+ \;\middle|\; \begin{array}{c} 0 \le N \le K \\ i_A \ge 0 \\ i_B \ge 0 \\ i_{AB} \ge 0 \\ i_A + i_B + i_{AB} \le 1 \\ N_V \ge 0 \\ v_A \ge 0 \\ v_B \ge 0 \\ v_A + v_B \le 1 \end{array} \right\}.$$

Choosing an initial value $\mathbf{x}_0 \in D$ gives the initial value problem (IVP)

$$\frac{d\mathbf{x}}{dt} = f(\mathbf{x}), \quad \mathbf{x}(0) = \mathbf{x}_0. \tag{14}$$

We begin by proving existence and uniqueness of solutions of (14).

Theorem 2 (Uniqueness of Solutions)**.** *The IVP* (14) *has a unique solution* $\mathbf{x}(t)$ *on the interval* $[-a, a]$ *for some* $a > 0$.

Proof. Observe that $f(\mathbf{x}) \in C'(D)$. Then by the Fundamental Existence-Uniqueness Theorem [22], a unique solution $\mathbf{x}(t)$ exists on $[-a, a]$, for some $a > 0$. □

Next, we prove existence of the solutions of (14) for all $t \geq 0$ and positive invariance in the region D.

Theorem 3 (Existence and Positive Invariance)**.** *Let* $\mathbf{x}_0 \in D$. *Then any solution* $\mathbf{x}(t)$ *of* (14) *through* $\mathbf{x}_0$ *is defined for all* $t \geq 0$. *Furthermore, the region* D *is positively invariant.*

Proof. The existence of solutions on $[0, \infty)$ follows from Section 2.4, Theorem 3, Corollary 1 in [22]. To prove positive invariance, suppose that $\mathbf{x}_0$ lies on the boundary of D. We show that on every such boundary, the derivatives given in (13) prevent the solution $\mathbf{x}(t)$ from leaving the region.

If $N = 0$, then $\frac{dN}{dt} = 0$. Since the quantities p, s, ψ and i_{AB} are all positive and bounded above by 1, $\frac{dN}{dt} \leq r_0 N \left(1 - \frac{N}{K}\right)$. Thus, when $N = K$, $\frac{dN}{dt} \leq 0$.

If $i_A = 0$, then $\frac{di_A}{dt} = \beta_H N_V s v_A > 0$. If $i_B = 0$, then $\frac{di_B}{dt} = \beta_H N_V s v_B > 0$. If $i_{AB} = 0$, then $\frac{di_{AB}}{dt} = \beta_H \psi N_V (i_A v_B + i_B v_A) > 0$.

Similarly, if $N_V = 0$, then $\frac{dN_V}{dt} = 0$. If $v_A = 0$, then $\frac{dv_A}{dt} = \beta_{v_A} N v_S (i_A + i_{AB}) > 0$. If $v_B = 0$, then $\frac{dv_B}{dt} = \beta_{v_B} N v_S (i_B + i_{AB}) > 0$.

Suppose $i_A + i_B + i_{AB} = 1$. Then by (8), $s = 0$, and by (9), $p > 0$, so

$$\frac{d(i_A + i_B + i_{AB})}{dt} = \beta_H N_V s(v_A + v_B) - b_0 \left(1 - \frac{N}{\theta}\right) p - \mu_1 s = -b_0 \left(1 - \frac{N}{\theta}\right) p < 0,$$

since $N \leq K < \theta$.

Suppose $v_A + v_B = 1$. Then by (8), $v_S = 0$, so

$$\frac{d(v_A + V_B)}{dt} = N v_S \left(\beta_{V_A}(i_A + i_{AB}) + \beta_{V_B}(i_B + i_{AB})\right) - b_V = -b_V < 0.$$

This proves positive invariance. □

3.1 Linear Stability Analysis of the Disease-Free Equilibrium

The disease-free equilibrium (DFE) of the system (13) is found by assuming no infected populations, i.e. $i_A = i_B = i_{AB} = v_A = v_B = 0$. These populations are then fixed at zero for all time. In addition, the total populations N and N_V must also be constant. By (8) and (9), $s = v_S = p = 1$. Using (7) to rewrite θ in terms of K,

$$\begin{aligned}
\frac{dN}{dt} &= N\left[b_0\left(1 - \frac{N}{\theta}\right) - \mu_0\right] = r_0 N\left(1 - \frac{N}{K}\right), \\
\frac{dN_V}{dt} &= r_V N_V.
\end{aligned}$$

Thus, the total host population is constant when either $N = 0$ (at the trivial equilibrium) or $N = K$ (at the DFE). Due to the assumption of exponential growth in the vector, $\frac{dN_V}{dt} = 0$ is only possible under the assumption

(A1) The vector birth and death rates are equal, i.e. $b_V = \mu_V \Leftrightarrow r_V = 0$.

In this case, the vector population remains constant and fixed to its initial value. We denote this equilibrium population K_V. The DFE is thus

$$(N, i_A, i_B, i_{AB}, N_V, v_A, v_B)^T = (K, 0, 0, 0, K_V, 0, 0)^T.$$

We next use this equilibrium to determine the basic reproduction number $\mathcal{R}_0$ of the disease. This quantity is related to the average number of new infections resulting

from the introduction of a single infectious individual into an entirely susceptible population. If this value is above the threshold 1, the disease will persist and solutions tend towards the endemic equilibrium; if below, it will die out and solutions tend towards the DFE.

Theorem 4 (Basic Reproduction Number)**.** *The basic reproduction number of the disease is*

$$\mathcal{R}_0 = \begin{cases} \sqrt{\dfrac{\beta_{V_A}\beta_H K K_V}{\mu_V(\mu_0+\mu_1)}} \text{ if } \beta_{V_A} \geq \beta_{V_B}, \text{ and} \\ \sqrt{\dfrac{\beta_{V_B}\beta_H K K_V}{\mu_V(\mu_0+\mu_1)}} \text{ if } \beta_{V_A} < \beta_{V_B}. \end{cases}$$

Thus, $\mathcal{R}_0$ is a function of the pathogen strain with the larger host-to-vector transmission rate. To prove this, we use the next generation approach [29].

Proof. Let $X = \begin{bmatrix} i_A & i_B & i_{AB} & v_A & v_B \end{bmatrix}^T$. Then the system (13) can be written as $\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X)$ where

$$\mathcal{F}(X) = \begin{bmatrix} \beta_H N_V s v_A \\ \beta_H N_V s v_B \\ \psi\beta_H N_V (v_B i_A + v_A i_B) \\ \beta_{V_A} N v_S (i_A + i_{AB}) \\ \beta_{V_B} N v_S (i_B + i_{AB}) \end{bmatrix}$$

represents new infections and

$$\mathcal{V}(X) = \begin{bmatrix} \psi\beta_H N_V i_A v_B + i_A \left[b_0 \left(1 - \frac{N}{\theta}\right) p + \mu_1 s - \psi\mu_2 i_{AB} \right] \\ \psi\beta_H N_V i_B v_A + i_B \left[b_0 \left(1 - \frac{N}{\theta}\right) p + \mu_1 s - \psi\mu_2 i_{AB} \right] \\ i_{AB} \left[b_0 \left(1 - \frac{N}{\theta}\right) p + \mu_1 s - \psi\mu_2 (1 - i_{AB}) \right] \\ \mu_V v_A \\ \mu_V v_B \end{bmatrix}$$

represents all other dynamics. Let F and V be the Jacobians of $\mathcal{F}$ and $\mathcal{V}$ respectively, evaluated at the DFE. Then using the definition of θ from (7),

$$F = \begin{bmatrix} 0 & 0 & 0 & \beta_H K_V & 0 \\ 0 & 0 & 0 & 0 & \beta_H K_V \\ 0 & 0 & 0 & 0 & 0 \\ \beta_{V_A} K & 0 & \beta_{V_A} K & 0 & 0 \\ 0 & \beta_{V_B} K & \beta_{V_B} K & 0 & 0 \end{bmatrix},$$

and

$$V = \operatorname{diag}(\mu_0 + \mu_1, \mu_0 + \mu_1, \mu_0 + \mu_1 - \psi\mu_2, \mu_V, \mu_V).$$

