Coexistence of Competing Species with a Directly Transmitted Pathogen

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Abstract

In this paper we consider models of two competing species that are both affected by a pathogen which is transmitted directly. We consider both mass action as well as frequency incidence models of disease spread, and Lotka-Volterra competition. Our aim is to address the interaction between competition and disease dynamics. We do this by presenting previously known results along with new results in a unified manner that stresses the role of the basic reproduction number as well as the relative strengths of intra- versus inter-specific competition for both species.

For both types of disease models we compute the basic reproduction numbers. For the mass action case we compute all the equilibria except infected coexistence, and analyze the equilibria for their stability. To analyze infected coexistence, we consider a simplified ecologically relevant model and prove a conjecture made in [18, 9] about the stability of the infected coexistence equilibrium. The model with frequency incidence is shown to have a unique endemic equilibrium whose existence and stability depends on the value of the basic reproduction number.

Keywords: Lotka-Volterra Competition, SI disease, basic reproduction number, coexistence

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1 Introduction

Competitive interactions as well as predator prey dynamics have dominated investigations of species interactions in ecology and influence community structure via the distribution, abundance and resource use of species in natural communities [35, 14, 17]. Classical competition theory predicts competitive exclusion of species with similar requirements. An important ecological question is as follows: what mechanisms drive the coexistence of competing species?

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The effect that parasites and pathogens have on the coexistence or exclusion of their hosts and in the structure of biological communities is also important in community ecology. Pathogens can be a very important controlling factor in wildlife communities. For example, it was found that during the first half of the 20th century, the wildebeest herds in the Serengeti were in fact being regulated not exclusively by predator prey interactions, but primarily by a virus called rinderpest. Once rinderpest was controlled through vaccination, both predator and prey populations in the area changed dramatically [16]. Pathogens seem to be difficult to detect and quantify in an empirical study, but host-pathogen interactions can be studied through mathematical models that combine elements of population dynamics and epidemiology [7, 12, 13]. Such models can give important qualitative insight into the effects of pathogens on plant and animal populations and the factors that influence species coexistence or exclusion in communities [5, 26, 18].

Both theoretical and empirical investigations have shown that a generalist pathogen can alter host species diversity and community composition [7, 10, 23, 27, 29, 32]. Empirical studies have also demonstrated the importance of the combined effects of inter- and intra-specific competition between species and the effects of pathogens (apparent competition) on the population dynamics of multi-host systems [20]. A review of empirical studies in [15] finds strong evidence for parasite-induced extinction of one species (usually a native species replaced by an introduced exotic) as a result of the reservoir effects of apparent competition.

Recent work suggests that multi-host pathogens can mediate the outcome of inter-specific competition, facilitating and maintaining invasion by novel species [7]. For example, in [38], the authors show that it is likely that a shared disease, in addition to competition for space and food, is the impetus for the continued decline of the native red squirrel in the U.K. in the presence of the introduced grey squirrel. Another experimental study in [28] considered the effects of a pathogenic water mold on competitive interactions between two larval amphibian species in the Cascade mountains of Oregon, USA. The authors in this work showed that the presence of the mold reversed the outcome of competitive interactions between the two species. Similarly, the composition of the host community can control pathogen dynamics. For example, in one experiment with a generalist grass pathogen, all grass species in treatments containing a highly competent virus reservoir species had higher pathogen prevalence compared to their counterparts in communities lacking the spillover species [32]. As in this example, the composition of host communities can cause either amplification or fadeout of a pathogen. Thus we can see that the interaction between community and disease ecology can help us understand the structure of a biological system and the reasons why species coexist with each other [25].

Mathematical models that include competition between multiple species in addition to a shared pathogen are notoriously hard to analyze. The correct choice of the type of disease incidence (for example, mass action or frequency incidence transmission) depends on many factors. These include the species that is infected, the transmission routes of infection of the disease, and population sizes, among other things. In [6], the authors considered the cowpox virus in coexisting populations of bank voles and wood mice. Their analysis indicates that for each species in isolation frequency dependent transmission is a superior descriptor. In [33, 39] the authors used a SIR/SI type model with mass action disease transmission and density independent death rates to study the effects of a parapoxvirus in competing grey/red squirrel species in the United Kingdom. Using parameters estimated from data they found

that the invading grey squirrels eventually win the competition, displacing the native red squirrel. They also found that the presence of disease speeds up this process of replacement. We refer the reader to the papers [24, 4, 30] for a good synopsis of different disease incidence types and their appropriate use.

1.1 Interacting Species and Disease

Two species models in which one or both species share a common pathogen and may or may not interact competitively have been discussed in several papers. Models for two host species which share a pathogen but do not compete directly have been studied in [26, 5, 18, 24]. Although the equilibria that result in the exclusion of one of the species were analyzed, finding conditions for the stability of the coexistence equilibria proved to be quite difficult. Through numerical simulations it was found that two host SIS models with mass action incidence can have complicated behaviors including several infected coexistence equilibria and multiple attractive periodic solutions.

In [11], the authors consider a model of the population dynamics of two host species which share a common pathogen, but do not interact competitively and do not self regulate. The transmission of disease is via mass action. This model, developed in [26], evolved from a single host model considered in [2]. The authors in [11] identified circumstances under which the shared pathogen leads to the coexistence of the two host species in either a periodic or persistent form that depend largely on the overall growth and death rates. This study shows the importance of the differences in birth rates and death rates of the two hosts.

In [3, 42, 41] the authors consider a two species model in which both species compete directly and one is subject to a pathogen. The models assume mass action transmission of disease and in [42] the existence of limit cycles is shown. In [41] it was found that in the absence of disease there is competitive exclusion between the two species and the presence of disease can lead to stable or oscillatory coexistence of both species.

In [9], the authors consider a two species model in which both species compete via Lotka-Volterra competition and both species share a common pathogen transmitted via mass action. In this model death rates were density independent. They partially analyzed their model using the notions of forces of infection and invasion criteria. These criteria determine whether resident populations allow small invasions of other species to prosper or cause them to decay. They are therefore relevant to questions of species coexistence or exclusion and allow biologically motivated classifications of such long term outcomes to be obtained. As with previous models, the coexistence equilibria proved impossible to fully analyze. In [22], the authors consider a model with Lotka-Volterra competition between the two species which share a common pathogen. Mass action disease transmission is used in the model which in its complete generality is intractable. Both density-dependent and disease related death rates are considered. The birth rates are density dependent, but do not include inter-specific effects. The authors provide local stability results only for boundary equilibria, and mainly concentrate on conditions that guarantee the persistence of either hosts or pathogens. Using Hopf bifurcation theory and numerical simulations, complex behaviors of a simplified model are demonstrated.

In [21] the authors considered an SIRS epidemic model of two competitive species using frequency incidence disease transmission with no disease related deaths. Under these con-

ditions, the authors in [21] were able to show stability conditions for all possible equilibria. In [24] the authors considered many different models with frequency incidence disease transmission. The models were shown to have the classic endemic model behavior; the disease dies out below a threshold and approaches an endemic equilibrium above the threshold.

1.2 Outline of Paper

In this paper we consider two species models in which both the species compete directly via Lotka-Volterra competition and share a directly transmitted pathogen. We consider both mass action and frequency incidence type transmission and investigate the stability of the infected coexistence equilibrium. We present previously known results in this area along with new results in a unified setting that simplifies the analysis, and stresses the role of the basic reproduction number as well as the relative strengths of intra- versus inter-specific competition for both species. This unification makes our new results more intuitive from both a mathematical and ecological point of view.

Our results extend the work done in [9] for the case of mass action disease transmission. Similar to the mass action model in [9], we keep the natural mortality rates for the two species to be density-independent, while the birth rates are density dependent. Our motivation for this choice comes from the case of the red/grey squirrel system discussed in [33, 39], and other similar systems [28, 37, 36]. In this aspect our model differs from other similar two species models analyzed in [24, 22, 21]. As opposed to the model in [9] we also consider frequency incidence disease transmission. Our results provide a rigorous mathematical analysis as opposed to the biologically motivated analysis provided in [9], except for the case of infected coexistence which was not analyzed in [9]. For this case we consider an ecologically simplified version of the mass action model in which a complete analysis of infected coexistence is provided. Such an analysis is usually not possible in the most general case, and to our knowledge is not attempted in any of the papers mentioned for the case of density dependent birth rates and mass action transmission. Our paper has two main new results:

1. In [5, 18, 9] a conjecture was made, based on numerical simulations, about the stability of the infected coexistence equilibrium for the model with mass action disease transmission. The conjecture stated that the conditions under which this equilibrium is stable cause all the other equilibria to be unstable. We prove this conjecture for a special case in which the infected coexistence equilibrium is tractable. This allows the full analysis of the infected coexistence equilibrium to be achieved. Such an analysis is usually not possible.

2. For the case of frequency incidence transmission we prove existence and uniqueness of an infected coexistence equilibrium. We also prove partial results on stability of infected coexistence with density dependent birth rates which include inter-specific effects. In the literature one can find similar results for the case of density dependent death rates with no inter-specific effects, and density independent birth rates [31]. However, the analysis is simpler than the case that we consider here.

The outline of the paper is as follows. In Section 2 we develop a model for two competitive species that share a common pathogen. In Section 3 we analyze the two species competition

model assuming mass action disease transmission. Using local stability analysis we compute the basic reproduction number for the model and analyze all the equilibria except the infected coexistence equilibrium which is intractable. We consider a special ecologically relevant case [8] in which infected coexistence is tractable and analyze this case completely. For the special case we prove a conjecture made in [5, 18, 9] about the stability of the infected coexistence equilibria.

In Section 4 we analyze the behavior of a two species competition model with frequency incidence disease transmission. We compute the basic reproduction number R_0 and stability conditions for the infected coexistence equilibrium. We present conclusions in Section 5.

2 Two Species Models Combining Population Dynamics and Disease Transmission

We consider two species models which incorporate a species birth function and a disease incidence function in the form

$$\frac{dS_1}{dt} = g_1(N_1, N_2)N_1 - b_1 S_1 - \mathcal{I}_1(I_1, I_2)S_1, \tag{2.1a}$$

$$\frac{dS_2}{dt} = g_2(N_1, N_2)N_2 - b_2 S_2 - \mathcal{I}_2(I_1, I_2)S_2, \tag{2.1b}$$

$$\frac{dI_1}{dt} = \mathcal{I}_1(I_1, I_2)S_1 - \Gamma_1 I_1, \tag{2.1c}$$

$$\frac{dI_2}{dt} = \mathcal{I}_2(I_1, I_2)S_2 - \Gamma_2 I_2. \tag{2.1d}$$

For $i, j = 1, 2$, S_i denotes the density of susceptible individuals in the population of species i, I_i represents the density of infected individuals in the population of species i, and $N_i = S_i + I_i$ is the total population density of species i. We assume that the birth terms are density-dependent. Thus the birth functions for species i, denoted by g_i, are functions of N_1 and N_2. We consider Lotka-Volterra competition including both intra-specific and inter-specific competition. Thus, the birth functions for the two species are

$$g_1(N_1, N_2) = a_1\left(1 - \frac{N_1}{\theta_{11}} - \frac{N_2}{\theta_{12}}\right), \tag{2.2a}$$

$$g_2(N_1, N_2) = a_2\left(1 - \frac{N_2}{\theta_{22}} - \frac{N_1}{\theta_{21}}\right). \tag{2.2b}$$

We define $K_{ij} := \dfrac{r_i\theta_{ij}}{a_i}$. The carrying capacity for species i is K_{ii} and the terms θ_{ij}^{-1} for $i \neq j$ are competition coefficients. Here $r_i := a_i - b_i$ is the intrinsic per capita growth rate for species i, with $a_i(1 - N_i/\theta_{ii})$, and b_i, the per capita birth and natural death rates, respectively, for species i in isolation. We assume that $a_i > b_i > 0$ and hence $r_i > 0$ for $i = 1, 2$. The terms $\Gamma_i := \alpha_i + b_i$, for species i, are per capita net rates of loss of infected individuals incorporating death due to disease $\alpha_i \geq 0$, and natural mortality b_i.

The disease transmission term, given here by the disease incidence functions $\mathcal{I}_i$ for species i, describes the rate at which susceptible hosts are converted into infected hosts by their contact with infectious material. Transmission is the driving force in the dynamics of any infectious disease and hence the functions $\mathcal{I}_i$ are a very important part of epidemiological models. We model the disease incidence functions $\mathcal{I}_i$ as

$$\mathcal{I}_1(I_1, I_2) = \Lambda_{11}(N_1)\frac{I_1}{N_1} + \Lambda_{12}(N_2)\frac{I_2}{N_2}, \tag{2.3a}$$

$$\mathcal{I}_2(I_1, I_2) = \Lambda_{22}(N_2)\frac{I_2}{N_2} + \Lambda_{21}(N_1)\frac{I_1}{N_1}, \tag{2.3b}$$

with an intra-species transmission term with rate $\Lambda_{ii}(N_i)$, and an inter-species transmission term with rate $\Lambda_{ij}(N_j)$ for $i = 1, 2$. Depending on whether the mass action or frequency incidence approach is used these transmission terms take on different forms.

