An Analysis of the Coexistence of Three Competing Species with a Shared Pathogen

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Abstract

We consider an SI model of three competing species that are all affected by a single pathogen which is transmitted directly via mass action. The total population sizes of the three species satisfy a three-dimensional Lotka-Volterra competition model. We address the interaction between competition and disease dynamics, and show that infected coexistence in the model is determined by the values of the basic reproduction numbers as well as the relative strengths of intraspecific crowding versus interspecific competition for all three species.

Keywords: Lotka-Volterra Competition, three-species coexistence, SI mass-action model, basic reproduction number

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1 Introduction

Competitive interactions are one of the primary ways that multiple species can interact. Although many mathematical models of competing species have been developed and analyzed, the influence of disease on a competitive system is less understood, particularly among systems of more than two species.

Examples of disease that affect systems of competing species can be readily found in nature [7]. The American grey squirrel was recently introduced to the UK, where they have proven both competitively stronger than the native red squirrel and serve as a vector of squirrelpox virus (SQPV) to which they are immune. This virus is

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nearly always fatal to red squirrels, who have little to no natural resistance to the disease [11]. SQPV accelerates ecological replacement of red squirrels to 17–25 times its rate in the absence of disease [9].

Competition between the native noble crayfish in Europe and the introduced American signal crayfish is similarly affected by crayfish plague. Signal crayfish are competitively stronger and are not killed by plague, which is fatal to noble crayfish [8].

The impact of the tick-borne louping ill virus can extend to much larger systems of host species. Gilbert et al. investigated its effect on varying systems including grouse, hares, and deer [3]. In their model, the host species do not directly compete for resources and the population of the tick vector was explicitly modeled.

In 2006, Hatcher et al. presented a survey of literature describing the effect of parasites on competitive and predatory relationships [4]. They note that complex host communities may be able to better support disease, citing the tick-grouse-hare-deer system. Additionally, they note that disease can produce apparent competition between species that do not directly compete, since the presence of a vector species is detrimental to the host species.

One of the first papers to examine a mathematical model for a system of three species was published by May and Leonard in 1975 [5]. The model consists of a Lotka-Volterra type system, assuming equal birth rates for all three species and symmetric competition (species 2 affects species 1 as 3 affects 2 as 1 affects 3). The authors demonstrate the existence of nonperiodic solutions with indefinitely increasing cycle times. Although mathematically intriguing and an excellent example of the complex dynamics of three-species systems, the biological relevance of this behavior is limited.

This work was continued in 1979 when Schuster et al. published an analysis of three models of three competing species [10], including the original May-Leonard model, a generalized version without the assumption of symmetry, and a related model incorporating constraints of constant organization to simulate macromolecular self-organization. They found that solutions of all models converged to cycles of three saddle points and three orbits between them.

Chi et al. conducted a further analysis of the asymmetric May-Leonard model in 1998, using alternate assumptions on its parameters to find and determine stability of equilibria, as well as obtain conditions for the existence of periodic solutions and neutral orbits [2].

Our primary reference for models involving both competition and disease are those developed by Bokil and Manore [1]. The authors examine two-species competition and disease systems with either mass action or frequency incidence disease transmission. Their analysis of the models includes locating equilibria and determining the biological conditions (in terms of the basic reproduction number and relative competitive strengths) necessary for their feasibility and stability. In the mass-action transmission case, the authors consider bifurcations in the model resulting in neutral state equilibria.

In this paper, we extend and combine these previous models and results to the

three-species system with Lotka-Volterra competition and mass action disease transmission. In particular, we show that in our model, the presence of disease cannot reverse the competitive outcome, indicating that system dynamics are primarily driven by competitive interactions.

The remainder of the paper is organized as follows. In Section 2, we introduce our model and the biologically-meaningful parameters with which we perform our analysis. In Section 3, we list the equilibria of the model and derive the conditions for their existence, using the basic reproductive numbers of the disease among 1, 2, and 3 species. In Section 4 we conduct a local stability analysis on each equilibrium. A summary of our results and further biological interpretation is given in Section 5.

2 Three Species Competition and Disease Model

Our three-species competition and mass action disease model is extended from the two-species SI model by Bokil and Manore [1]. For species $i = 1, 2, 3$, we have

$$\frac{dS_i}{dt} = aN_i\left(1 - \frac{N_1}{\theta_{i1}} - \frac{N_2}{\theta_{i2}} - \frac{N_3}{\theta_{i3}}\right) - bS_i - \beta S_i(I_1 + I_2 + I_3), \tag{2.1a}$$

$$\frac{dI_i}{dt} = \beta S_i(I_1 + I_2 + I_3) - bI_i, \tag{2.1b}$$

where S_i is the population of susceptible individuals of species i, I_i is the population of infected individuals of species i, and $N_i = S_i + I_i$ is the total population of species i. This results in a system of six equations, which we simplify by making the following assumptions on its parameters:

(A1) The death rate b is constant, density-independent, and the same for all species and for both susceptible and infected individuals.