From [29], $\mathcal{R}_0$ is the spectral radius of the next-generation matrix M given by

$$M = FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & \dfrac{\beta_H K_V}{\mu_V} & 0 \\ 0 & 0 & 0 & 0 & \dfrac{\beta_H K_V}{\mu_V} \\ 0 & 0 & 0 & 0 & 0 \\ \dfrac{\beta_{V_A} K}{\mu_0 + \mu_1} & 0 & \dfrac{\beta_{V_A} K}{\mu_0 + \mu_1 - \psi\mu_2} & 0 & 0 \\ 0 & \dfrac{\beta_{V_B} K}{\mu_0 + \mu_1} & \dfrac{\beta_{V_B} K}{\mu_0 + \mu_1 - \psi\mu_2} & 0 & 0 \end{bmatrix}. \tag{15}$$

A routine computation shows the eigenvalues of M are

$$0, \quad \pm\sqrt{\frac{\beta_{V_A}\beta_H K K_V}{\mu_V(\mu_0 + \mu_1)}}, \quad \text{and} \quad \pm\sqrt{\frac{\beta_{V_B}\beta_H K K_V}{\mu_V(\mu_0 + \mu_1)}},$$

so the spectral radius depends on $\max\{\beta_{V_A}, \beta_{V_B}\}$. □

We now show that stability of the DFE is implied by $\mathcal{R}_0 < 1$ while $\mathcal{R}_0 > 1$ implies its instability.

Theorem 5 (Stability of the DFE)**.** *If $\mathcal{R}_0 < 1$ then the DFE has strictly negative*

eigenvalues and is locally asymptotically stable. If $\mathcal{R}_0 > 1$ then the DFE has at least one positive eigenvalue and is unstable.

Proof. Due to the assumption of a constant total vector population, we omit the equation (13e) from the system and consider only the remaining six equations. The Jacobian of this reduced system, evaluated at the DFE, is

$$\begin{bmatrix} -r_0 & -K\left(\mu_0+\mu_1-\frac{\mu_0 b_1}{b_0}\right) & -K\left(\mu_0+\mu_1-\frac{\mu_0 b_1}{b_0}\right) & -K\left(\mu_0+\mu_1+\psi\mu_2-\frac{\mu_0 b_2}{b_0}\right) & 0 & 0 \\ 0 & -\mu_0-\mu_1 & 0 & 0 & \beta_H K_V & 0 \\ 0 & 0 & -\mu_0-\mu_1 & 0 & 0 & \beta_H K_V \\ 0 & 0 & 0 & -\mu_0-\mu_1+\psi\mu_2 & 0 & 0 \\ 0 & \beta_{V_A} K & 0 & \beta_{V_A} K & -\mu_V & 0 \\ 0 & 0 & \beta_{V_B} K & \beta_{V_B} K & 0 & -\mu_V \end{bmatrix}.$$

Clearly, this matrix has two eigenvalues $-r_0$ and $-\mu_0-\mu_1+\psi\mu_2$, since they are diagonal elements and are the only nonzero elements in their column or row respectively. We have already assumed that the host intrinsic growth rate $r_0 > 0$; This guarantees that the eigenvalue $-r_0$ is negative. This corresponds to the host population growing instead of remaining constant in the absence of disease.

Since $0 \leq \psi \leq 1$ and $\mu_0 \geq \mu_1 \geq \mu_2 \geq 0$ with $\mu_0 > 0$,

$$-\mu_0 - \mu_1 + \psi\mu_2 \leq -\mu_0 - \mu_1 + \mu_2 \leq -\mu_0 < 0,$$

so this eigenvalue is strictly negative.

To find the remaining four eigenvalues, we examine the matrix formed by omitting the first and fourth rows and columns from the Jacobian, as these contained the first two eigenvalues. Factoring the characteristic polynomial of this matrix gives that the eigenvalues are the roots of

$$x^2 + (\mu_0 + \mu_1 + \mu_V)x - \beta_{V_A}\beta_H K K_V + \mu_V(\mu_0 + \mu_1)$$

and

$$x^2 + (\mu_0 + \mu_1 + \mu_V)x - \beta_{V_B}\beta_H K K_V + \mu_V(\mu_0 + \mu_1).$$

Both polynomials are similar, with β_{V_A} replaced by β_{V_B} in the second. Letting $y = A$ or B, then the four eigenvalues are

$$\lambda_1^y, \lambda_2^y = \frac{1}{2}\left[-\mu_0 - \mu_1 - \mu_V \pm \sqrt{(\mu_0+\mu_1+\mu_V)^2 + 4\left[\beta_{V_y}\beta_H K K_V - \mu_V(\mu_0+\mu_1)\right]}\right],$$

where $\lambda_1^y \geq \lambda_2^y$ and $\lambda_2^y < 0$. Thus

$$\max\{\lambda_1^A, \lambda_1^B\} \geq \min\{\lambda_1^A, \lambda_1^B\} \geq \max\{\lambda_2^A, \lambda_2^B\} \geq \min\{\lambda_2^A, \lambda_2^B\}.$$

We note that the discriminant is

$$(\mu_0 + \mu_1 - \mu_V)^2 + 4\beta_{V_y}\beta_H K K_V \geq 0.$$

Since all parameters are positive, all four eigenvalues have zero complex part and the DFE is a nodal source or sink, not a spiral.

Since we know $\lambda_2^A < 0$ and $\lambda_2^B < 0$, we are interested in determining the signs of λ_1^A and λ_1^B. It suffices to show that $\mathcal{R}_0 > 1$ implies the largest of the two is positive and that $\mathcal{R}_0 < 1$ implies the largest is negative.

By Theorem 4,

$$\mathcal{R}_0 = \max\left\{\sqrt{\frac{\beta_{V_A}\beta_H K K_V}{\mu_V(\mu_0+\mu_1)}},\ \sqrt{\frac{\beta_{V_B}\beta_H K K_V}{\mu_V(\mu_0+\mu_1)}}\right\}.$$

Suppose $\mathcal{R}_0 < 1$. This implies $\beta_{V_y}\beta_H K K_V - \mu_V(\mu_0+\mu_1) < 0$, so

$$\max\{\lambda_1^A, \lambda_1^B\} < \frac{1}{2}\left(-\mu_0 - \mu_1 - \mu_V + \sqrt{(\mu_0+\mu_1+\mu_V)^2}\right) = 0.$$

Thus, all the eigenvalues are bounded below zero, so the DFE is locally asymptotically stable.

Suppose $\mathcal{R}_0 > 1$. Then

$$\max\{\lambda_1^A, \lambda_1^B\} > \frac{1}{2}\left(-\mu_0 - \mu_1 - \mu_V + \sqrt{(\mu_0 + \mu_1 + \mu_V)^2}\right) = 0.$$

Since at least one eigenvalue is positive, the DFE is unstable. This completes the proof. □

3.2 Biological Interpretation of R_0

Although $\mathcal{R}_0$ is a useful quantity for determining the stability of the disease-free equilibrium (as proven in Theorem 5), it is difficult to interpret it biologically for models involving heterogeneous populations. When there are multiple pathogen strains and multiple types of infection, the concept of a single infectious individual is not well defined. We follow the computation in [6] to interpret $\mathcal{R}_0$ computed in Theorem 4.

We define for $x = A, B$, the two quantities

- N_{hv}^x: number of secondary infections generated by a single infected (by pathogen strain x) vector in a completely susceptible host population.
- N_{vh}^x: number of secondary infections generated by a single infected (by pathogen strain x) host in a completely susceptible vector population.