3 Two Species Models with Lotka-Volterra Competition and Mass Action Disease Transmission

In this section we consider two species models in which the disease transmission dynamics follows the mass action approach. The intra-species and inter-species transmission rates in (2.3a)-(2.3b) are defined as

$$\Lambda_{ij}(N_j) = \beta_{ij}N_j, \tag{3.1}$$

where for $i, j = 1, 2$, $\beta_{ij} > 0$ and are constants. From (2.3a), (2.3b) and (3.1) we can write the disease incidence functions as

$$\mathcal{I}_1(I_1, I_2) = \beta_{11}I_1 + \beta_{12}I_2, \tag{3.2a}$$

$$\mathcal{I}_2(I_1, I_2) = \beta_{21}I_1 + \beta_{22}I_2. \tag{3.2b}$$

In [26, 24], the authors consider mass action transmission dynamics and the case of no direct competition, either within or between two species. Thus, the birth functions g_1 and g_2 were modeled as exponential growth and each population increases exponentially in the absence of the disease. In [5, 24], each of the host populations is subject to self-regulation (intra-specific competition) and settle at their individual carrying capacity in the absence of disease. In this case, the birth functions g_1 and g_2 were modeled as logistic growth and mass action disease transmission is used.

In [9, 19, 24, 22], both hosts are inhibited by intra- and/or interspecific competition and the birth functions g_1 and g_2 are modeled as Lotka-Volterra type competition along with mass action disease transmission. As mentioned in the introduction, the model that we consider in this section is the same as that considered in [9], in which the authors partially analyzed the model from a biological perspective to determine stability conditions for exclusion and uninfected coexistence equilibria.

Using the birth functions defined in (2.2a)-(2.2b) and the disease incidence functions

defined in (3.2a)-(3.2b) we obtain the two species SI model

$$\frac{dS_1}{dt} = a_1\left(1 - \frac{N_1}{\theta_{11}} - \frac{N_2}{\theta_{12}}\right)N_1 - b_1S_1 - (\beta_{11}I_1 + \beta_{12}I_2)S_1, \tag{3.3}$$

$$\frac{dS_2}{dt} = a_2\left(1 - \frac{N_2}{\theta_{22}} - \frac{N_1}{\theta_{21}}\right)N_2 - b_2S_2 - (\beta_{22}I_2 + \beta_{21}I_1)S_2, \tag{3.4}$$

$$\frac{dI_1}{dt} = (\beta_{11}I_1 + \beta_{12}I_2)S_1 - \Gamma_1 I_1, \tag{3.5}$$

$$\frac{dI_2}{dt} = (\beta_{22}I_2 + \beta_{21}I_1)S_2 - \Gamma_2 I_2. \tag{3.6}$$

The model (3.3)-(3.6) makes ecological sense and is mathematically well-posed in the domain $\mathcal{D}^1 = \{(S_1, S_2, I_1, I_2) \in \mathbb{R}^4 | S_1, S_2, I_1, I_2 \geq 0, 0 \leq N_i \leq K_{ii}, i = 1, 2\}$.

Before analyzing model (3.3)-(3.6) we summarize from the literature the relevant results for a two species pure competition model and a single species SI mass action disease model with logistic growth in the species.

3.1 The Logistic Growth and Mass Action Disease Model for a Single Species

In this section, we summarize from the literature results of the analysis of the SI disease model for one species with mass action transmission (see for e.g., [5, 22]). Our contribution here is to rewrite the coexistence equilibria in a form that stressed the role of the basic reproduction number, $\mathcal{R}_0 \leq 1$, of the species. We will use this same form for the equilibria of the combined competition and disease model for two species (3.3)-(3.6).

Consider the single species SI model with logistic growth in the species,

$$\frac{dS}{dt} = a\left(1 - \frac{N}{\theta}\right)N - bS - \beta SI, \tag{3.7}$$

$$\frac{dI}{dt} = \beta SI - \Gamma I, \tag{3.8}$$

where the variables and parameters have the same meaning as in Section 3 with $\Gamma = \alpha + b$, and $N = S + I$. The model (3.7)-(3.8) is well-posed on the domain $\Omega^D = \{(S, I)^T | S, I \geq 0, 0 \leq N \leq K\}$. Let $r = a - b > 0$. The carrying capacity of the species is $K = \dfrac{r\theta}{a}$. The equilibria for model (3.7)-(3.8) can be written in the form $E_1^D = (0, 0)$, $E_2^D = (K, 0)$, and

$$E_3^D = \left(\frac{\Gamma}{\beta}, \frac{\Gamma}{\beta}\left[-\left(1 + \frac{\mathcal{R}_0\lambda}{2}\right) + \sqrt{\left(1 + \frac{\mathcal{R}_0\lambda}{2}\right)^2 + (\mathcal{R}_0 - 1)}\right]\right), \tag{3.9}$$

where $\lambda = \dfrac{\alpha - r}{r}$ and $\mathcal{R}_0 = \dfrac{\beta K}{\Gamma}$ is the basic reproduction number for the model. We have the following result.

Lemma 3.1 *For the model* (3.7)-(3.8)*, the trivial equilibrium E_1^D is always unstable. If $\mathcal{R}_0 < 1$ then the disease-free equilibrium E_2^D is globally asymptotically stable in the domain Ω^D. If $\mathcal{R}_0 > 1$ then the infected coexistence equilibrium E_3^D is globally asymptotically stable in the domain Ω^D.*

3.2 The Pure Competition Model for Two Species

We summarize, from the literature (see for e.g., [22]), the analysis of the two-species Lotka-Volterra (pure) competition model. Our contribution here is to rewrite the equilibria of the pure competition model in a form that involves two parameters ξ_1 and ξ_2, as defined below in (3.14)-(3.15). This form of the equilibria simplifies the analysis and we obtain stability results based on the values taken by these two parameters. This is very useful as we can apply similar notation to the computation of equilibria of the two competitive species SI model with mass action disease transmission (3.3)-(3.6), and again obtain stability results depending on the values that the parameters ξ_1 and ξ_2 assume.

Consider the two species model with Lotka-Volterra competition,

$$\frac{dN_1}{dt} = r_1 \left(1 - \frac{N_1}{K_{11}} - \frac{N_2}{K_{12}}\right) N_1, \tag{3.10}$$

$$\frac{dN_2}{dt} = r_2 \left(1 - \frac{N_2}{K_{22}} - \frac{N_1}{K_{21}}\right) N_2, \tag{3.11}$$

where N_i is the total population density of species i, for $i = 1, 2$. This model is well-posed on the domain $\Omega^C = \{(N_1, N_2)^T | 0 \leq N_i \leq K_{ii}, i = 1, 2\}$. The parameters r_i, K_{ij} have the same meaning as described in Section 2. The equilibria for model (3.10)-(3.11) are $E_1^C = (0, 0)$, $E_2^C = (K_{11}, 0)$, $E_3^C = (0, K_{22})$, and the coexistence equilibrium $E_4^C = (N_1^C, N_2^C)$, where

$$N_1^C = \frac{K_{11}K_{12}}{K_{12} + K_{11}(\xi_1/\xi_2)}, \tag{3.12}$$

$$N_2^C = \frac{\xi_1}{\xi_2} N_1^C. \tag{3.13}$$

The parameters ξ_1, and ξ_2 are defined as

$$\xi_1 := \frac{1}{K_{11}} - \frac{1}{K_{21}}, \tag{3.14}$$

$$\xi_2 := \frac{1}{K_{22}} - \frac{1}{K_{12}}. \tag{3.15}$$

For this pure competition model, the existence (feasibility) and stability of equilibria depend on the positivity or negativity of the parameters ξ_1 and ξ_2. We can interpret the term $1/K_{ij}$ as the inhibition strength of species j on species i [34]. We have the following result

Lemma 3.2 *For the pure competition model* (3.10)-(3.11)*, the trivial equilibrium E_1^C is always unstable. In addition, we have the following cases*

1. *$\xi_1 > 0, \xi_2 > 0$: Intra-specific competition is stronger than inter-specific competition for both species. The equilibria E_2^C, E_3^C are unstable while E_4^C is globally asymptotically stable in the domain Ω^C.*

2. *$\xi_1 < 0, \xi_2 > 0$: Intra-specific competition is stronger for species 2 and inter-specific competition is stronger for species 1. E_4^C is not feasible. E_2^C is globally asymptotically stable, while E_3^C is unstable.*

3. $\xi_1 > 0, \xi_2 < 0$: *Intra-specific competition is stronger for species 1 and inter-specific competition is stronger for species 2.* E_4^C *is not feasible.* E_3^C *is globally asymptotically stable, while* E_2^C *is unstable.*

4. $\xi_1 < 0, \xi_2 < 0$: *Inter-specific competition is stronger than intra-specific competition for both species. The coexistence equilibrium* E_4^C *is a saddle. There is a separatrix that separates the domain* Ω^C *into two regions. We have bistability of* E_2^C *and* E_3^C *with stability (or instability) determined by the location of the initial conditions in two regions of* Ω^C*. If the initial conditions lie on the separatrix, then the solution tends to* E_4^C*.*

3.3 Equilibria of the Combined Two Species Competition and SI Mass Action Disease Model

We will denote equilibrial susceptible densities for species i by $S_{i,\infty}$ and similarly $I_{i,\infty}$ for the infected equilibrial densities of species i, for $i = 1, 2$. The equilibria for model (3.3)-(3.6) are

1. **The Trivial or Zero Equilibrium**

$$E_1 = (S^1_{1,\infty} = 0, S^1_{2,\infty} = 0, I^1_{1,\infty} = 0, I^1_{2,\infty} = 0). \tag{3.16}$$

2. **The Disease Free One-Host Equilibria**

   (a) Species 1 survives in an uninfected state and reaches carrying capacity. Species 2 dies out. The corresponding disease free one-host equilibrium is

$$E_2 = (S^2_{1,\infty} = K_{11}, S^2_{2,\infty} = 0, I^2_{1,\infty} = 0, I^2_{2,\infty} = 0). \tag{3.17}$$

   (b) Species 2 survives in an uninfected state and reaches carrying capacity. Species 1 dies out. The corresponding disease free one-host equilibrium is

$$E_3 = (S^3_{1,\infty} = 0, S^3_{2,\infty} = K_{22}, I^3_{1,\infty} = 0, I^3_{2,\infty} = 0). \tag{3.18}$$

3. **The Disease Free Coexistence Equilibrium**

$$E_4 = (S^4_{1,\infty}, S^4_{2,\infty}, I^4_{1,\infty} = 0, I^4_{2,\infty} = 0), \tag{3.19}$$

   with

$$S^4_{1,\infty} = \frac{K_{11}K_{12}}{K_{12} + K_{11}(\xi_1/\xi_2)}, \tag{3.20}$$

$$S^4_{2,\infty} = \frac{\xi_1}{\xi_2}\left(\frac{K_{11}K_{12}}{K_{12} + K_{11}(\xi_1/\xi_2)}\right) = \frac{\xi_1}{\xi_2}S^4_{1,\infty}, \tag{3.21}$$

   and the parameters ξ_1 and ξ_2 are as defined in (3.14), and (3.15), respectively. We note that $S^4_{i,\infty} = N^C_i, i = 1, 2$, where N^C_1, and N^C_2 are as defined in (3.12)-(3.13).

4. **The Infected One-Host Equilibria**

(a) Species 1 survives and species 2 dies out. The corresponding infected one-host equilibria are

$$E_{5,6} = (S_{1,\infty}^{5,6} = \frac{\Gamma_1}{\beta_{11}}, S_{2,\infty}^{5,6} = 0, I_{1,\infty}^{5,6} = \frac{I_{5,6}^*}{\beta_{11}}, I_{2,\infty}^{5,6} = 0), \tag{3.22}$$

where $I_{5,6}^*$ are roots of the quadratic polynomial

$$P_{56}(x) = x^2 + 2\Gamma_1 \left(1 + \frac{\mathcal{R}_0^1 \lambda_1}{2}\right) x + \Gamma_1^2 \left(1 - \mathcal{R}_0^1\right), \tag{3.23}$$

with the parameter λ_1 defined as

$$\lambda_1 := \frac{\alpha_1 - r_1}{r_1}, \tag{3.24}$$

and $\mathcal{R}_0^1$ is the basic reproduction number for species 1 alone, defined as

$$\mathcal{R}_0^1 := \frac{K_{11}\beta_{11}}{\Gamma_1}. \tag{3.25}$$

Solving for the roots, we have the infected component of species 1 in the one-host equilibria $E_{5,6}$ to be

$$I_{1,\infty}^{5,6} = S_{1,\infty}^{5,6} \left[-\left(1 + \frac{\mathcal{R}_0^1 \lambda_1}{2}\right) \pm \sqrt{\left(1 + \frac{\mathcal{R}_0^1 \lambda_1}{2}\right)^2 + (\mathcal{R}_0^1 - 1)} \right]. \tag{3.26}$$

In the next section we will show that only the root I_5^* is positive and the equilibrium E_5 is conditionally feasible, whereas the root I_6^* is always negative and thus the equilibrium E_6 is always infeasible.