(A2) Birth rates are density-dependent, with the intrinsic birth rate a reduced by both interspecific and intraspecific Lotka-Volterra competition.

We define the intrinsic per capita growth rate for each species to be $r = a - b > 0$.

We focus on mass action disease transmission, which is density-dependent and assumes contacts between individuals occur at a rate proportional to the total population size. Mass action is often used for directly-transmitted disease, whereas frequency-incidence transmission, which assumes a fixed contact rate, is more suitable for vector-transmitted disease [6]. Furthermore, mass action transmission is used in the model of the red-grey squirrel system developed by Tompkins et al. [11], which also leads us to assume:

(A3) The transmission coefficient β is constant for all species.

This model can be divided between dynamics due to competition and crowding and dynamics due to disease, as shown in Figures 1 and 2.

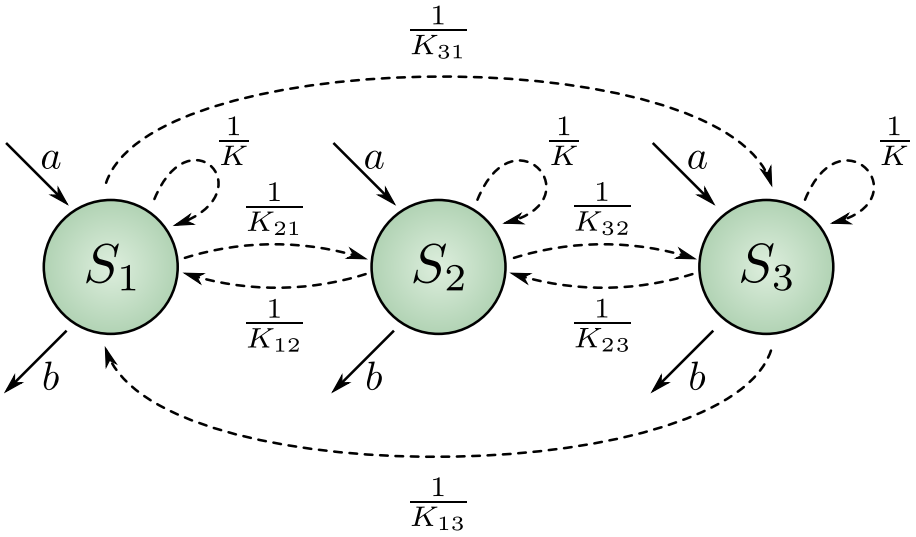


Figure 1: Conceptual model of competitive interactions between species. Dotted lines indicate inhibitive relationships; solid lines indicate non-competitive dynamics.

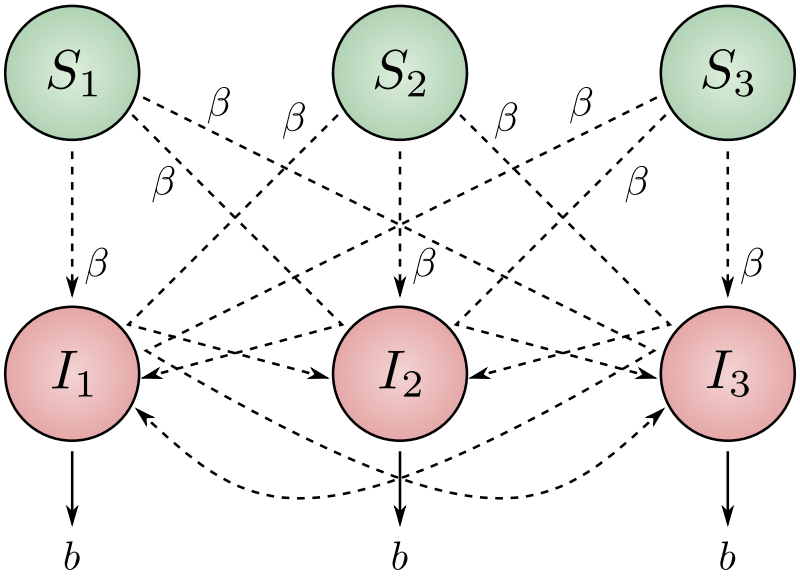


Figure 2: Conceptual model of disease dynamics between species. Dotted lines indicate infective relationships; solid lines indicate non-infective dynamics.