We can compute the numbers N_{hv}^x and N_{vh}^x as the product of the number of the relevant adequate (for disease to be transmitted) contacts per unit time (of a vector with susceptible hosts, or of a host with susceptible vectors, respectively) with the mean waiting time (of vector, or host, respectively) spent in the infectious compartment. Thus, we have for $x = A, B$

$$N_{hv}^x = \frac{\beta_H K}{\mu_V}, \tag{16}$$

$$N_{vh}^x = \frac{\beta_{V_x} K_V}{\mu_0 + \mu_1}. \tag{17}$$

We next introduce the single strain reproduction numbers $\mathcal{R}_A$ and $\mathcal{R}_B$.

Definition 1 (Single Strain Basic Reproduction Numbers)**.** The single strain (A or B) basic reproduction numbers are defined as

$$\mathcal{R}_A = \sqrt{N_{hv}^A N_{vh}^A},$$
$$\mathcal{R}_B = \sqrt{N_{hv}^B N_{vh}^B}.$$

We note that the interpretation of $\mathcal{R}_x^2$ for $x = A, B$ is the number of hosts that a single host infected with strain x will infect through a generation of infections in vectors, assuming all other vectors and hosts are susceptible. Theorem 4 computes the basic reproduction of model (13) as

$$\mathcal{R}_0 = \max\{\mathcal{R}_A, \mathcal{R}_B\}.$$

Thus we can interpret (or define) $\mathcal{R}_0^2$ as the maximum of the number of secondary infections of type A or B that are generated by a single host infected with strain A or B.

3.3 Type Reproduction Numbers

The *type reproduction number*, introduced by Roberts and Heesterbeek [23], isolates infections generated by a single type of individual in a heterogeneous population. For n epidemiologically distinct types of hosts, the type reproduction number of type i is defined as

Definition 2 (Type Reproduction Number)**.** The type reproduction number T_i is the expected number of cases in individuals of type i caused by one infected individual of type i in a completely susceptible population, either directly or through chains of infection passing through any sequence of the other types.

This number can be derived from the next generation matrix M (15) and is given by the formula

$$T_i = e_i' M(I - (I - P)M)^{-1} e_i, \tag{18}$$

where e_i is the unit vector with ith component equal to 1 and all other components equal to zero, I is the identity matrix, and P is the projection matrix on type i, where $P_{ii} = 1$ and all other entries are zero [23].

In our case, we are interested in the individual dynamics of each strain of the virus. Since the virus is found in both hosts and vectors, this gives four epidemiologically distinct host types and thus four type reproduction numbers.

The following theorem shows that these type reproduction numbers are the same for hosts and vectors but different for the two strains. For simplicity, we thus index the numbers by the corresponding strains rather than the host or vector types.

Theorem 6 (Type-Reproduction Numbers)**.** *The type-reproduction number of the disease is*

$$\mathcal{T}_A = \frac{\beta_{V_A}\beta_H K K_V}{\mu_V(\mu_0 + \mu_1)} \text{ for strain } A, \text{ and}$$
$$\mathcal{T}_B = \frac{\beta_{V_B}\beta_H K K_V}{\mu_V(\mu_0 + \mu_1)} \text{ for strain } B.$$

Proof. The type-reproduction numbers can be computed easily using the formula given by Roberts and Heesterbeek [23]. For $i = 1$ and $i = 4$ corresponding to hosts and vectors infected with strain A respectively, we obtain

$$T_1 = T_4 = \frac{\beta_{V_A}\beta_H K K_V}{\mu_V(\mu_0 + \mu_1)} = \mathcal{T}_A = \mathcal{R}_A^2,$$

and for $i = 2$ and $i = 5$ corresponding to hosts and vectors infected with strain B

respectively,

$$T_2 = T_5 = \frac{\beta_{V_B}\beta_H K K_V}{\mu_V(\mu_0 + \mu_1)} = \mathcal{T}_B = \mathcal{R}_B^2.$$

□

The type reproduction numbers corresponding to strains A and B are the corresponding single strain reproduction numbers squared. We also note that from formula (18) we can compute $T_3 = 0$. This type-reproduction number corresponds to coinfected host individuals I_{AB}.

An interesting consequence of Theorem 6 is that

$$\mathcal{R}_0^2 = \max\{\mathcal{T}_A, \mathcal{T}_B\}. \tag{19}$$

Using this result, it follows from Theorem 5 that the DFE is stable if and only if both $\mathcal{T}_A < 1$ and $\mathcal{T}_B < 1$. This effect is illustrated in Figure 3. Even though $R_0 > 1$ in both the second and third plots, coinfection is only possible when both type reproduction numbers are greater than one, as in the third plot. In this case, both strains remain endemic and coinfection can occur if the pathogen taxa are distinct and cross-protection is incomplete ($\psi \neq 0$).

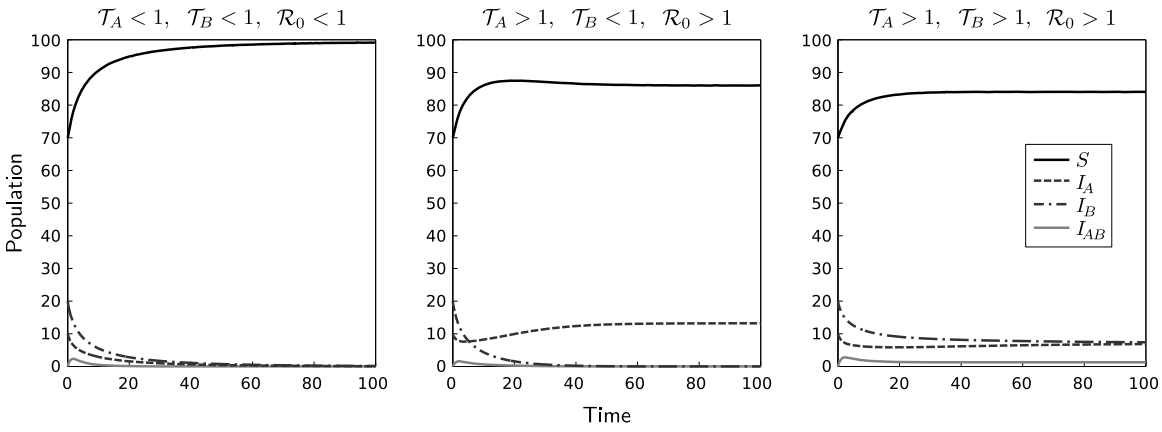

Figure 3: Plots illustrating the threshold effect of the type reproduction numbers. At left, both reproduction numbers are less than one and all three infected classes tend towards extinction. At center, strain A persists, but strain B does not, so coinfection is not possible in the long term. At right, both strains persist and coinfection is possible.

3.4 Numerical Simulations

We run simulations using the base parameter values given in Table 2. By varying specific parameters, particularly transmission rates (β_H, β_{V_A}, β_{V_B}), the cross-protection factor (ψ), and initial conditions, we can explore the resulting changes in system dynamics.

Appendix B describes how the model examined by Seabloom et al. [26] is a special case of the system (1) and (3). To illustrate this, we reproduce Figure 4 from Seabloom et al. [26], as shown in our Figure 4. This figure shows the prevalences of infection and coinfection under varying values of ψ and $\beta := \beta_H = \beta_{V_A} = \beta_{V_B}$. In order to reproduce these results, we simulate the system (13) until it reaches equilibrium and plot the resulting prevalences.

As discussed in [26], prevalences are higher for a single generalist vector species that can transmit both pathogen strains than for two specialist vector species that can only transmit a single strain. Although the prevalence of coinfection varies strongly with respect to the taxa similarity/cross-protection factor ψ, total prevalence (including both singly infected and coinfected individuals) varies only slightly. Thus, as the taxa similarity factor ψ increases (and cross-protection decreases), singly infected individuals move into the coinfected compartment, but few susceptible individuals leave their compartment or are added. This is reasonable since the parameter ψ only governs the rate that individuals acquire secondary infections and the additional mortality of coinfected individuals.

However, the pathogen transmission parameters (β_H, β_{V_A}, and β_{V_B}) are highly influential on long-term pathogen prevalences, both of coinfection and total infection prevalence. Biologically, this makes sense because these parameters control infections of all compartments, including both the rate of movement from susceptible to singly infected and from singly infected to coinfected.