We note that with appropriate definitions of parameters, the equilibrium $E_5 = E_3^D$, where E_3^D is as defined in (3.9).

(b) Species 2 survives in a partially infected state and species 1 dies out. The corresponding infected one-host equilibrium are

$$E_{7,8} = (S_{1,\infty}^{7,8} = 0, S_{2,\infty}^{7,8} = \frac{\Gamma_2}{\beta_{22}}, I_{1,\infty}^{7,8} = 0, I_{2,\infty}^{7,8} = \frac{I_{7,8}^*}{\beta_{22}}), \tag{3.27}$$

where $I_{7,8}^*$ are roots of the quadratic polynomial

$$P_{78}(x) = x^2 + 2\Gamma_2 \left(1 + \frac{\mathcal{R}_0^2 \lambda_2}{2}\right) x + \Gamma_2^2 \left(1 - \mathcal{R}_0^2\right). \tag{3.28}$$

with $\lambda_2 := \dfrac{\alpha_2 - r_2}{r_2}$, and $\mathcal{R}_0^2$ is the basic reproduction number for species 2 alone, defined as

$$\mathcal{R}_0^2 := \frac{K_{22}\beta_{22}}{\Gamma_2}. \tag{3.29}$$

Solving for the roots, we have the infected component of species 2 in the one-host equilibria $E_{7,8}$, to be

$$I_{2,\infty}^{7,8} = S_{2,\infty}^{7,8}\left[-\left(1+\frac{\mathcal{R}_0^2\lambda_2}{2}\right) \pm \sqrt{\left(1+\frac{\mathcal{R}_0^2\lambda_2}{2}\right)^2 + (\mathcal{R}_0^2-1)}\right], \tag{3.30}$$

As for the previous case we will see in the next section that only the root I_7^* is positive and the equilibrium E_7 is conditionally feasible, whereas the root I_8^* is always negative and thus the equilibrium E_8 is always infeasible.

Similar to the case of the infected one host equilibria in which species one survives, we note that with appropriate definitions of parameters, the equilibrium $E_7 = E_3^D$, where E_3^D is as defined in (3.9).

5. **Infected Coexistence Equilibria** As discussed in [9] the infected coexistence equilibria are intractable. It is possible to have multiple such equilibria present in the model with mass action disease transmission. We will consider a special case in section 3.5 in which the infected coexistence equilibrium is given by an analytical formula making analysis more amenable.

3.4 Local Stability Analysis of Equilibria for the Competition and Disease Model

3.4.1 The Trivial Equilibrium

First we show that the trivial equilibrium E_1 of model (3.3)-(3.6) is always unstable. The Jacobian of this model evaluated at E_1 is

$$\mathcal{J}(E_1) = \begin{bmatrix} r_1 & 0 & a_1 & 0 \\ 0 & r_2 & 0 & a_2 \\ 0 & 0 & -\Gamma_1 & 0 \\ 0 & 0 & 0 & -\Gamma_2 \end{bmatrix}.$$

The eigenvalues of $\mathcal{J}(E_1)$ are $r_i, -\Gamma_i$ for $i = 1, 2$. Thus, by assumption at least two of the eigenvalues are always positive, and hence the equilibrium E_1 is always unstable.

3.4.2 Disease Free Equilibria

In this section we address the stability of the disease free equilibria (DFE), E_2, E_3 and E_4. The stability of a DFE depends on the corresponding basic reproduction number, $\mathcal{R}_0$. The basic reproduction number (BRN) is defined as the average number of secondary infections that occur when an infected individual is introduced into a completely susceptible population. If $\mathcal{R}_0 > 1$, then the disease may emerge in one of the populations, whereas if $\mathcal{R}_0 < 1$, then the DFE is locally asymptotically stable [40]. In this case if the disease is introduced into the populations of competing species it will eventually die out leaving the population in a competition only state.

The Coexistence DFE :

As a DFE, the coexistence equilibrium is biologically feasible when

$$\frac{\xi_1}{\xi_2} > 0. \tag{3.31}$$

Theorem 3.1 *The basic reproduction number for model* (3.3)-(3.6) *with coexisting species is*

$$\mathcal{R}_0^C = \frac{\mathcal{R}_{11} + \mathcal{R}_{22}}{2} + \frac{\sqrt{(\mathcal{R}_{11} - \mathcal{R}_{22})^2 + 4\mathcal{R}_{12}\mathcal{R}_{21}}}{2}, \tag{3.32}$$

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

$$\mathcal{R}_{ij} = \frac{\beta_{ij}}{\Gamma_j} S_{i,\infty}^4, \tag{3.33}$$

with $S_{i,\infty}^4$ *as defined in* (3.20)-(3.21). *The condition* $\mathcal{R}_0^C < 1$ *leads to the inequality*

$$\mathcal{R}_{11} + \mathcal{R}_{22} + \mathcal{R}_{12}\mathcal{R}_{21} - \mathcal{R}_{11}\mathcal{R}_{22} < 1. \tag{3.34}$$

Proof. We will use the next generation matrix method [40] to determine the stability of the coexistence DFE, E_4. Let $X = (S_1, S_2, I_1, I_2)^T$. Then we can rewrite system (3.3)-(3.6) in the form

$$\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X) \tag{3.35}$$

where $\mathcal{F}(X)$ represents the vector function that includes the new infectious cases and $\mathcal{V}(X)$ contains all other dynamics due to death and recovery. We compute the Jacobian of $\mathcal{F}$ and $\mathcal{V}$ and evaluate these at the coexistence DFE, $E_4 = (S_{1,\infty}^4, S_{2,\infty}^4, 0, 0)$. Let F and V be the matrices defined by

$$F = \left[\frac{\partial \mathcal{F}_i}{\partial x_j}(E_4)\right]; \quad V = \left[\frac{\partial \mathcal{V}_i}{\partial x_j}(E_4)\right], \tag{3.36}$$

where $3 \leq i, j \leq 4$ and x_j is the jth component of the vector X defined in (3.35). Computing these matrices we have

$$F = \begin{bmatrix} \beta_{11} S_{1,\infty}^4 & \beta_{12} S_{1,\infty}^4 \\ \beta_{21} S_{2,\infty}^4 & \beta_{22} S_{2,\infty}^4 \end{bmatrix}, \tag{3.37}$$

and $V = \text{diag}(\Gamma_i)$. The BRN $\mathcal{R}_0^C$ for model (3.3)-(3.6) with coexisting species is given as

$$\mathcal{R}_0^C = \rho(FV^{-1}), \tag{3.38}$$

where $\rho(A)$ is the spectral radius of the matrix A. We have

$$FV^{-1} = \begin{bmatrix} \dfrac{\beta_{11} S_{1,\infty}^4}{\Gamma_1} & \dfrac{\beta_{12} S_{1,\infty}^4}{\Gamma_2} \\ \dfrac{\beta_{21} S_{2,\infty}^4}{\Gamma_1} & \dfrac{\beta_{22} S_{2,\infty}^4}{\Gamma_2} \end{bmatrix}. \tag{3.39}$$

Thus, using the definition (3.33) it is easily shown that the spectral radius of the matrix FV^{-1} is given by the formula (3.32).

Assuming $\mathcal{R}_0^C < 1$ in (3.32) we can now easily derive

$$\frac{\beta_{11}}{\Gamma_1}S_{1,\infty}^4 + \frac{\beta_{22}}{\Gamma_2}S_{2,\infty}^4 + \big(\frac{\beta_{12}\beta_{21}}{\Gamma_1\Gamma_2} - \frac{\beta_{11}\beta_{22}}{\Gamma_1\Gamma_2}\big)S_{1,\infty}^4 S_{2,\infty}^4 < 1, \tag{3.40}$$

which is equivalent to the inequality (3.34).

■

Remark 3.1 *The condition* (3.34) *is equivalent to the condition for stability of uninfected coexistence that is obtained in [9], given as*

$$(\beta_{11}S_{1,\infty}^4 - \Gamma_1)(\beta_{22}S_{2,\infty}^4 - \Gamma_2) - \beta_{12}\beta_{21}S_{1,\infty}^4 S_{2,\infty}^4 > 0. \tag{3.41}$$

Theorem 3.2 *The coexistence DFE, E_4 is feasible and stable if and only if the conditions $\xi_1 > 0$, $\xi_2 > 0$ and $\mathcal{R}_0^C < 1$ are satisfied.*

Proof. The Jacobian of the system (3.3)-(3.6) evaluated at the DFE $E_4 = (S_{1,\infty}^4, S_{2,\infty}^4, I_{1,\infty}^4 = 0, I_{2,\infty}^4 = 0)$ is the block triangular matrix

$$\mathcal{J}(E_4) = \begin{bmatrix} \mathcal{A}^* & \mathcal{B}^* \\ \mathbf{0} & F - V \end{bmatrix}, \tag{3.42}$$

where the matrix $\mathcal{A}^*$ is the Jacobian matrix of the system (3.10)-(3.11) evaluated at $E_4^C = (N_1^C, N_2^C) = (S_{1,\infty}^4, S_{2,\infty}^4)$ (see section 3.2), and the matrices F and V are as defined in (3.36). Since the Jacobian $\mathcal{J}(E_4)$ is block triangular, its eigenvalues are the eigenvalues of the matrices $\mathcal{A}^*$ and $F - V$.

From Lemma 3.2, $E_4^C = (N_1^C, N_2^C)$ is globally asymptotically stable if and only if $\xi_1 > 0$ and $\xi_2 > 0$. Thus, the eigenvalues of the matrix $\mathcal{A}^*$ are negative if and only if $\xi_1 > 0$ and $\xi_2 > 0$. We note that the conditions $\xi_1 > 0$ and $\xi_2 > 0$ also guarantee feasibility of the DFE E_4.

From the next generation approach, the eigenvalues of the matrix $F - V$ are negative if and only if $\mathcal{R}_0^C = \rho(FV^{-1}) < 1$ [40].

■

The Disease Free One-Host Equilibrium :

When $\xi_1/\xi_2 < 0$ the coexistence DFE is infeasible. We have the following two cases.

1. $\xi_1 > 0$ and $\xi_2 < 0$.

   In this case the one-host DFE $E_2 = (K_{11}, 0, 0, 0)$ is feasible and stable if in addition the condition

   $$\mathcal{R}_0^1 = \frac{K_{11}\beta_{11}}{\Gamma_1} < 1 \tag{3.43}$$

   is satisfied. The condition (3.43) implies that the basic reproduction number for species 1 alone is less than 1. This result follows from Lemma 3.2 for conditions on stability of E_2^C and from Lemma 3.1 for conditions on stability of E_2^D.

2. $\xi_1 < 0$ and $\xi_2 > 0$.

   In this case the one-host DFE $E_3 = (0, K_{22}, 0, 0)$ is feasible and stable if in addition the condition

$$\mathcal{R}_0^2 = \frac{K_{22}\beta_{22}}{\Gamma_2} < 1 \tag{3.44}$$

   is satisfied. As in case 1, this result follows from Lemma 3.2 for conditions on stability of E_3^C and from Lemma 3.1 for conditions on stability of E_2^D.

3.4.3 Infected One-Host Equilibria

We define the parameters,

$$\chi_{5,6} = -\left(1 + \frac{\mathcal{R}_0^1\lambda_1}{2}\right) \pm \sqrt{\left(1 + \frac{\mathcal{R}_0^1\lambda_1}{2}\right)^2 + (\mathcal{R}_0^1 - 1)}. \tag{3.45}$$

Using (3.45), we can rewrite equation (3.26) as

$$I_{1,\infty}^{5,6} = S_{1,\infty}^{5,6}\chi_{5,6}. \tag{3.46}$$

Lemma 3.3 *The infected one-host equilibrium E_5 is biologically feasible if and only if $\mathcal{R}_0^1 > 1$, whereas the equilibrium E_6 is always infeasible.*

Proof. Case 1: Let $\mathcal{R}_0^1 > 1$, then $\chi_5 > 0$, and $\chi_6 < 0$. Thus, E_5 is feasible and E_6 is biologically infeasible.
Case 2: Let $0 < \mathcal{R}_0^1 \leq 1$. In this case we note that the first term of $\chi_{5,6}$ in (3.45) can be rewritten as

$$-\left(1 + \frac{\mathcal{R}_0^1\lambda_1}{2}\right) = -\left(1 - \frac{\mathcal{R}_0^1}{2}\right) - \frac{\alpha_1\mathcal{R}_0^1}{2r_1} < 0, \tag{3.47}$$

as the rates α_1, r_1 are both positive. Thus, in this case as well $\chi_6 < 0$, and E_6 is biologically infeasible. If $\mathcal{R}_0^1 = 1$, then $\chi_5 = 0$, and the equilibrium E_5 reduces to the disease free one-host equilibrium E_2, whereas, if $0 < \mathcal{R}_0^1 < 1$ then $\chi_5 < 0$ and E_5 is also biologically infeasible.