Figure 5 illustrates convergence of the system with two sets of initial conditions

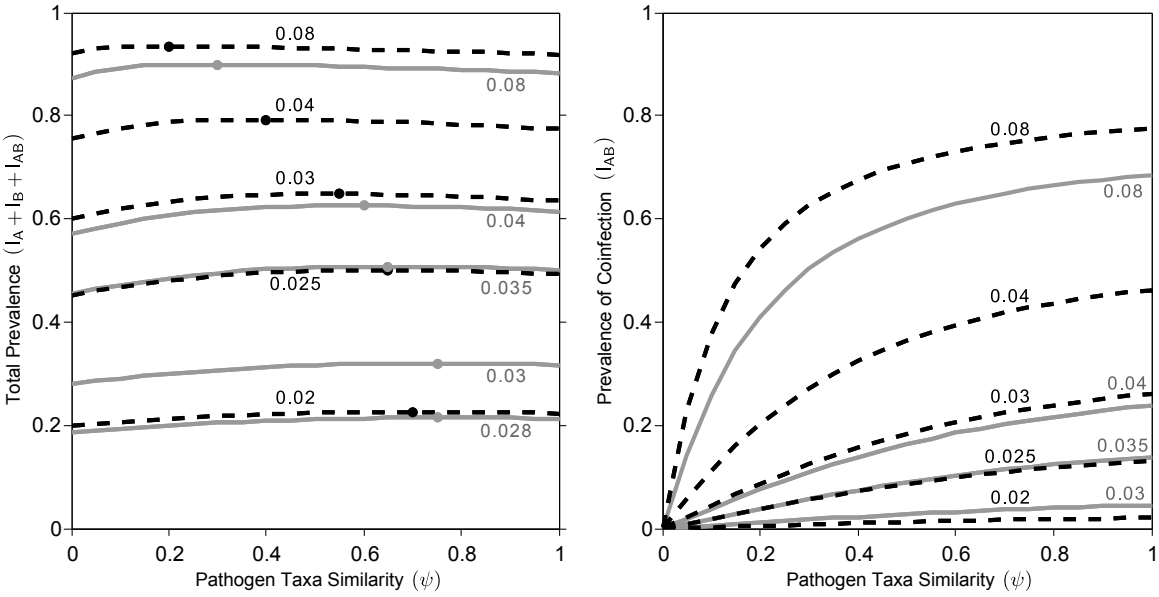

Figure 4: Prevalence of infection and coinfection against ψ. Labels indicate varying values of β (reproduced from Seabloom et al. [26]). Dashed black lines represent a single generalist vector species; solid gray lines represent two specialist vector species.

but the same parameter values. We set $\beta_{V_A} = \beta_{V_B} = 0.04$ so these strains are not distinguished. On the top in Figure 5, a susceptible population of vectors encounters an infected host community with no coinfection. The vectors rapidly acquire the infection and spread it within the hosts, and the prevalence of coinfection rises and stabilizes. On the bottom in Figure 5, the host community is entirely susceptible, and a population of mixed susceptible/infected vectors is introduced. In this case, hosts rapidly acquire one or two infections. The prevalence of coinfection lags behind that of each isolated strain until $t = 5$, at which point coinfections surpass infections in prevalence. However, the total prevalence of isolated infections, including both strains, remains higher.

3.5 Sensitivity Analysis of the Coinfected Equilibrium

We continue to make the assumption (A1) in Section 3.1 of a fixed vector population. Although numerical exploration shows this to be unnecessary for existence of the coinfected/endemic equilibrium, it maintains consistency with our previous analysis.

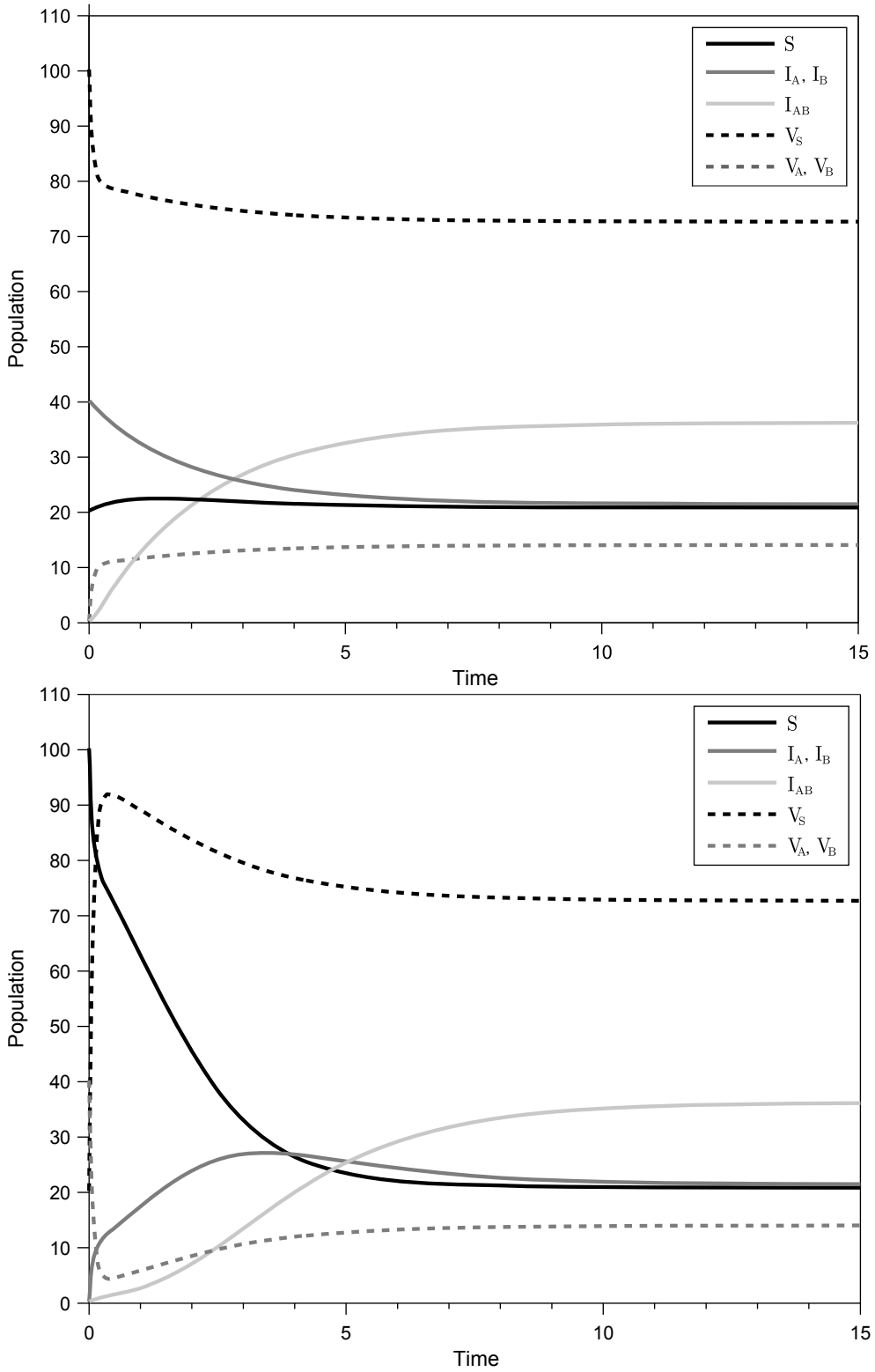

Figure 5: Simulations using the parameters in Table 2. Pathogen strains have equal transmission rates and graphs differ only by initial conditions. Top: all 100 vectors begin susceptible; 40 hosts have each strain and the remaining 20 are susceptible. Bottom: hosts begin with 100 susceptibles; 40 vectors have each strain and the remaining 20 are susceptible.

Although we do not have an analytical formula for the components of the endemic equilibrium, a sensitivity analysis can be conducted using methodology similar to that of Chitnis et al. [7], which we describe below.

Sensitivity analysis is a useful tool to measure the strength of influence of a model's parameters on its dynamics. This indicates which parameters are most influential and thus which are of greatest concern for measurement error [5]. For our models, we are interested in determining the factors that most affect the prevalences of infection and coinfection at the disease's endemic equilibrium [26]. We first define sensitivity indices that give us comparative measures of the effect of each parameter of the model on each component of the infected coexistence or endemic equilibrium.

Definition 3. The *normalized sensitivity index* [7] or *elasticity* [5] of a quantity x with respect to a parameter p represents the percentage increase in x resulting from a one percent increase in the parameter p. It is defined as

$$\mathcal{I}(x;p) := \frac{p}{x}\frac{\partial x}{\partial p}.$$

In this definition, the quantity $\frac{\partial x}{\partial p}$ represents the additive change in x resulting from a change in p. The normalizing factor $\frac{p}{x}$ is used to convert these indices into unitless measures of multiplicative change that can be easily compared.

The specific quantities of interest are the components of the endemic equilibrium of system (13). Since the constant vector population N_V is independent of the parameters, we omit it from the sensitivity analysis, leaving six remaining equilibrium components. For simplicity, we denote these values x_i for $i = 1, \ldots, 6$ and define

$$X := \begin{bmatrix} x_1 & \cdots & x_6 \end{bmatrix}^T = \begin{bmatrix} N & i_A & i_B & i_{AB} & v_A & v_B \end{bmatrix}^T.$$

The right-hand sides of the ODEs in the system (13), again excluding that for N_V, involve a total of twelve parameters. All other parameters (e.g. θ) can be derived

from these independent base parameters, which we denote as p_j for $j = 1, \ldots, 12$. The vector of parameters is

$$P := \begin{bmatrix} p_1 & \cdots & p_{12} \end{bmatrix}^T = \begin{bmatrix} b_0 & b_1 & b_2 & \mu_0 & \mu_1 & \mu_2 & K & \mu_V & \beta_H & \beta_{V_A} & \beta_{V_B} & \psi \end{bmatrix}^T.$$

The right hand sides of the ODEs in the system (13) are denoted as $g_k(X; P)$ for $k = 1, \ldots, 6$ and we set

$$G := \begin{bmatrix} g_1(X;P) & \cdots & g_6(X;P) \end{bmatrix}^T.$$

In order to compute the sensitivity indices of the components x_i of the endemic equilibrium, it is necessary to calculate $\frac{\partial x_i}{\partial p_j}$. At the endemic equilibrium, $g_k(X; P) = 0$, and differentiating each of these six functions with respect to each of the twelve parameters p_j gives 72 equations of the form

$$\frac{dg_k}{dp_j} = \sum_{i=1}^{6} \frac{\partial g_k}{\partial x_i}\frac{\partial x_i}{\partial p_j} + \sum_{\ell=1}^{12} \frac{\partial g_k}{\partial p_\ell}\frac{\partial p_\ell}{\partial p_j} = 0,$$

for $1 \leq k \leq 6$ and $1 \leq j \leq 12$. Since the base parameters are independent of one another, $\frac{\partial p_\ell}{\partial p_j}$ is 0 for $\ell \neq j$ and is 1 otherwise. This allows us to rewrite the above system of equations as

$$\sum_{i=1}^{6} \frac{\partial g_k}{\partial x_i}\frac{\partial x_i}{\partial p_j} = -\frac{\partial g_k}{\partial p_j},$$

for $1 \leq k \leq 6$ and $1 \leq j \leq 12$. For fixed j, we can combine the six equations corresponding to each variable x_i to form twelve linear systems, one for each j, $1 \leq j \leq 12$, in the form

$$Az^{(j)} = b^{(j)},$$

where $A = \frac{\partial G}{\partial X}$ is the Jacobian of G with respect to X evaluated at the infected coexistence equilibrium, $b^{(j)}$ is the vector $-\frac{\partial G}{\partial p_j}$ again evaluated at the infected equilibrium,

and $z^{(j)}$ is the desired vector of unknowns $\frac{\partial X}{\partial p_j}$.

Once these linear systems have been solved for the indices $\frac{\partial x_i}{\partial p_j}$, the normalized sensitivity indices $\mathcal{I}(x;p)$ are then obtained by scaling the sensitivities $\frac{\partial x_i}{\partial p_j}$ by the factor $\frac{p_j}{x_i}$, at the baseline parameter values given in Table 2, and at the component values of the endemic equilibrium.

We conduct sensitivity analyses for two cases of baseline parameter values, differing by the host-to-vector transmission rates β_{V_A} and β_{V_B}. In both cases, we assume the fixed vector population to be $N_V = 100$.

Case 1 ($\beta_{V_A} = \beta_{V_B}$) The first case uses the baseline parameter values given in Table 2, with resulting sensitivities given in Table 3. With these parameters, the endemic equilibrium is approximately

$$X = \begin{bmatrix} 98.77 & 0.1902 & 0.1902 & 0.4416 & 0.1466 & 0.1466 \end{bmatrix}^T. \tag{20}$$

In the model, the only parameters that can distinguish between i_A and i_B and between v_A and v_B are β_{V_A} and β_{V_B}. However, for this case, the baseline transmission rates are equal, so the two strains share similar dynamics. Coinfection is common in the population and only about 17.8% of the population is uninfected.

Case 2 ($\beta_{V_A} < \beta_{V_B}$) In the second case, the strains are distinguished by strain A having a much lower transmission rate ($\beta_{V_A} = 0.02$) than strain B ($\beta_{V_B} = 0.08$). All other parameters use the baseline parameter values given in Table 2. The corresponding sensitivity indices are tabulated in Table 4. In this case, the endemic equilibrium becomes approximately

$$X = \begin{bmatrix} 98.98 & 0.0079 & 0.7289 & 0.1151 & 0.0129 & 0.3531 \end{bmatrix}^T. \tag{21}$$

	N	i_A	i_B	i_{AB}	v_A	v_B
b_0	0.0018	−0.0005	−0.0005	0.0009	0.0016	0.0016
b_1	0.0049	−0.0013	−0.0013	0.0026	0.0045	0.0045
b_2	0.0029	−0.0008	−0.0008	0.0015	0.0026	0.0026
μ_0	0.0001	0.1969	0.1969	−0.3740	−0.1425	−0.1425
μ_1	−0.0017	0.1974	0.1974	−0.3750	−0.1441	−0.1441
μ_2	−0.0015	−0.0135	−0.0135	0.0182	0.0051	0.0051
K	**0.9955**	−0.2707	−0.2707	0.5194	0.9022	0.9022
b_V	0.0045	0.2687	0.2687	−0.5156	−0.9007	−0.9007
β_H	−0.0064	−0.3802	−0.3802	**0.7295**	0.2744	0.2744
β_{V_A}	−0.0023	1.2848	**−1.5535**	0.2578	**1.3787**	−0.4780
β_{V_B}	−0.0023	**−1.5535**	1.2848	0.2578	−0.4780	**1.3787**
ψ	−0.0037	−0.5445	−0.5445	0.5020	0.1290	0.1290

Table 3: Normalized sensitivity indices for the endemic equilibrium, with $\beta_{V_A} = \beta_{V_B} = 0.04$. The index with the largest magnitude in each column is highlighted .