■

Lemma 3.4 *Let $\mathcal{R}_0^1 > 1$. If $\alpha_1 > 0$, then $N_{1,\infty}^5 = S_{1,\infty}^5(1 + \chi_5) < K_{11}$. If $\alpha_1 = 0$ then $N_{1,\infty}^5 = K_{11}$.*

Proof. The condition $\mathcal{R}_0^1 > 1$ guarantees the feasibility of the equilibrium E_5. By assumption $\alpha_1 > 0$, and hence $\lambda_1 > -1$. We then have

$$\lambda_1 + 1 < \mathcal{R}_0^1(1 + \lambda_1) < \mathcal{R}_0^1\left(1 + \frac{\lambda_1}{2}\right)^2 - \mathcal{R}_0^1\frac{\lambda_1^2}{4} \tag{3.48}$$

$$\implies \left(1 + \frac{\mathcal{R}_0^1\lambda_1}{2}\right)^2 + (\mathcal{R}_0^1 - 1) < \left(\mathcal{R}_0^1 + \frac{\mathcal{R}_0^1\lambda_1}{2}\right)^2 \tag{3.49}$$

$$\implies \sqrt{\left(1+\frac{\mathcal{R}_0^1\lambda_1}{2}\right)^2+(\mathcal{R}_0^1-1)} < \mathcal{R}_0^1+\frac{\mathcal{R}_0^1\lambda_1}{2} \tag{3.50}$$

$$\implies \frac{\Gamma_1}{\beta_{11}}\left\{\left(\frac{-\mathcal{R}_0^1\lambda_1}{2}\right)+\sqrt{\left(1+\frac{\mathcal{R}_0^1\lambda_1}{2}\right)^2+(\mathcal{R}_0^1-1)}\right\} < K_{11}, \tag{3.51}$$

since from (3.25) $\mathcal{R}_0^1 = (K_{11}\beta_{11})/\Gamma_1$. From (3.22) and (3.26) we finally have

$$N_{1,\infty}^5 = S_{1,\infty}^5(1+\chi_5) < K_{11}. \tag{3.52}$$

If $\alpha_1 = 0$ (as in Section 3.5), then $\lambda_1 = -1$ and $N_{1,\infty}^5 = K_{11}$. Hence, we can see that the total population of the infected one-host equilibrium is less than (or equal to) the carrying capacity for species 1 in the case that the disease related mortality $\alpha_1 > 0$ ($\alpha_1 = 0$).

■

Theorem 3.3 *Assume $\alpha_1 > 0$. If $\mathcal{R}_0^1 > 1$ and $K_{21} < N_{1,\infty}^5$, then the infected one-host equilibrium for species 1, E_5, is feasible and stable.*

Proof. From Lemma 3.4 we know that the condition $\mathcal{R}_0^1 > 1$ guarantees the feasibility of E_5.

The Jacobian for the (species 1) infected one-host equilibrium E_5, with the order changed to $E_5 = (S_{1,\infty}^5, I_{1,\infty}^5, S_{2,\infty}^5, I_{2,\infty}^5)$ for convenience, is

$$\mathcal{J}(E_5) = \begin{bmatrix} P & R \\ 0 & Q \end{bmatrix}, \tag{3.53}$$

where

$$P = \begin{bmatrix} a_1\left(1-\frac{2N_{1,\infty}^5}{\theta_{11}}\right)-b_1-\beta_{11}I_{1,\infty}^5 & a_1\left(1-\frac{2N_{1,\infty}^5}{\theta_{11}}\right)-\beta_{11}S_{1,\infty}^5 \\ \beta_{11}I_{1,\infty}^5 & \beta_{11}S_{1,\infty}^5-\Gamma_1 \end{bmatrix},$$

$$R = \begin{bmatrix} \frac{-a_1N_{1,\infty}^5}{\theta_{12}} & \frac{-a_1N_{1,\infty}^5}{\theta_{12}}-\beta_{12}S_{1,\infty}^5 \\ 0 & \beta_{12}S_{1,\infty}^5 \end{bmatrix},$$

$$Q = \begin{bmatrix} a_2\left(1-\frac{N_{1,\infty}^5}{\theta_{21}}\right)-b_2-\beta_{21}I_{1,\infty}^5 & a_2\left(1-\frac{N_{1,\infty}^5}{\theta_{21}}\right) \\ \beta_{21}I_{1,\infty}^5 & -\Gamma_2 \end{bmatrix}.$$

Since $\mathcal{J}(E_5)$ is block triangular we need only consider the eigenvalues of P and Q. We notice that the upper left block matrix, P, is the same as the Jacobian for species 1 alone with the disease, i.e., the Jacobian of the system (3.7)-(3.8) evaluated at the equilibrium E_3^D (with the parameters and variables appropriately defined); see Section 3.1. From Lemma 3.1, the eigenvalues of P are negative if and only if $\mathcal{R}_0^1 > 1$.

We next consider the bottom right block matrix, Q, and use the trace determinant theorem to arrive at conditions for stability. With some algebraic manipulations the trace and determinant of the matrix Q can be written as

$$\mathrm{Tr}[Q] = r_2\left(1-\frac{N_{1,\infty}^5}{K_{21}}\right)-(\beta_{21}I_{1,\infty}^5+\Gamma_2), \tag{3.54}$$

and

$$\det[Q] = -r_2\left(1 - \frac{N^5_{1,\infty}}{K_{21}}\right)(\beta_{21}I^5_{1,\infty} + \Gamma_2) + \beta_{21}I^5_{1,\infty}\alpha_2. \tag{3.55}$$

If $K_{21} < N^5_{1,\infty}$, then $\left(1 - \frac{N^5_{1,\infty}}{K_{21}}\right) < 0$ and hence $\mathrm{Tr}(Q) < 0$ and $\det(Q) > 0$, as all the parameters are positive. Thus, if $\mathcal{R}^1_0 > 1$ and $K_{21} < N^5_{1,\infty}$ then the infected one host equilibrium E_5 is stable.

■

Remark 3.2 *The condition $K_{21} < N^5_{1,\infty}$ is not necessary for the stability of E_5. Necessary conditions for stability of E_5 are obtained by the application of the Trace-determinant theorem. From* (3.55), $\det(Q) > 0$ *gives us the condition*

$$r_2\left(1 - \frac{N^5_{1,\infty}}{K_{21}}\right)(\beta_{21}I^5_{1,\infty} + \Gamma_2) - \beta_{21}I^5_{1,\infty}\alpha_2 < 0. \tag{3.56}$$

By similar arguments we can prove

Theorem 3.4 *Assume $\alpha_2 > 0$. If $\mathcal{R}^2_0 > 1$ and $K_{12} < N^7_{2,\infty}$ then the infected one-host equilibrium E_7 is biologically feasible and stable. The equilibrium E_8 is always infeasible.*

Proof. The proof is similar to the proof of Theorem 3.3

■

3.5 Analysis of the Infected Coexistence Equilibrium of the Competition and Disease Model Under Additional Assumptions

In this section, we derive an analytical expression for the infected coexistence equilibrium of the two species model (3.3)-(3.6) under additional assumptions. Consequently we perform a full stability analysis. This allows us, under certain assumptions, to prove the conjecture of [26] and [5] about the behavior of the infected coexistence equilibrium of population models that are combined with mass action disease models. Based on numerical simulations, the authors in [26, 5, 18, 9] conjecture that if all other equilibria are unstable then the infected coexistence equilibrium is stable and, conversely, that if any of the other equilibria are stable then the infected coexistence equilibrium is unstable.

Here we make the following additional assumptions on the model (3.3)-(3.6) described in Section 3.

(A1) $\alpha_i = 0$, so that there is no increased death rate as a result of the disease. In addition, we will assume

(A2) $a = a_1 = a_2$, $b = b_1 = b_2$, $\theta = \theta_{11} = \theta_{22}$, and $\beta = \beta_{ij}$ all i, j. As before, let $r := a - b$ be the intrinsic growth rate for both the species. Also, $K = K_{11} = K_{22} = \frac{r\theta}{a}$, so the carrying capacity is the same for both species.

In order to retain a difference between the species we will also require that

(A3) $\theta_{12} \neq \theta_{21}$.

As before, we define $K_{ij} := \frac{r\theta_{ij}}{a}$. These simplifications are not only didactic but result in a model that can represent actual ecological systems. For example, if two species are limited by different resources then they may have very similar intra-specific competition but quite different inter-specific competition while still being susceptible to a generalist pathogen or parasite [8].

Now the possible equilibria, in the form $E_i = (S_{1,\infty}, S_{2,\infty}, I_{1,\infty}, I_{2,\infty})$, for the competing two species SI model with mass action disease transmission, (3.3)-(3.6) under the additional assumptions (A1), (A2) and (A3) are:

1. **Trivial equilibrium**:
   $E_1 = (0, 0, 0, 0)$

2. **Disease Free One Host Equilibria**:
   $E_2 = (K, 0, 0, 0)$ and $E_3 = (0, K, 0, 0)$

3. **Disease Free Coexistence Equilibria**:
   $E_4 = (S^4_{1,\infty}, S^4_{2,\infty}, 0, 0)$ with

$$S^4_{1,\infty} = \frac{KK_{12}}{K_{12} + K(\xi_1/\xi_2)}, \tag{3.57}$$

$$S^4_{2,\infty} = \frac{\xi_1}{\xi_2}\left(\frac{KK_{12}}{K_{12} + K(\xi_1/\xi_2)}\right) = \frac{\xi_1}{\xi_2}S^4_{1,\infty}, \tag{3.58}$$

   where the parameters ξ_1 and ξ_2 defined in (3.14)-(3.15) reduce to

$$\xi_1 = (\frac{1}{K} - \frac{1}{K_{21}}), \tag{3.59}$$

$$\xi_2 = (\frac{1}{K} - \frac{1}{K_{12}}). \tag{3.60}$$

4. **Infected One Host Equilibria**:
   $E_5 = (S^5_{1,\infty}, 0, I^5_{1,\infty}, 0)$ and $E_6 = (0, S^6_{2,\infty}, 0, I^6_{2,\infty})$, where for $i = 1, 2$

$$S^5_{1,\infty} = S^6_{2,\infty} = \frac{b}{\beta}, \tag{3.61}$$

$$I^5_{1,\infty} = \frac{r\theta}{a} - \frac{b}{\beta} = (\mathcal{R}_0 - 1)S^5_{1,\infty}, \tag{3.62}$$

$$I^6_{2,\infty} = \frac{r\theta}{a} - \frac{b}{\beta} = (\mathcal{R}_0 - 1)S^6_{1,\infty}. \tag{3.63}$$

   and $\mathcal{R}_0 = \mathcal{R}^1_0 = \mathcal{R}^2_0 = \dfrac{K\beta}{b}$ is the same for both species.

5. **Infected Coexistence Equilibria**:

$E_7 = (S^7_{1,\infty}, S^7_{2,\infty}, I^7_{1,\infty}, I^7_{2,\infty})$, with

$$S^7_{1,\infty} = \frac{b}{\beta\left(1+\frac{\xi_1}{\xi_2}\right)}, \tag{3.64}$$

$$S^7_{2,\infty} = \frac{\xi_1}{\xi_2} S^7_{1,\infty}, \tag{3.65}$$

$$I^7_{1,\infty} = \frac{\beta\left(1+\frac{\xi_1}{\xi_2}\right) - b\left(\frac{1}{K}+\frac{\xi_1}{K_{12}\xi_2}\right)}{\beta\left(1+\frac{\xi_1}{\xi_2}\right)\left(\frac{1}{K}+\frac{\xi_1}{K_{12}\xi_2}\right)}, \tag{3.66}$$

$$I^7_{2,\infty} = \frac{\xi_1}{\xi_2} I^7_{1,\infty}. \tag{3.67}$$

Lemma 3.5 *The equilibrium value $I^7_{i,\infty}$ can be rewritten as*

$$I^7_{i,\infty} = (R_0^C - 1) S^7_{i,\infty}, \tag{3.68}$$

for $i = 1, 2$. The basic reproduction number for the two species model under the additional assumptions (A1), (A2) and (A3), can be derived from equation (3.32) *to be*

$$\mathcal{R}_0^C = \frac{\beta}{b}\left(S^4_{1,\infty} + S^4_{2,\infty}\right). \tag{3.69}$$

Proof. From equations (3.64), (3.66) and (3.57), we have

$$I^7_{1,\infty} = S^7_{1,\infty}\left\{\frac{\beta}{b}\left(1+\frac{\xi_1}{\xi_2}\right) S^4_{1,\infty} - 1\right\} \tag{3.70}$$

$$= S^7_{1,\infty}\left\{\frac{\beta}{b}\left(S^4_{1,\infty} + S^4_{2,\infty}\right) - 1\right\} \tag{3.71}$$

$$= S^7_{1,\infty}(\mathcal{R}_0^C - 1). \tag{3.72}$$

Similarly, we can show that $I^7_{2,\infty} = (R_0^C - 1) S^7_{2,\infty}$.