	N	i_A	i_B	i_{AB}	v_A	v_B
b_0	−0.0001	−0.0002	0.0000	−0.0003	−0.0003	−0.0001
b_1	0.0069	0.0137	−0.0020	0.0181	0.0218	0.0046
b_2	0.0005	0.0011	−0.0002	0.0014	0.0017	0.0004
μ_0	0.0010	−2.0283	0.3126	−2.5659	−2.4701	−0.0185
μ_1	−0.0011	−2.0325	0.3132	−2.5714	−2.4767	−0.0199
μ_2	−0.0013	0.9928	−0.1662	1.0611	1.0419	−0.0137
K	**0.9963**	1.9693	−0.2951	2.6143	3.1357	0.6643
b_V	0.0037	−1.9489	0.2920	−2.5885	−3.1110	−0.6641
β_H	−0.0058	3.0739	−0.4605	4.0827	3.9067	0.0474
β_{V_A}	−0.0044	**5.6675**	**−0.7816**	**4.7851**	**5.7717**	−0.0926
β_{V_B}	0.0008	−3.7186	0.4896	−2.1966	−2.6608	**0.7567**
ψ	−0.0047	3.6777	−0.7395	4.6645	4.5402	−0.0640

Table 4: Normalized sensitivity indices for the endemic equilibrium, with $\beta_{V_A} = 0.02$ and $\beta_{V_B} = 0.08$. The largest value in each column is highlighted.

We observe that the fractions of hosts and vectors with strain A, including coinfected hosts, is drastically reduced. Conversely, strain B is much more prevalent in the population, resulting in a greater total prevalence of infection than in Case 1.

3.5.1 Results on Prevalence of Infection

The host growth rates (b_0, b_1, b_2) do not substantially affect the equilibrium populations in either case considered above. The death rates (μ_0, μ_1, μ_2) are generally more influential, although the additional mortality of coinfected hosts (μ_2) has little impact when the strains are equivalent (transmission rates are the same). When they are different, increasing coinfection mortality unexpectedly increases the density of singly infected hosts and vectors with strain A, as well as the prevalence of coinfections.

As expected, in both cases, a 1% increase in carrying capacity K corresponds almost exactly to a 1% increase in total host population N. When strain A has smaller transmission rate, increases in K cause larger increases in i_A, i_{AB}, and v_A than when strains have equal transmission rates.

For equal strains, increasing the rate of vector turnover ($b_V = \mu_V$) by 1% strongly decreases the vector's disease prevalence (v_A and v_B both decrease by 0.9%). This makes biological sense as increasing vector mortality also increases vector birth rate, and all vectors are born uninfected. In addition, the single infected prevalences increase by 0.27% and coinfected prevalence decreases by 0.5%. When strain A has a slower rate of transmission, a 1% increase in b_V results in strong decreases in prevalence of strain A (1.9%) and of coinfection (2.6%) in the host; however the prevalence of strain B in the host stays about the same as in the case of equal transmission rates. In addition, the vector's disease prevalence of type A decreases by 3% while type B increases by 0.66%.

The parameter with the greatest impact on the prevalence of coinfected individuals was the vector-to-host transmission rate (a 1% decrease in β_H causes a 0.7% decrease

in i_{AB}) when the strains were equivalent, and the transmission rate of strain A (a 1% decrease in β_{V_A} causes a 4.8% decrease in i_{AB}) when this strain had a much smaller transmission rate.

With equivalent strains, density of i_A is most affected by β_{V_B}, and density of i_B by β_{V_A} (1% increases cause 1.6% decreases in the prevalences). The transmission rates of the strains associated with each compartment are also influential (1% increases cause 1.3% increases in the prevalences). However, for $\beta_{V_A} < \beta_{V_B}$, density of both i_A and i_B are both affected strongly by β_{V_A}, while the vector disease prevalences are affected by their correponding transmission rates (i.e. v_A affected more by β_{V_A}, while v_B affected more by β_{V_B}.

An increase in the parameter ψ represents reduced cross protection and lowered pathogen taxa similarity. A 1% increase raises the prevalence of coinfection by 0.5% if the transmission rates are the same, and by 4.7% if they are different. When strain A has a much lower transmission rate, a 1% increase in ψ also strongly increases the density of hosts and vectors with strain A, by 3.7% and 4.5% respectively.

4 Vector–Host Model with Logistic Vector Growth

The birth function (6) used in the previous analysis assumes an exponential vector growth rate. In addition, the analysis in Section 3.1 relies on the assumption (A1): the rate of exponential growth is zero, so the total vector population is a fixed constant. Biologically, this assumption may not be reasonable. We consider an alternative logistic birth function similar to that used by Jeger et al. [15],

$$r(V_{i,S}, V_{i,A}, V_{i,B}) = b_V N_{V,i} \left(1 - \frac{N_{V,i}}{mN}\right). \tag{22}$$

Using this function, the vector equations in the proportions model (13) for one vector species have the following form.

Vectors:

$$\begin{aligned}
\frac{dN_V}{dt} &= r_V N_V \left(1 - \frac{N_V}{\tilde{m}N}\right), \\
\frac{dv_A}{dt} &= \beta_{V_A} N v_S (i_A + i_{AB}) - b_V v_A \left(1 - \frac{N_V}{mN}\right), \\
\frac{dv_B}{dt} &= \beta_{V_B} N v_S (i_B + i_{AB}) - b_V v_B \left(1 - \frac{N_V}{mN}\right),
\end{aligned}$$

where

$$\tilde{m} := m\frac{r_V}{b_V}$$

represents the maximum number of vectors that each host can sustain (such that at a given host population N, the vector carrying capacity is $\tilde{m}N$). The equations for the host populations remain unchanged.

Figure 6 shows infection and coinfection prevalences for two values of m. As μ_V decreases (and thus r_V increases), prevalence increases (cf. Figure 6). Notably, using logistic vector growth, prevalence is almost always higher with two specialist vectors

than with a single generalist (except for $\beta = 0.02$ in the first case shown in Figure 6). This is the reverse of what is observed in the case of a constant vector population (cf. Figure 4).

We also examine the sensitivities of the endemic equilibrium components as in Section 3.5, setting $m = 100$, $b_V = 12$, and $\mu_V = 11$ with all other parameters as in Table 2. When both strains have the same transmission rate $\beta_{V_A} = \beta_{V_B} = 0.4$, the equilibrium is

$$\begin{aligned} X &= \begin{bmatrix} N & i_A & i_B & i_{AB} & N_V & v_A & v_B \end{bmatrix}^T \\ &= \begin{bmatrix} 97.58 & 0.04275 & 0.04275 & 0.8914 & 813.2 & 0.1993 & 0.1993 \end{bmatrix}^T. \end{aligned}$$

Compared to the equilibrium using the exponential growth function given in (20), the host population is very similar (98 versus 99), but the prevalence of isolated infections is much lower (8.6% versus 38%) and coinfection is far more prevalent (89% versus 44%). The prevalence of infection in vectors is not greatly increased (40% versus 30%), although the vector population has stabilized to 813 individuals compared to the fixed population of 100 from before.

When the strains have different rates ($\beta_{V_A} = 0.2$, $\beta_{V_B} = 0.8$) the equilibrium is

$$X = \begin{bmatrix} 97.73 & 0.007948 & 0.1524 & 0.8202 & 814.40 & 0.08003 & 0.3759 \end{bmatrix}^T.$$

Comparing to the exponential growth analog (21), the host populations and the prevalence of isolated cases of strain A are very similar, but isolated cases of strain B are scarce (15% versus 73%) and coinfection is common (82% versus 12%).

The normalized sensitivities are given in Tables 5 and 6. Overall, the most influential parameters are by far the vector's birth and death rates (b_V and μ_v). For example, a 1% increase in vector mortality results in 1.7% decrease in prevalence of

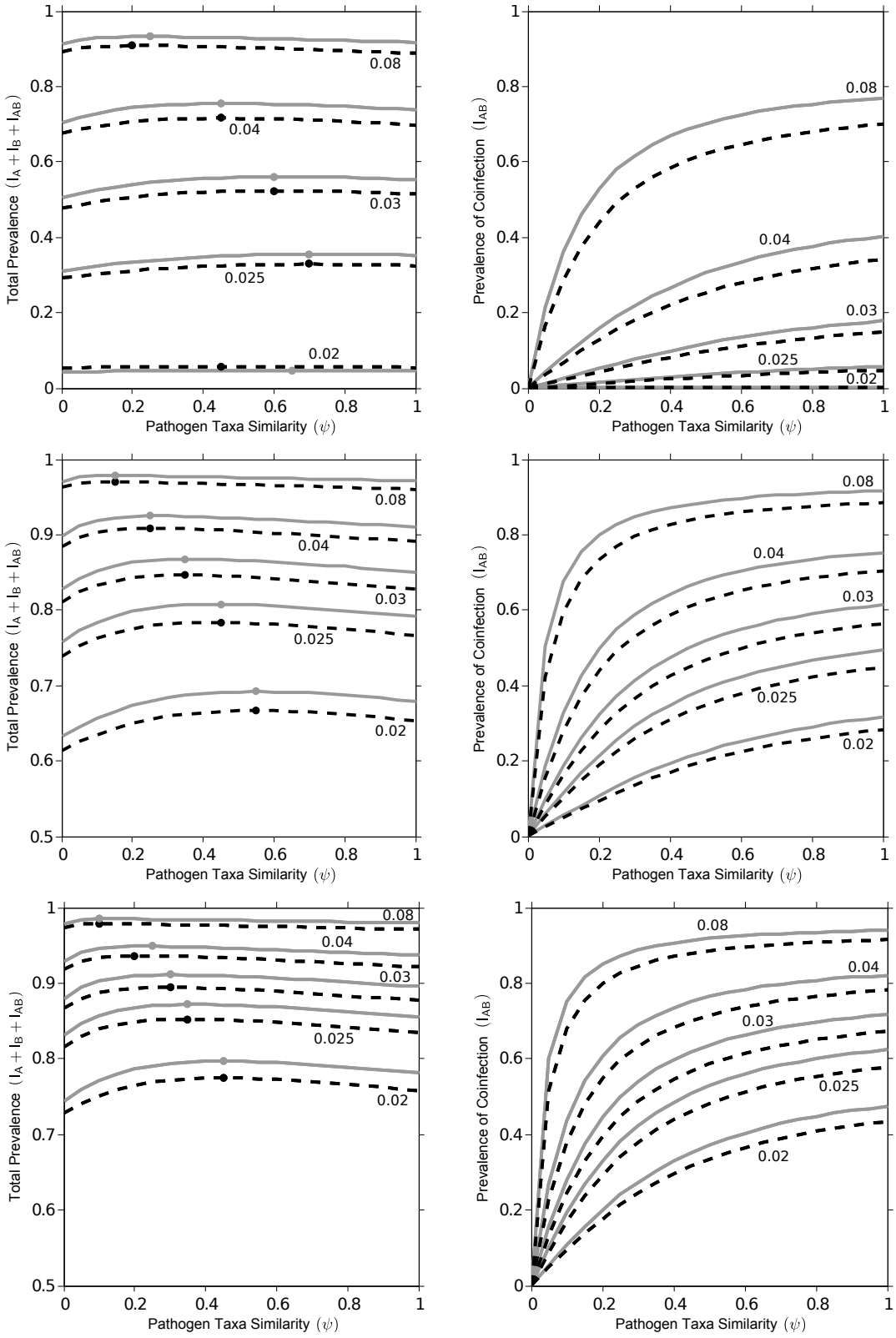

Figure 6: Plots of prevalence assuming logistic vector growth with birth rate $b_V = 12$. Top: $\mu_V = 11$, $m = 100$. Center: $\mu_V = 11$, $m = 300$. Bottom: $\mu_V = 10$, $m = 100$. All other parameters are as in Table 2.

coinfection when the strains have equal transmission rates, and a 27.8% increase in coinfection prevalence when the strains have different rates. This also increases the prevalence of singly infected hosts by 10.2% in the first case and in the second case, i_A decreases by 20% while i_B decreases by 41%.

The vector's per-host carrying capacity m is also influential, although a 1% change in this parameter never changes the prevalences by more than 2%. The pathogen transmission rates remain fairly influential, as they were under the assumption of constant vector population.

Since the parameters governing vector growth are critical, these parameters should be estimated with a high degree of accuracy to ensure the model output is reasonable.

	N	i_A	i_B	i_{AB}	N_V	v_A	v_B
b_0	-0.0009	0.0013	0.0013	-0.0002	-0.0009	-0.0006	-0.0006
b_1	0.0037	-0.0052	-0.0052	0.0006	0.0037	0.0024	0.0024
b_2	0.0191	-0.0271	-0.0271	0.0034	0.0191	0.0126	0.0126
μ_0	-0.0057	0.3792	0.3792	-0.0472	-0.0057	-0.0201	-0.0201
μ_1	-0.0084	0.3830	0.3830	-0.0477	-0.0084	-0.0218	-0.0218
μ_2	-0.0039	0.1511	0.1511	-0.0184	-0.0039	-0.0088	-0.0088
K	$\mathbf{0.9939}$	-1.4128	-1.4128	0.1753	0.9939	0.6595	0.6595
b_V	-0.0416	-9.7039	-9.7039	1.2038	$\mathbf{10.9577}$	0.3988	0.3988
μ_V	0.0438	$\mathbf{10.2345}$	$\mathbf{10.2345}$	$\mathbf{-1.2696}$	-10.9554	$\mathbf{-1.0219}$	$\mathbf{-1.0219}$
β_H	-0.0038	-0.8822	-0.8822	0.1094	-0.0038	0.0363	0.0363
β_{V_A}	-0.0011	0.7907	-1.3212	0.0329	-0.0011	0.8600	-0.2368
β_{V_B}	-0.0011	-1.3212	0.7907	0.0329	-0.0011	-0.2368	0.8600
ψ	-0.0060	-0.7520	-0.7520	0.0676	-0.0060	0.0145	0.0145
m	-0.0038	-0.8822	-0.8822	0.1094	0.9962	0.0363	0.0363

Table 5: Normalized sensitivity indices for the endemic equilibrium using a logistic vector growth function, with $\beta_{V_A} = \beta_{V_B} = 0.04$. The largest value in each column is highlighted.

	N	i_A	i_B	i_{AB}	N_V	v_A	v_B
b_0	-0.0007	-0.0003	-0.0007	0.0005	0.0013	0.0001	-0.0004
b_1	0.0085	0.0040	0.0087	-0.0059	-0.0156	-0.0007	0.0044
b_2	0.0071	0.0033	0.0073	-0.0049	-0.0130	-0.0006	0.0037
μ_0	-0.0020	0.1761	0.3657	-0.2489	0.0036	-0.2175	-0.0023
μ_1	-0.0045	0.1749	0.3631	-0.2471	0.0083	-0.2173	-0.0037
μ_2	-0.0021	0.0019	0.0371	-0.0232	0.0038	-0.0220	-0.0005
K	$\mathbf{1.0083}$	0.4693	1.0287	-0.6967	-1.8472	-0.0824	0.5223
b_V	0.3535	$\mathbf{19.9490}$	$\mathbf{43.7342}$	$\mathbf{-29.6193}$	$\mathbf{-54.7840}$	$\mathbf{-27.2577}$	$\mathbf{-1.5494}$
μ_V	-0.3316	-18.7105	-41.0191	27.7805	51.9117	25.0367	0.9243
β_H	-0.0063	-0.3577	-0.7843	0.5312	0.0116	0.4596	-0.0014
β_{V_A}	-0.0050	1.5621	-0.9886	0.5334	0.0092	1.4715	-0.1093
β_{V_B}	0.0014	-1.7680	0.5372	-0.2277	-0.0025	-0.6314	0.6841
ψ	-0.0065	-0.3930	-0.7821	0.4528	0.0119	0.4046	-0.0280
m	0.0120	0.6751	1.4800	-1.0023	-1.8539	-0.9224	-0.0524

Table 6: Normalized sensitivity indices for the endemic equilibrium using a logistic vector growth function, with $\beta_{V_A} = 0.02$ and $\beta_{V_B} = 0.08$. The largest value in each column is highlighted.