■

Lemma 3.6 *The total population size $N^7_{i,\infty} = S^4_{i,\infty}$, for $i = 1, 2$.*

Proof. From Lemma 3.5 and equations (3.69), (3.64), and (3.58), we have

$$N^7_{1,\infty} = S^7_{1,\infty} + I^7_{1,\infty} = S^7_{1,\infty}\mathcal{R}_0^C \tag{3.73}$$

$$= \frac{b}{\beta(1+\frac{\xi_1}{\xi_2})}\frac{\beta}{b} S^4_{1,\infty}\left(1+\frac{\xi_1}{\xi_2}\right) = S^4_{1,\infty} \tag{3.74}$$

Similarly, we can show that $N^7_{2,\infty} = S^4_{2,\infty}$.

■

3.5.1 Local Stability Analysis

We will now use the Jacobian of our simplified model to establish stability conditions for all the equilibria. Specifically, we will show that when there is no additional death due to disease and no recovery then the conjecture of [26] and of [5] holds. The Jacobian for this system computed at an equilibrium $E_e = (S^e_{1,\infty}, S^e_{2,\infty}, I^e_{1,\infty}, I^e_{2,\infty})$ is

$$\mathcal{J}(E_e) = \begin{bmatrix} \mathcal{A}(E_e) & \mathcal{B}(E_e) \\ \mathcal{C}(E_e) & \mathcal{D}(E_e) \end{bmatrix}, \tag{3.75}$$

where, the 2×2 matrices $\mathcal{A}$, $\mathcal{B}$, $\mathcal{C}$, and $\mathcal{D}$ evaluated at an equilibrium E_e are defined as

$$\mathcal{A}(E_e) = \begin{bmatrix} A(E_e) - b - \mathcal{I}(E_e) & A_{12}(E_e) \\ B_{21}(E_e) & B(E_e) - b - \mathcal{I}(E_e) \end{bmatrix}, \tag{3.76}$$

$$\mathcal{B}(E_e) = \begin{bmatrix} A(E_e) - \beta S^e_{1,\infty} & A_{12}(E_e) - \beta S^e_{1,\infty} \\ B_{21}(E_e) - \beta S^e_{2,\infty} & B(E_e) - \beta S^e_{2,\infty} \end{bmatrix}, \tag{3.77}$$

$$\mathcal{C}(E_e) = \begin{bmatrix} \mathcal{I}(E_e) & 0 \\ 0 & \mathcal{I}(E_e) \end{bmatrix}, \tag{3.78}$$

and

$$\mathcal{D}(E_e) = \begin{bmatrix} \beta S^e_{1,\infty} - b & \beta S^e_{1,\infty} \\ \beta S^e_{2,\infty} & \beta S^e_{2,\infty} - b \end{bmatrix}. \tag{3.79}$$

with the definitions

$$A(E_e) := \frac{-aN^e_{1,\infty}}{\theta} + g_1(N^e_{1,\infty}, N^e_{2,\infty}), \tag{3.80}$$

$$A_{12}(E_e) := \frac{-aN^e_{1,\infty}}{\theta_{12}}, \tag{3.81}$$

$$B(E_e) := \frac{-aN^e_{2,\infty}}{\theta} + g_2(N^e_{1,\infty}, N^e_{2,\infty}), \tag{3.82}$$

$$B_{21}(E_e) := \frac{-aN^e_{2,\infty}}{\theta_{21}}. \tag{3.83}$$

For $i = 1, 2$, we have $N_{i,\infty} = S_{i,\infty} + I_{i,\infty}$. From (3.2a) and (3.2b) we have the disease incidence function,

$$\mathcal{I}(E_e) = \beta(I^e_{1,\infty} + I^e_{2,\infty}), \tag{3.84}$$

($\mathcal{I}_1 = \mathcal{I}_2$), and for $i = 1, 2$, the birth functions g_i as defined in (2.2a) and (2.2b) (with $\theta = \theta_{11} = \theta_{22}$) evaluated at E_e are given as

$$g_1(E_e) = a\left(1 - \frac{N^e_{1,\infty}}{\theta} - \frac{N^e_{2,\infty}}{\theta_{12}}\right), \tag{3.85a}$$

$$g_2(E_e) = a\left(1 - \frac{N^e_{2,\infty}}{\theta} - \frac{N^e_{1,\infty}}{\theta_{21}}\right). \tag{3.85b}$$

The Trivial and Disease Free One-Host Equilibria :

As before, E_1 is always unstable for positive parameters. The disease free one-host equilibria $E_2 = (K, 0, 0, 0)$ is stable if conditions

(C1) $\mathcal{R}_0 = \frac{K\beta}{b} < 1$, and

(C2) $\xi_1 < 0$,

hold. In the symmetric case, $E_3 = (0, 0, K, 0)$ is stable if condition (C1) holds and if the condition

(C3) $\xi_2 < 0$,

holds.

The Disease Free Coexistence Equilibrium :

The disease free coexistence equilibrium $E_4 = (S^0_{1,\infty}, S^0_{2,\infty}, 0, 0)$ is feasible when $\xi_1/\xi_2 > 0$.

The Jacobian (3.75) evaluated at E_4 is of the form

$$\mathcal{J}(E_4) = \begin{bmatrix} \mathcal{A}(E_4) & \mathcal{B}(E_4) \\ 0 & \mathcal{D}(E_4) \end{bmatrix}, \tag{3.86}$$

where the 2×2 matrices $\mathcal{A}$, $\mathcal{B}$, and $\mathcal{D}$ defined in (3.76), (3.77), and (3.79), respectively are all evaluated at the equilibrium E_4.

Lemma 3.7 *Assume that $\xi_1/\xi_2 > 0$, so that the disease free coexistence equilibrium E_4 is feasible. In this case*

$$\det[\mathcal{A}](E_4) = r^2 S^4_{1,\infty} \xi_1, \tag{3.87}$$

$$\mathrm{Tr}[\mathcal{A}](E_4) = -\frac{r S^4_{1,\infty}}{K}\left(1 + \frac{\xi_1}{\xi_2}\right). \tag{3.88}$$

Thus, $\mathrm{Tr}[\mathcal{A}](E_4)$ *is always negative, whereas* $\det[\mathcal{A}](E_4) > 0$ *if and only if* $\xi_1 > 0$ *and (by assumption)* $\xi_2 > 0$.

Proof. We prove this lemma in the appendix, as the algebra is tedious but straightforward. ■

Since the Jacobian $\mathcal{J}(E_4)$ is block upper triangular, its eigenvalues are the same as those of matrices $\mathcal{A}(E_4)$ and $\mathcal{D}(E_4)$. The matrix $\mathcal{A}(E_4)$ is the Jacobian of the two species model with pure competition, (3.10)-(3.11) evaluated at (N_1^C, N_2^C) (see Section 3.2) under the assumptions (A2) and (A3). From Lemma 3.7, the eigenvalues of $\mathcal{A}(E_4)$ are negative if and only if the conditions

(C4) $\xi_1 > 0$, and

(C5) $\xi_2 > 0$,

hold. The matrix $\mathcal{D}(E_4)$ on the other hand is related to the disease parameters and its eigenvalues are $\lambda_1 = -b$ and $\lambda_2 = \beta(S^4_{1,\infty} + S^4_{2,\infty}) - b$. The eigenvalue λ_1 is always negative and λ_2 is negative under the condition

$$\text{(C6)}\ \ \mathcal{R}_0^C = \frac{\beta(S^4_{1,\infty} + S^4_{2,\infty})}{b} < 1$$

holds. So, the DFE E_4 is feasible and stable if and only if the conditions (C4), (C5) and (C6) hold.

We note that this result is a special case of Theorem 3.2 derived from Lemma 3.2 in Section 3.2. The condition (C6) is the analogue of the inequality (3.34) for this special case.

The Infected One-Host Equilibrium The Jacobian, (3.75), evaluated at E_5 is

$$\mathcal{J}(E_5) = \begin{bmatrix} a\left(1-\frac{2K}{\theta}\right) - \beta K & \frac{-aK}{\theta_{12}} & a\left(1-\frac{2K}{\theta}\right) - b & \frac{-aK}{\theta_{12}} - b \\ 0 & a\left(1-\frac{K}{\theta_{21}}\right) - \beta K & 0 & a\left(1-\frac{K}{\theta_{21}}\right) \\ \beta K - b & 0 & 0 & b \\ 0 & \beta K - b & 0 & -b \end{bmatrix}.$$

The eigenvalues of this matrix are $\lambda_1 = -K\beta$, $\lambda_2 = b(1-\mathcal{R}_0)$, $\lambda_3 = -r$, and $\lambda_4 = rK\xi_1$. We can see that λ_1 and λ_3 are always negative. Thus, the stability (and feasibility) conditions for E_5 are

$$\text{(C7)}\ \ \mathcal{R}_0 = \frac{K\beta}{b} > 1,$$

which guarantees that $\lambda_2 < 0$ and condition (C2) which guarantees that $\lambda_4 < 0$. For the symmetric case, E_6 is feasible and stable if conditions (C7) and (C3) hold.

The Infected Coexistence Equilibrium :

Lastly, we consider the infected coexistence equilibrium E_7. The characteristic polynomial of $\mathcal{J}(E_7)$ is given as

$$P_7(x) = (x+\eta)(x+\epsilon)(x^2 + \delta_1 x + \delta_2), \tag{3.89}$$

where

$$\eta = b + \beta(I^7_{1,\infty} + I^7_{2,\infty}), \tag{3.90}$$
$$\epsilon = b + \beta(I^7_{1,\infty} + I^7_{2,\infty}) - \beta(S^7_{1,\infty} + S^7_{2,\infty}), \tag{3.91}$$
$$\delta_1 = -(A(E_7) + B(E_7)) + 2b, \tag{3.92}$$
$$\delta_2 = -A_{12}(E_7)B_{21}(E_7) + A(E_7)B(E_7) - b(A(E_7) + B(E_7)) + b^2, \tag{3.93}$$

where for $i = 1, 2$, $N^7_{i,\infty} = S^7_{i,\infty} + I^7_{i,\infty}$. The terms A, B, A_{12} and B_{21} as defined in (3.80)-(3.83) are all evaluated at the infected coexistence equilibria E_7. Then, the eigenvalues of the Jacobian $\mathcal{J}(E_7)$ are

$$\lambda^7_1 = -\eta = -b - \beta(I^7_{1,\infty} + I^7_{2,\infty}), \tag{3.94}$$

$$\lambda^7_2 = -\epsilon = -b - \beta(I^7_{1,\infty} + I^7_{2,\infty}) + \beta(S^7_{1,\infty} + S^7_{2,\infty}), \tag{3.95}$$

$$\lambda^7_{3,4} = \frac{1}{2}\left(-\delta_1 \pm \sqrt{\delta_1^2 - 4\delta_2}\right). \tag{3.96}$$

Lemma 3.8 *The condition that $\lambda^7_2 < 0$ is equivalent to $\mathcal{R}^C_0 > 1$*

Proof. From (3.65), (3.67) and Lemma 3.5 we have

$$\begin{aligned}
&\lambda^7_2 < 0 \\
&\iff -b - \beta(I^7_{1,\infty} + I^7_{2,\infty}) + \beta(S^7_{1,\infty} + S^7_{2,\infty}) < 0 \\
&\iff S^7_{1,\infty} - I^7_{1,\infty} + S^7_{2,\infty} - I^7_{2,\infty} < \frac{b}{\beta} \\
&\iff (1 + \frac{\xi_1}{\xi_2})(S^7_{1,\infty} - I^7_{1,\infty}) < \frac{b}{\beta} \\
&\iff (2 - \mathcal{R}^C_0)(1 + \frac{\xi_1}{\xi_2})\frac{b}{\beta(1 + \frac{\xi_1}{\xi_2})} < \frac{b}{\beta} \\
&\iff \mathcal{R}^C_0 > 1.
\end{aligned}$$

Thus, $\mathcal{R}^C_0 > 1$ is both a feasibility and stability condition for the infected coexistence equilibrium E_7.

■

Lemma 3.9 *The eigenvalues λ^7_3 and λ^7_4 are roots of the polynomial equation*

$$x^2 - \mathrm{Tr}[\mathcal{A}](E_4)x + \det[\mathcal{A}](E_4) = 0. \tag{3.97}$$

Proof. From Lemma 3.6, we have $N^7_{i,\infty} = S^4_{i,\infty} = N^4_{i,\infty}$ (as $I^4_{i,\infty} = 0$ for the disease free equilibrium E_4), for $i = 1, 2$. Thus, from (3.92) and (3.93) and the definitions of the functions A, B, A_{12}, and B_{21} in (3.80)-(3.83), we have

$$\delta_1 = -(A(E_4) + B(E_4)) + 2b, \tag{3.98}$$

$$\delta_2 = -A_{12}(E_4)B_{21}(E_4) + A(E_4)B(E_4) - b(A(E_4) + B(E_4)) + b^2. \tag{3.99}$$

From the definition of the matrix $\mathcal{A}$ in (3.76), we observe that

$$\delta_1 = -\mathrm{Tr}[\mathcal{A}](E_4) \tag{3.100}$$

$$\delta_2 = \det[\mathcal{A}](E_4) \tag{3.101}$$

From equation (3.96), it is clear that the eigenvalues λ^7_3 and λ^7_4 are roots of the polynomial equation (3.97).

■

Theorem 3.5 *Assume that $\xi_1/\xi_2 > 0$ so that the infected coexistence equilibrium E_7 is feasible. Then E_7 is stable if and only if $\xi_1 > 0, \xi_2 > 0$, and $\mathcal{R}_0^C > 1$. In this case all the other equilibria, i.e., E_1, E_2, E_3, E_4, E_5 and E_6 are either infeasible and/or unstable.*

Proof. It is easy to see that λ_1^7 given in (3.94) is negative for all $I_{1,e} + I_{2,e} \geq 0$. Thus, since the infected coexistence equilibrium E_7 is feasible by assumption ($\xi_1/\xi_2 > 0$) we have $\lambda_1 < 0$. As a result of Lemma 3.8, the first condition for stability of E_7 is

(C8) $\mathcal{R}_0^C > 1$

Since $I_{1,\infty}^7 = (\mathcal{R}_0^C - 1)S_{1,\infty}^7$, the condition (C8) is also a feasibility condition for E_7.

From Lemma 3.7, Lemma 3.9, and the Trace-Determinant theorem [1], we see that the eigenvalues λ_3 and λ_4 are negative if and only if the conditions (C4) and (C5) are satisfied.

When conditions (C4), (C5) and (C8) are satisfied, all the other equilibria, i.e., E_1-E_6 are either infeasible or unstable based on the linear stability analysis presented above for each of these equilibria.

■

3.6 Bifurcations

Considering the parameters ξ_1, and ξ_2, defined in (3.59) and (3.60), respectively, as bifurcation parameters we can make the following observations.

Remark 3.3 *If $\xi_1 = 0$ and/or $\xi_2 = 0$ then $\mathcal{R}_0^C = \frac{K\beta}{b} = \mathcal{R}_0$.*

Remark 3.4 *If $\xi_1 = 0$ and $\xi_2 > 0$ then $E_4 = E_2$ and $E_7 = E_5$. Similarly, if $\xi_1 > 0$ and $\xi_2 = 0$ then $E_4 = E_3$ and $E_7 = E_6$.*

Remark 3.5 *If both $\xi_1 = 0$ and $\xi_2 = 0$ then the sum of the state variables behaves as one species with logistic growth. In this case, the equilibrium E_4 is any solution $(S_{1,\infty}, S_{2,\infty}, 0, 0)$ on the line $S_{1,\infty} + S_{2,\infty} = K$. Similarly, E_7 becomes any solution $(S_{1,\infty}, S_{2,\infty}, I_{1,\infty}, I_{2,\infty})$ on the plane $S_{1,\infty} + S_{2,\infty} = \frac{b}{\beta}$, $I_{1,\infty} + I_{2,\infty} = \frac{b}{\beta}(\mathcal{R}_0 - 1)$. Notice that in both cases, since there is no additional death due to disease, $N_{1,\infty} + N_{2,\infty} = K$.*

Corollary 3.1 *Assume $\xi_1 = 0$ and $\xi_2 > 0$. Then,*

1. *If $\mathcal{R}_0^C = \mathcal{R}_0 < 1$, the equilibrium $E_4 = E_2$ exists in a neutral state.*
2. *If $\mathcal{R}_0^C = \mathcal{R}_0 > 1$, the equilibrium $E_7 = E_5$ exists in a neutral state.*

Proof. In the first case, the eigenvalues for E_4 are $\lambda_1 = -r$, $\lambda_{2,3} = 0$, and $\lambda_4 = \beta K - b = b(\mathcal{R}_0 - 1)$. We can see that if $\mathcal{R}_0^C = \mathcal{R}_0 < 1$ then $\lambda_4 < 0$ and E_4 is neutral. In fact, E_4 exchanges stability with E_2 as it moves through the half plane $\xi_1 = 0$, $\xi_2 > 0$ when $\mathcal{R}_0 < 1$.

In the second case, the eigenvalues for E_7 are $\lambda_1 = -r$, $\lambda_2 = 0$, $\lambda_3 = -K\beta$, and $\lambda_4 = b(1 - \mathcal{R}_0)$. We can see that if $\mathcal{R}_0^C = \mathcal{R}_0 > 1$ then $\lambda_3 < 0$, hence E_7 is neutral. In fact, E_7 exchanges stability with E_5 as it moves through the half plane $\xi_1 = 0$, $\xi_2 > 0$ when $\mathcal{R}_0 > 1$.

■

Corollary 3.2 *Assume $\xi_1 > 0$ and $\xi_2 = 0$. Then,*

1. *If $\mathcal{R}_0^C = \mathcal{R}_0 < 1$, the equilibrium $E_4 = E_3$ exists in a neutral state.*
2. *If $\mathcal{R}_0^C = \mathcal{R}_0 > 1$, the equilibrium $E_7 = E_6$ exists in a neutral state.*

Proof. The proof omitted as it is similar to the proof of Corollary 1.

■

Corollary 3.3 *Assume $\xi_1 = 0$ and $\xi_2 = 0$. Then,*

1. *If $\mathcal{R}_0^C = \mathcal{R}_0 < 1$, the equilibrium E_4 exists in a neutral state.*
2. *If $\mathcal{R}_0^C = \mathcal{R}_0 > 1$, the equilibrium E_7 exists in a neutral state.*

Proof. In the first case the eigenvalues of E_4 are $\lambda_1 = -r$, $\lambda_2 = 0$, $\lambda_3 = -b$, and $\lambda_4 = b(\mathcal{R}_0 - 1)$. We can see if $\mathcal{R}_0^C = \mathcal{R}_0 < 1$ then E_4 is neutral. In fact, as E_4 moves along the line $\xi_1 = \xi_2$ from $\xi_1, \xi_2 > 0$ through $\xi_1, \xi_2 = 0$ into $\xi_1, \xi_2 < 0$, it progresses from stable to neutral to stable.

In the second case the eigenvalues of E_7 are $\lambda_1 = -r$, $\lambda_2 = 0$, $\lambda_3 = -\beta K$, and $\lambda_4 = b(1 - \mathcal{R}_0)$. We can see if $\mathcal{R}_0^C = \mathcal{R}_0 > 1$ then E_7 is neutral. Similarly to E_4, as E_7 moves along the line $\xi_1 = \xi_2$ through $\xi_1, \xi_2 = 0$ it also progresses from stable to neutral to stable.

■

4 Models with Frequency Incidence Disease Transmission

In this section we consider two species models in which the transmission dynamics follows the frequency incidence approach. In this approach the intra-species and inter-species transmission rates in (2.3a) - (2.3b) are defined as

$$\Lambda_{ij}(N_j) = \beta_{ij}, \tag{4.1}$$

where for $i = 1, 2$, $\beta_{ij} > 0$ are constant terms. From (2.3a), (2.3b) and (4.1) we can write the disease incidence functions as

$$\mathcal{I}_1(I_1, I_2) = \left(\beta_{11}\frac{I_1}{N_1} + \beta_{12}\frac{I_2}{N_2}\right) S_1, \tag{4.2}$$

$$\mathcal{I}_2(I_1, I_2) = \left(\beta_{22}\frac{I_2}{N_2} + \beta_{21}\frac{I_1}{N_1}\right) S_2. \tag{4.3}$$

In [31, 34, 21, 24] the authors consider two species models with frequency incidence disease transmission. In the model considered in [31, 24], inter-specific competition is not included, but intra-specific competition is so that each species reaches a carrying capacity at equilibrium. Thus, the birth functions g_1 and g_2 model logistic growth. In [34] and [21], both inter- and intra-specific competition between the species is modeled, so g_1 and

g_2 are Lotka-Volterra competition terms. In [21] Lotka-Volterra competition dynamics are also included in the death term so that both the death rates as well as the birth rates are density-dependent. However, death due to disease is not accounted for. In [34], the authors fully analyze a model similar to the one that we consider in this section. As opposed to our model, they assume density-dependent death rates and density-independent birth rates.

In this section, we prove the existence, uniqueness and global stability of the infected coexistence equilibrium under the assumption that coexistence of the species is feasible. Thus, our work complements and extends the work done in [24, 34]. As would be expected, the stability of the coexistence equilibrium depends on the basic reproduction number (BRN) being greater than one.

As done in the mass action case, we model the birth functions for the two species by equations (2.2a) and (2.2b). With these assumptions, the two-species competition model with frequency incidence disease transmission is:

$$\frac{dS_1}{dt} = a_1\left(1 - \frac{N_1}{\theta_{11}} - \frac{N_2}{\theta_{12}}\right)N_1 - b_1S_1 - \left(\beta_{11}\frac{I_1}{N_1} + \beta_{12}\frac{I_2}{N_2}\right)S_1, \tag{4.4a}$$

$$\frac{dS_2}{dt} = a_2\left(1 - \frac{N_2}{\theta_{22}} - \frac{N_1}{\theta_{21}}\right)N_2 - b_2S_2 - \left(\beta_{22}\frac{I_2}{N_2} + \beta_{21}\frac{I_1}{N_1}\right)S_2, \tag{4.4b}$$

$$\frac{dI_1}{dt} = \left(\beta_{11}\frac{I_1}{N_1} + \beta_{12}\frac{I_2}{N_2}\right)S_1 - \Gamma_1 I_1, \tag{4.4c}$$

$$\frac{dI_2}{dt} = \left(\beta_{22}\frac{I_2}{N_2} + \beta_{21}\frac{I_1}{N_1}\right)S_2 - \Gamma_2 I_2. \tag{4.4d}$$

The model (4.4a)-(4.4d) makes ecological sense and is mathematically well-posed in the domain $\mathcal{D}^1 = \{(S_1, S_2, I_1, I_2) \in \mathbb{R}^4 | S_1, S_2, I_1, I_2 \geq 0, 0 \leq N_i \leq K_{ii}\}$. The total population size $N_i = S_i + I_i$ of species i satisfy the differential equations,

$$\frac{dN_1}{dt} = a_1\left(1 - \frac{N_1}{\theta_{11}} - \frac{N_2}{\theta_{12}}\right)N_1 - b_1N_1 - \alpha_1 I_1, \tag{4.5a}$$

$$\frac{dN_2}{dt} = a_2\left(1 - \frac{N_2}{\theta_{22}} - \frac{N_1}{\theta_{21}}\right)N_2 - b_2N_2 - \alpha_2 I_2, \tag{4.5b}$$

4.1 Single Species Logistic growth Model with Frequency incidence Disease Transmission

We recall the analysis of the SI disease model for one species with frequency incidence,

$$\frac{dS}{dt} = a\left(1 - \frac{N}{\theta}\right)N - bS - \beta\frac{I}{N}S, \tag{4.6}$$

$$\frac{dI}{dt} = \beta\frac{I}{N}S - \Gamma I, \tag{4.7}$$

where the parameters retain the same meaning as in Section 2. In particular $\Gamma = \alpha + b$. The model (4.6)-(4.7) is well-posed on the domain $\Omega^F = \{(S, I)^T | S, I \geq 0, 0 \leq N \leq K\}$. The equilibria for model (4.6)-(4.7) are $E_1^F = (0, 0)$, $E_2^F = (K, 0)$, and

$$E_3^F = \left(S_3^F, (\mathcal{R}_0 - 1)S\right), \tag{4.8}$$

where $S_3^F = \frac{\Gamma}{\beta}\theta\left[1+\frac{\alpha-\beta}{a}\frac{\Gamma}{\beta}\right]$, and $\mathcal{R}_0 := \frac{\beta}{\Gamma}$ is the basic reproduction number for the model. Thus, $\mathcal{R}_0 > 1$ is a feasibility condition for the equilibrium E_3^F.

We have the following lemma [34].

Lemma 4.1 *For the model* (4.6)-(4.7)*, the trivial equilibrium E_1^F is always unstable. If $\mathcal{R}_0 < 1$ then the disease-free equilibrium E_2^F is asymptotically stable in the domain Ω^D. If $\mathcal{R}_0 > 1$ then the infected equilibrium E_3^F is asymptotically stable in the domain Ω^F.*

4.2 Analysis of Equilibria for Two Species Competition with Frequency Incidence Disease Transmission

In this section, we analyze the disease free equilibrium and the infected coexistence for the model (4.4a)-(4.5b).

4.2.1 Disease Free Equilibrium

We have the following result for the DFE of model (4.4a)-(4.5b)

Theorem 4.1 *The basic reproduction number for model* (4.4a)-(4.5b) *with coexisting species is*

$$\mathcal{R}_0^C = \frac{\mathcal{R}_{11}+\mathcal{R}_{22}}{2} + \frac{\sqrt{(\mathcal{R}_{11}-\mathcal{R}_{22})^2+4\mathcal{R}_{12}\mathcal{R}_{21}}}{2}, \tag{4.9}$$

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

$$\mathcal{R}_{ij} = \frac{\beta_{ij}S_{i,\infty}^4}{\Gamma_j S_{j,\infty}^4}, \tag{4.10}$$

and where $S_{j,\infty}^4$, for $j = 1, 2$ are the susceptible equilibrial densities of the disease free equilibrium E_4 as defined in (3.20)-(3.21)*. The condition $\mathcal{R}_0^C < 1$ leads to the inequality*

$$\frac{\beta_{11}}{\Gamma_1}+\frac{\beta_{22}}{\Gamma_2}+\left(\frac{\beta_{12}\beta_{21}}{\Gamma_1\Gamma_2}-\frac{\beta_{11}\beta_{22}}{\Gamma_1\Gamma_2}\right) < 1. \tag{4.11}$$

Proof. The proof is omitted as it is similar to the proof for Theorem 1.