5 Conclusions and Future Work

In this thesis, we have considered a modified version of a vector–host model for coinfection by two strains of the pathogen barley/cereal yellow dwarf virus that was initially constructed by Seabloom et al. [26]. Whereas in [26], the authors use a quasiequilibrium argument to reduce the number of differential equations by assuming that vector numbers are at an equilibrium, in our model we explicitly track the vector dynamics. With this modification our model can be easily extended to incorporate multiple vector species. We consider two different vector growth functions, a constant vector population and logistic vector growth, and analyze the case of a single-vector, single-host, two-pathogen-strain model. The parameters of the model are fit to data given in [26]. We use this data as our baseline data for the sensitivity analysis of the infected coexistence equilibrium for our models.

We first analyze the case of a constant vector population. We compute the basic and type reproduction numbers for this model and prove the linear stability of the disease-free equilibrium. The infected coexistence equilibrium proves to be analytically intractable and we have used sensitivity analysis on this equilibrium to understand which of the 12 parameters (using baseline values) involved in the model are the most influential for the prevalence of infection and coinfection.

The linear stability analysis of the disease free equilibrium (DFE) for the single-host, single-vector (at constant total population) and two-pathogen strain model in Section 3.1 and accompanying Figure 3 show that the basic reproduction number $\mathcal{R}_0$ does not completely describe the model's long-term dynamics. This is because initial disease transmission is directly related to $\mathcal{R}_0$, but disease prevalence is directly related to the endemic or infected coexistence equilibrium. Thus, the dynamics of coinfection cannot be deduced from a knowledge of $\mathcal{R}_0$, and we must analyze the infected coexistence equilibrium to understand coinfection dynamics.

The basic reproduction number $\mathcal{R}_0$ can be calculated from the type reproduction

numbers $\mathcal{T}_A$ and $\mathcal{T}_B$, which describe individual strain persistence. Coinfection may not be possible if $\mathcal{R}_0 > 1$, which only indicates that at least one viral strain persists, but if both $\mathcal{T}_A > 1$ and $\mathcal{T}_B > 1$, then both type of strains persist and coinfection occurs if cross-protection is incomplete.

Furthermore, neither the basic nor type reproduction numbers depend on strain similarity or mortality of coinfected hosts. The computed explicit formula for $\mathcal{R}_0$ gives an indication of control efforts that can be adopted to lower the initial transmission of disease. In particular the reproductive numbers can be reduced by reducing the pathogen transmission rates (β_H, β_{V_A}, and β_{V_B}) and carrying capacities of both host and vector (K and K_V). In addition, increased mortality of susceptible (μ_0) and singly infected (μ_1) hosts reduces the reproductive numbers, as does faster vector replacement, i.e. elevated birth (b_V) and death rates (μ_V). This is reasonable since we assume a constant vector population, so each vector death corresponds to a vector birth, and all vectors are born uninfected.

The sensitivity analysis of the infected coexistence equilibrium of the model with constant vector population in Section 3.5 suggests that the disease transmission rates have the most influence on equilibrium populations sizes of infected compartments. The host's birth and death rates have relatively little influence, unlike the vector's. The strain similarity and cross-protection factor (ψ) has fairly strong influence on the infected and coinfected host compartments; in particular, if one strain has higher transmission rate, then ψ becomes highly influential to all compartments whose members are infected by the other strain (including hosts, vectors, and coinfections).

For the model with logistic vector growth, we have only conducted a sensitivity analysis of the infected coexistence equilibrium in Section 4. This analysis indicates that changing the vector population dynamics changes the infection and coinfection dynamics of the vector–host model. In particular, the most influential parameters for infection and coinfection dynamics now are the vector's birth and death rates (b_V

and μ_v), with the vector's per-host carrying capacity (m) also playing a critical role. The pathogen transmission rates also remain influential in the infection dynamics, but the parameters governing vector growth seem to be the most critical.

Our results show that the vector growth function used makes a difference in the relative importance of parameters in the spread and coinfection of the host by B/CYDV. Numerical simulations of the two models with different vector growth functions indicate that the relative magnitude of infected and coinfected prevalences between specialist and generalist vectors seem to depend on the particular vector population dynamics being modeled.

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Appendices

A Glossary

Basic reproduction number The average number of secondary infections over the lifetime of a single infectious individual introduced into an entirely susceptible population of given size [19].

Carrying capacity The maximum sustainable population of a species. A population at carrying capacity has zero net growth as its birth and death rates are equal and opposite.

Contact rate The rate at which susceptible and infectious individuals meet such a way that disease transmission may occur.

Coinfection When an individual host is infected by multiple viruses or viral strain simultaneously.

Cross-protection An effect triggered by a virus that protects its host against further infections, e.g. by inoculating it or by elevating its immune response.

Disease-free equilibrium An equilibrium at which the disease has become and remains extinct, so all infected population classes have zero population. In most models, the population then comprises only susceptible individuals.

Density-dependent transmission A model for disease transmission in which contact rate is proportional to the population of infectious individuals [19,28]. Also known as *mass action transmission*.

Equilibrium A set of population densities (e.g. the susceptible and infectious populations) at which the flow of individuals into each compartment exactly balances the flow out, so the compartmental populations are stable and constant.

Endemic equilibrium An equilibrium at which the disease persists indefinitely, i.e. where the population of infectious individuals stabilizes at some nonzero density rather than the disease dying out.

Standard incidence disease transmission A model for disease transmission in which contact rate is proportional to the infected fraction of the population [19] but independent of the total population itself [28]. Also known as *frequency-dependent transmission*.

Incidence The rate at which new cases of a disease occur within a given population [9].

Trivial equilibrium The equilibrium at which all population compartments have zero density. This equilibrium exists in most models for closed populations that do not include immigration or emigration.

Type-reproduction number The expected number of cases in individuals of type i caused by one infected individual of type i in a completely susceptible population, either directly or through chains of infection passing through any sequence of the other types [23].

Population pressure The population size at which the net birth rate is reduced to zero, due to factors such as overcrowding and intraspecific competition. This would be the population carrying capacity in the absence of death.

Prevalence The number of cases of infection within a given population or the proportion of which is infected [19].

Vector An intermediate agent, such as a mosquito, aphid, or tick, that transmits a pathogen between hosts.

Virulence The disease-induced mortality of a host [2].

B Vector Quasiequilibrium Analysis

The system (1) and (3) is a generalization of that analyzed by Seabloom et al. [26], obtained by relaxing the assumption of $n = 2$ vector species and constant vector populations. Seabloom et al. use a quasiequilibrium (pseudoequilibrium) argument, based on constant vector populations, to model vector populations implicitly as a function of given host populations. This simplifies the model by reducing the dimension of the system.

Extending this argument to the n-species case, we suppose that the vector population $N_{V,i}$ for each species i is fixed at a constant $N^*_{V,i} > 0$. Then from (4), this implies $r(t) = \mu_V N^*_{V,i}$. Since $\frac{dV_{i,S}}{dt} = 0$, the equation (3a) gives

$$V_{i,S} = \frac{\mu_V}{\beta_{V_{i,A}}(I^*_A + I^*_{AB}) + \beta_{V_{i,B}}(I^*_B + I^*_{AB}) + \mu_V} N^*_{V,i}.$$

Using this, it follows from (3b) and (3c) that

$$V_{i,A} = \frac{\beta_{V_{i,A}}(I^*_A + I^*_{AB})}{\beta_{V_{i,A}}(I^*_A + I^*_{AB}) + \beta_{V_{i,B}}(I^*_B + I^*_{AB}) + \mu_V} N^*_{V,i},$$
$$V_{i,B} = \frac{\beta_{V_{i,B}}(I^*_B + I^*_{AB})}{\beta_{V_{i,A}}(I^*_A + I^*_{AB}) + \beta_{V_{i,B}}(I^*_B + I^*_{AB}) + \mu_V} N^*_{V,i}.$$

In the case $n = 2$, by setting $V_A = V_{1,A} + V_{2,A}$, $V_B = V_{1,B} + V_{2,B}$, $X = N^*_{V,1}$ and $Y = N^*_{V,2}$, this is directly equivalent to the vector populations obtained and used by Seabloom et al. [26].