■

Remark 4.1 *Unlike the mass action case, $\mathcal{R}_{11} = \mathcal{R}_0^1 := \frac{\beta_{11}}{\Gamma_1}$ and $\mathcal{R}_{22} = \mathcal{R}_0^2 := \frac{\beta_{22}}{\Gamma_2}$, so that* (4.11) *can be rewritten in terms of the basic reproduction numbers of each species alone combined with inter-specific terms as*

$$\mathcal{R}_0^1+\mathcal{R}_0^2+\left(\frac{\beta_{12}\beta_{21}}{\Gamma_1\Gamma_2}-\mathcal{R}_0^1\mathcal{R}_0^2\right) < 1. \tag{4.12}$$

In the next section, we first rewrite model (4.4a)-(4.5b) using the proportion of infected individuals, and then analyze the stability of the infected coexistence equilibrium of this modified model.

4.2.2 Infected Coexistence Equilibrium

In this section we examine the infected coexistence (endemic) equilibrium of the system (4.4a)-(4.5b), assuming that the feasibility conditions are met and both species are present. Although the actual value of this equilibrium is algebraically intractable, we use methods similar to [31] to analyze the stability of the endemic equilibrium.

Assuming that $N_1, N_2 > 0$, we will express the model (4.4c)-(4.5b) in terms of the proportion of infected individuals. Let $i_1 = \frac{I_1}{N_1}$, $i_2 = \frac{I_2}{N_2}$. Since $N_j = S_j + I_j, j = 1, 2$ model (4.4c)-(4.5b) can be rewritten as

$$\frac{di_1}{dt} = (1 - i_1)(\beta_{11} i_1 + \beta_{12} i_2 - \alpha_1 i_1) - a_1 i_1 \left(1 - \frac{N_1}{\theta_{11}} - \frac{N_2}{\theta_{12}}\right), \tag{4.13a}$$

$$\frac{di_2}{dt} = (1 - i_2)(\beta_{22} i_2 + \beta_{21} i_1 - \alpha_2 i_2) - a_2 i_2 \left(1 - \frac{N_2}{\theta_{22}} - \frac{N_1}{\theta_{21}}\right), \tag{4.13b}$$

$$\frac{dN_1}{dt} = a_1 N_1 \left(1 - \frac{N_1}{\theta_{11}} - \frac{N_2}{\theta_{12}}\right) - b_1 N_1 - \alpha_1 i_1 N_1, \tag{4.13c}$$

$$\frac{dN_2}{dt} = a_2 N_2 \left(1 - \frac{N_2}{\theta_{22}} - \frac{N_1}{\theta_{21}}\right) - b_2 N_2 - \alpha_2 i_2 N_2. \tag{4.13d}$$

The model (4.13a)-(4.13d) makes ecological sense and is mathematically well-posed in the domain $\mathcal{D}^2 = \{(i_1, i_2, N_1, N_2) \in \mathbb{R}^4 | 0 \leq i_1, i_2 \leq 1, 0 < N_i \leq K_{ii}, i = 1, 2\}$. Unlike [31], in which density-dependent death rates were considered, the equations (4.13a)-(4.13d) do not decouple when rewritten in terms of proportions of infected individuals.

We follow the approach in [31] and prove the following results.

Theorem 4.2 *For frequency incidence, a unique endemic equilibrium exists for the SI model with competition,* (4.13a)-(4.13d)*, if and only if* (i) $\mathcal{R}_{jj} > 1$ *for either* $j = 1$ *or* $j = 2$ *or* (ii) $\mathcal{R}_{jj} \leq 1$ *for both* $j = 1, 2$ *and* $(1 - \mathcal{R}_{11})(1 - \mathcal{R}_{22}) < \mathcal{R}_{12}\mathcal{R}_{21}$.

Proof. We note that conditions (i) and (ii) are equivalent to $\mathcal{R}_0^C > 1$ for $\mathcal{R}_0^C$ defined in (4.9) and (4.10) (see [31] for the proof of a similar result).

We begin by setting (4.13c) and (4.13d) equal to zero, i.e., where $N_1' = 0$ and $N_2' = 0$, so that

$$N_1^*(i_1, i_2) = S_{1,\infty}^4 + H_1(i_1, i_2), \tag{4.14a}$$

$$N_2^*(i_1, i_2) = S_{2,\infty}^4 + H_2(i_1, i_2), \tag{4.14b}$$

for $(i_1, i_2) \in D = [0, 1] \times [0, 1]$, and $S_{i,\infty}^4, i = 1, 2$ are defined in (3.57)-(3.58). The functions H_1 and H_2 are defined as

$$H_1(i_1, i_2) = \left(\frac{\alpha_1 i_1 K_{12}}{r_1} - \frac{\alpha_2 i_2 K_{22}}{r_2}\right)\left(\frac{K_{22}}{K_{21}} - \frac{K_{12}}{K_{11}}\right)^{-1}, \tag{4.15a}$$

$$H_2(i_1, i_2) = \left(\frac{\alpha_2 i_2 K_{21}}{r_2} - \frac{\alpha_1 i_1 K_{11}}{r_1}\right)\left(\frac{K_{11}}{K_{12}} - \frac{K_{21}}{K_{22}}\right)^{-1}. \tag{4.15b}$$

We then substitute N_1^* and N_2^* into equations (4.13a), and (4.13b) resulting in the equations

$$\frac{di_1}{dt} = (1-i_1)(\beta_{11}i_1 + \beta_{12}i_2 - \alpha_1 i_1) - i_1(b_1 + \alpha_1 i_1), \tag{4.16a}$$

$$\frac{di_2}{dt} = (1-i_2)(\beta_{22}i_2 + \beta_{21}i_1 - \alpha_2 i_2) - i_2(b_2 + \alpha_2 i_2). \tag{4.16b}$$

Setting (4.16a) and (4.16b) equal to zero, we obtain the nullclines for i_1 and i_2 as

$$i_2 = f_1(i_1) = \frac{i_1[b_1 + \alpha_1 i_1 - (1-i_1)(\beta_{11} - \alpha_1)]}{(1-i_1)\beta_{12}}, \tag{4.17a}$$

$$i_1 = f_2(i_2) = \frac{i_2[b_2 + \alpha_2 i_2 - (1-i_2)(\beta_{22} - \alpha_2)]}{(1-i_2)\beta_{21}}. \tag{4.17b}$$

We note that the domain $D = [0,1] \times [0,1]$ is invariant for the sytem (4.16a) and (4.16b), since if $i_k = 0$ then $di_k/dt > 0$ and if $i_k = 1$ then $di_k/dt < 0$, for $k = 1, 2$. The nullclines always intersect at the origin in D. The function f_1 has an asymptote at $i_1 = 1$, and f_2 has an asymptote at $i_2 = 1$ and

$$\frac{df_1}{di_1}|_{i_1=0} = \frac{b_1 + \alpha_1 - \beta_{11}}{\beta_{12}}, \tag{4.18}$$

and

$$\frac{df_2}{di_2}|_{i_2=0} = \frac{b_2 + \alpha_2 - \beta_{22}}{\beta_{21}}. \tag{4.19}$$

Also,

$$\frac{d^2 f_k}{di_k^2}|_{i_k=0} = \frac{2b_k + 2\alpha_k}{\beta_{kj}} > 0, k = 1, 2, \tag{4.20}$$

since all parameters are positive indicating that both $f_1(i_1)$ and $f_2(i_2)$ are concave up on their respective axes.

Sufficiency part of proof: We break this part up into four cases:

Case (1): Assume that $\mathcal{R}_{11} > 1$ and $\mathcal{R}_{22} > 1$. Then, we can see from (4.10) that $\beta_{ii} > \Gamma_i = b_i + \alpha_i$, for $i = 1, 2$. Using this in equations (4.18) and (4.19), we find that

$$\frac{df_k}{di_k}|_{i_k=0} < 0, \tag{4.21}$$

which implies that there is one point of intersection in D (see Figure 1).

Case (2): Assume $\mathcal{R}_{11} < 1$ and $\mathcal{R}_{22} > 1$. Then $\frac{df_1}{di_1}|_{i_1=0} > 0$ and $\frac{df_2}{di_2}|_{i_2=0} < 0$, so that f_1 and f_2 again intersect uniquely in D (see Figure 2).

Case (3): Assume $\mathcal{R}_{11} > 1$ and $\mathcal{R}_{22} < 1$. Changing roles in Case (2), we again have that f_1 and f_2 intersect uniquely in D.

Case (4): Lastly, we consider the case where $\mathcal{R}_{11} < 1$ and $\mathcal{R}_{22} < 1$, and $(1-\mathcal{R}_{11})(1-\mathcal{R}_{22}) < \mathcal{R}_{12}\mathcal{R}_{21}$. This implies that $\frac{df_k}{di_k}|_{i_k=0} > 0$ for $k = 1, 2$. In order for the nullclines to cross in D, we must also have

$$\frac{df_1}{di_1}|_{i_1=0} < \frac{1}{\frac{df_2}{di_2}|_{i_2=0}}. \tag{4.22}$$

This is equivalent to $(1-\mathcal{R}_{11})(1-\mathcal{R}_{22}) < \mathcal{R}_{12}\mathcal{R}_{21}$, which holds by assumption for Case 4 (see Figure 3).

Necessary part of proof: Assume that there exists a unique endemic equilibrium but that conditions (i) and (ii) of Theorem 4.2 do not hold. So, $\mathcal{R}_{jj} < 1$ for $j = 1, 2$ and $(1-\mathcal{R}_{11})(1-\mathcal{R}_{22}) \geq \mathcal{R}_{12}\mathcal{R}_{21}$. This implies that $\frac{df_k}{di_k}|_{i_k=0} > 0$ for $k = 1, 2$. However, the condition $\frac{df_1}{di_1}|_{i_1=0} < \frac{1}{\frac{df_2}{di_2}|_{i_2=0}}$ no longer holds, hence the nullclines do not intersect in the interior of D, which contradicts the assumption of existence of a unique endemic equilibrium (see Figure 4).

Existence of a unique point of intersection $(i_{1,\infty}, i_{2,\infty})$ in D then gives us existence of the infected coexistence equilibrium $(i_{1,\infty}, i_{2,\infty}, N_1^*(i_{1,\infty}, i_{2,\infty}), N_2^*(i_{1,\infty}, i_{2,\infty}))$ in $\mathcal{D}^2$ of model (4.13a)-(4.13d), by substituting $(i_{1,\infty}, i_{2,\infty})$ into equations (4.14a) and (4.15b). ■

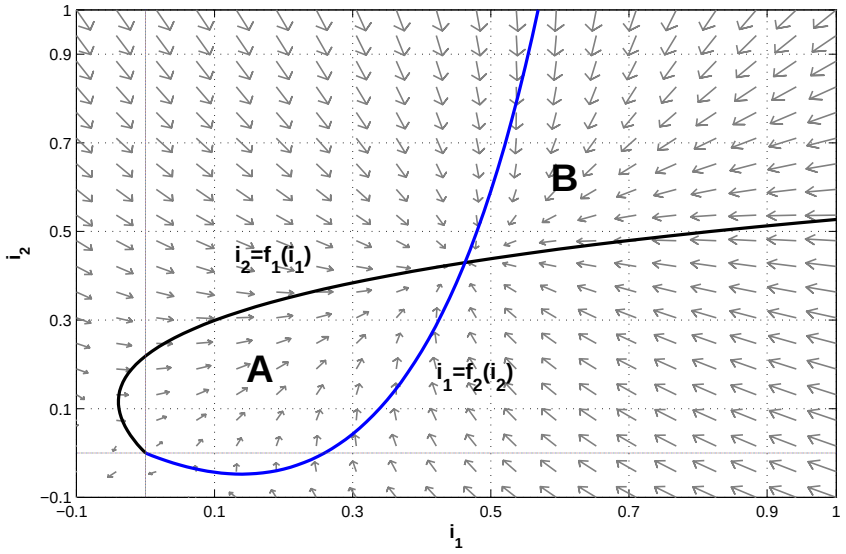

Figure 1: Isoclines for the case where both $\mathcal{R}_{11}, \mathcal{R}_{22} > 1$. Disease related parameters are $\beta_{11} = 2.7$, $\beta_{22} = 3.2$, $\beta_{12} = 1.1$, and $\beta_{21} = 1.1$. The parameters related to population dynamics are $\alpha_1 = 1$, $\alpha_2 = .5$, $b_1 = 1$, and $b_2 = 2$.

Theorem 4.3 *Consider the proportions model* (4.16a)-(4.16b). *If* $\mathcal{R}_0^C < 1$ *then the disease free equilibrium* $(i_{1,\infty} = 0, i_{2,\infty} = 0)$ *is asymptotically stable in the region* D *and if* $\mathcal{R}_0^C > 1$ *then the infected coexistence (endemic) equilibrium is asymptotically stable in* $D_+ = D \setminus \{(0,0)\}$.

Proof. Suppose $\mathcal{R}_0^C < 1$. Then by Theorem 4.2 there is no infected coexistence equilibrium in D. The only equilibrium for (4.16a)-(4.16b) is the origin in D, and is locally asymptotically stable by [40]. The Poincare-Bendixson Trichotomy [1] states that a positive orbit of the system that remains in a closed and bounded region of the plane with only a finite number of equilibria will have an omega limit set that takes on only one of three forms, namely, an equilibrium, a periodic orbit, or a finite number of equilibria. Since the solutions of our system are indeed bounded and the only equilibrium in the region $D = [0,1] \times [0,1]$ for

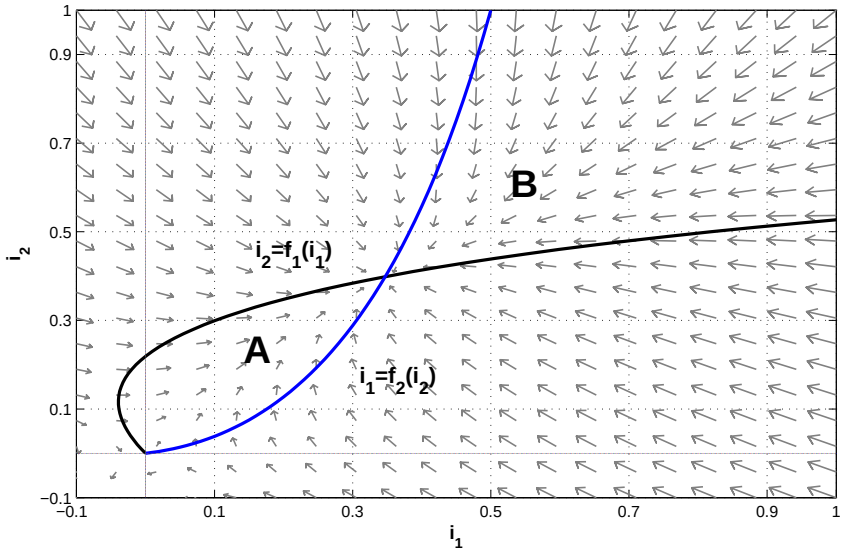

Figure 2: Isoclines for the case where $\mathcal{R}_{11} < 1$ and $\mathcal{R}_{22} > 1$. Disease related parameters are $\beta_{11} = 1.8$, $\beta_{22} = 3.2$, $\beta_{12} = 1.1$, and $\beta_{21} = 1.1$. The parameters related to population dynamics are $\alpha_1 = 1$, $\alpha_2 = .5$, $b_1 = 1$, and $b_2 = 2$.

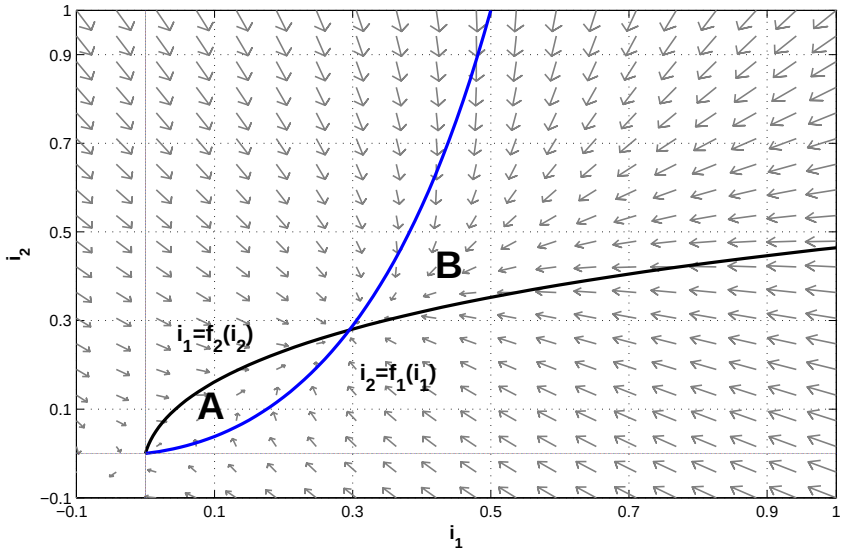

Figure 3: Isoclines for the case where $\mathcal{R}_{11}, \mathcal{R}_{22} < 1$ but $(1-\mathcal{R}_{11})(1-\mathcal{R}_{22}) < \mathcal{R}_{12}\mathcal{R}_{21}$. Disease related parameters are $\beta_{11} = 1.8$, $\beta_{22} = 2.3$, $\beta_{12} = 1.1$, and $\beta_{21} = 1.1$. The parameters related to population dynamics are $\alpha_1 = 1$, $\alpha_2 = .5$, $b_1 = 1$, and $b_2 = 2$.

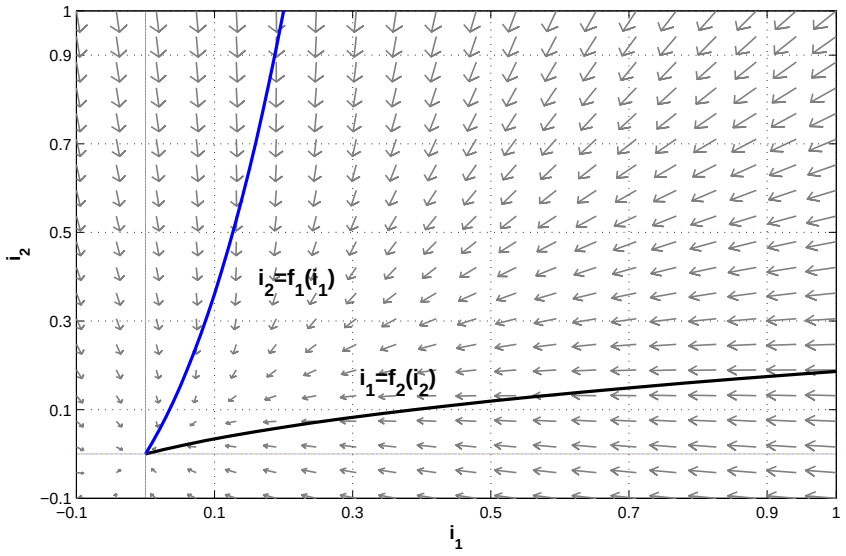

Figure 4: Isoclines for the case where neither condition (i) nor (ii) of Theorem 4.2 hold. Disease related parameters are $\beta_{11} = 1.5$, $\beta_{22} = 2$, $\beta_{12} = .2$, and $\beta_{21} = .2$. The parameters related to population dynamics are $\alpha_1 = 1$, $\alpha_2 = .5$, $b_1 = 1$, and $b_2 = 2$.

(4.16a)-(4.16b) is the origin which is stable, there are no periodic solutions in the region and the origin is stable for (4.16a)-(4.16b).

Next suppose $\mathcal{R}_0^C > 1$. Then by Theorem 4.2 there is a unique infected coexistence equilibrium, $(i_{1,\infty}, i_{2,\infty})$, for (4.16a)-(4.16b). We first will show that no solution of (4.16a)-(4.16b) in the invariant region D_+ will approach the origin. The Jacobian for (4.16a) and (4.16b) evaluated at the origin is

$$\mathcal{J}(0,0) = \begin{bmatrix} \beta_{11} - (\alpha_1 + b_1) & \beta_{12} \\ \beta_{21} & \beta_{22} - (\alpha_2 + b_2) \end{bmatrix},$$

which has eigenvalues

$$\lambda_1, \lambda_2 = \frac{1}{2}[(\beta_{11} - \Gamma_1) + (\beta_{22} - \Gamma_2) \pm \sqrt{[(\beta_{11} - \Gamma_1) - (\beta_{22} - \Gamma_2)]^2 + 4\beta_{21}\beta_{12}}], \tag{4.23}$$

where $\Gamma_i = \alpha_i + b_i$. Since $\mathcal{R}_0^C > 1$ then we know either one of $\beta_{11} - \Gamma_1$ and $\beta_{22} - \Gamma_2$ are positive or both are negative and $(\beta_{11} - \Gamma_1)(\beta_{22} - \Gamma_2) < \beta_{12}\beta_{21}$, both cases for which $\lambda_1 > 0$. Now, if $\lambda_2 > 0$ as well then the origin is a repellor. If, on the other hand, $\lambda_2 < 0$ then the eigenvector of λ_2 is

$$\begin{bmatrix} x_1 \\ x_2 \end{bmatrix} = \begin{bmatrix} \frac{1}{\beta_{21}}(\lambda_2 - (\beta_{22} - \Gamma_2)) \\ 1 \end{bmatrix}. \tag{4.24}$$

Since $\lambda_2 < 0$ then we can see that $x_1 < 0$ also and the stable manifold of the origin does not lie in D_+. Hence, none of the solutions in D_+ approach the DFE $(0,0)$ in D.

Lastly, we need to consider the endemic equilibrium and show that no periodic solutions exist inside D_+. We can see by examining the phase plane of the proportions system (4.16a) and (4.16b) and through computations that the region, A, enclosed by the nullclines of i_1 and

i_2 but to the left of and below the endemic equilibrium is invariant. Along the i_1 nullcline in A, $di_2/dt > 0$ and along the i_2 nullcline in A, $di_1/dt > 0$, which proves that the region A is invariant. The region to the right of and above the endemic equilibrium, B, enclosed by the nullclines is also invariant in the opposite direction. So, any solution trajectory that tries to orbit around the endemic equilibrium will be 'trapped' in either region A or region B and will approach the endemic equilibrium. Thus, no periodic solutions exist. Since the solutions are bounded, we can use the Poincare-Bendixson Trichotomy to deduce that all solution trajectories approach the infected coexistence equilibrium, and therefore it is asymptotically stable in the region D_+.

■

We note that the stability of the infected coexistence equilibrium of the proportions model (4.16a)-(4.16b) need not guarantee stability of the infected coexistence of the model (4.13a)-(4.13d).

5 Conclusion and Discussion

The effects of a shared disease on the outcome of competition between two species has been investigated by several authors in the ecological and mathematical ecology communities. Although many papers propose and analyze two species mathematical models of Lotka-Volterra competition between the two species that share a common (generalist) pathogen, some important cases are difficult to analyze. In particular, it has been difficult to find existence and stability conditions of the infected coexistence equilibrium for these models.

In this paper, we consider a competition model with density independent death rates and a shared disease that spreads by either mass action or frequency incidence transmission.

In the first set of results, we derive equilibrium values and stability conditions for a two species Lotka-Volterra competition model with density independent death rates and mass action disease incidence. All of the existence and stability conditions can be derived in terms of competitive interactions represented by two parameters, ξ_1 and ξ_2, that measure the relative importance of intra versus inter specific competition, and in terms of basic reproduction numbers (BRNs) for species in isolation or for coexisting species. The infected coexistence equilibrium, however, remains intractable. Hence, we simplify the model by assuming that the two species are similar enough to have the same intra-specific competition rates and to transmit the disease to each other at the same rates. We also assume that the pathogen does not cause death in its hosts, similar to the common cold in humans, for example. Under these constraints, we derive all the existence and stability conditions for the equilibria of the mass action disease model. We prove that a conjecture made in [26, 5, 18, 9] about the infected coexistence equilibrium, holds for our simplified model. In particular, we show that the conditions under which infected coexistence is stable guarantee that all other equilibria are unstable and vice versa.

Our results in the case of mass action disease transmission show that, if the death rate due to disease is positive, then disease can reduce the total equilibrium density for each species in isolation. This in turn affects competitive ability indirectly (apparent competition), and is another indication that in the presence of disease, the competitive outcome can change. However, it has thus far been impossible to analyze the stability of coexistence equilibria

for the most general mass action model, and hence to find conditions under which disease allows the coexistence of species that would not normally coexist. We hypothesize that the main driving force behind the possible switch of competitive outcomes is death due to disease. This force may be magnified by differing rates of transmission between and within species. Accordingly, we simplify the mass action model so that there is negligible death due to disease and no significant difference between transmission rates. Analysis of this simplified model is tractable and we determine that the presence of disease does not change the competitive outcome of the disease free case.

Next, we consider Lotka-Volterra competition between the two species with density-independent death rates and frequency incidence disease transmission. We prove the existence, and uniqueness of the infected coexistence equilibrium under the assumption that coexistence of the species is feasible. We show that the stability of the infected coexistence equilibrium of a related proportions model depends on the value of the basic reproduction number (BRN) being greater than one. However, the stability analysis of infected coexistence of the full model is not obtained.

In conclusion, we have presented a thorough literature review of models of two interacting species that are both affected by a pathogen. We have presented previously known results in this area along with new results in a unified setting that simplifies the analysis, and stresses the role of the basic reproduction number as well as the relative strengths of intra- versus inter-specific competition for both species.

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