2.1 Additional Parameters

The competition parameters θ_{ij} are defined by the equality

$$\frac{a}{\theta_{ij}} := \frac{r}{K_{ij}}, \qquad i,j = 1,2,3,$$

where $\frac{1}{K_{ij}} \leq 1$ represents the inhibition strength of species j on species i. The carrying capacity of species i is K_{ii}. We make a final simplifying assumption:

(A4) All species have the same carrying capacity. Define $K := K_{11} = K_{22} = K_{33}$; similarly, $\theta := \theta_{11} = \theta_{22} = \theta_{33}$.

We define the relative strength of intraspecific crowding versus interspecific competition of species j on i to be

$$\xi_{ij} = \frac{1}{K_{jj}} - \frac{1}{K_{ij}}, \qquad i,j = 1,2,3. \tag{2.2}$$

Since $K = K_{jj} = K_{ii}$, there are two possible interpretations for this parameter. First, $\xi_{ij} > 0$ implies species j restricts its own growth more than it restricts the growth of species i. Alternatively, $\xi_{ij} > 0$ also implies growth of species i is more inhibited by its own population than by the population of species j.

3 Equilibria and Feasibility

There are 15 equilibria. Nontrivial equilibria are written $E_{C,i}$ or $E_{I,i}$, where the subscript C denotes a disease-free equilibrium, I denotes an infected equilibrium, and i is a combination of 1,2, or 3 indicating the species present. Equilibrium components are notated

$$E_{C,i} = (S_1^{C,i}, S_2^{C,i}, S_3^{C,i}, 0, 0, 0)$$

and

$$E_{I,i} = (S_1^{I,i}, S_2^{I,i}, S_3^{I,i}, I_1^{I,i}, I_2^{I,i}, I_3^{I,i}).$$

It is convenient to introduce the following lemma at this time:

Lemma 1. *The per-species total population sizes of the nontrivial equilibria are the same regardless of the presence of disease. Thus, for $j = 1,2,3$, we have $N_j^{C,i} = N_j^{I,i}$, i.e. $S_j^{C,i} = S_j^{I,i} + I_j^{I,i}$.*

Proof. From (2.1), the change in total population does not depend on the presence of disease:

$$\frac{dN_i}{dt} = N_i\left[a\left(1 - \frac{N_1}{\theta_{i1}} - \frac{N_2}{\theta_{i2}} - \frac{N_3}{\theta_{i3}}\right) - b\right] = rN_i\left[1 - \frac{N_1}{K_{i1}} - \frac{N_2}{K_{i2}} - \frac{N_3}{K_{i3}}\right]. \tag{3.1}$$

□

We now begin the equilibrium analysis.

3.1 Trivial Equilibrium

The trivial equilibrium is

$$E_0 = (0, 0, 0, 0, 0, 0)$$

and is always feasible.

3.2 One Host Disease-Free Equilibria

With one species and no disease, the susceptible population reaches carrying capacity:

$$\begin{aligned} E_{C,1} &= (K, 0, 0, 0, 0, 0), \\ E_{C,2} &= (0, K, 0, 0, 0, 0), \\ E_{C,3} &= (0, 0, K, 0, 0, 0). \end{aligned}$$

These equilibria are always feasible.

3.3 One Host Infected Equilibria

In the presence of disease, the total population remains at carrying capacity but is divided between the susceptible and infected classes. For infected equilibria, a threshold quantity called the basic reproduction number (BRN) [12] plays a very important role.

The BRN is the expected number of secondary infections arising from the introduction of an initial infected individual into an entirely susceptible population. For a single species, the BRN is defined as

$$\mathcal{R}_{0,1} = \frac{K\beta}{b} \tag{3.2}$$

for the disease affecting a species i in isolation [1]. We can then write these equilibria as

$$\begin{aligned} E_{I,1} &= \left(\frac{b}{\beta}, 0, 0, \frac{b}{\beta}\left(\mathcal{R}_{0,1} - 1\right), 0, 0 \right), \\ E_{I,2} &= \left(0, \frac{b}{\beta}, 0, 0, \frac{b}{\beta}\left(\mathcal{R}_{0,1} - 1\right), 0 \right), \\ E_{I,3} &= \left(0, 0, \frac{b}{\beta}, 0, 0, \frac{b}{\beta}\left(\mathcal{R}_{0,1} - 1\right) \right). \end{aligned}$$

Feasibility of these equilibria requires $\mathcal{R}_{0,1} > 1$.

3.4 Two Host Disease-Free Equilibria

The two host disease-free equilibria are

$$E_{C,12} = \left(\frac{KK_{12}}{K_{12} + K\frac{\xi_{21}}{\xi_{12}}}, \frac{\xi_{21}}{\xi_{12}} \left(\frac{KK_{12}}{K_{12} + K\frac{\xi_{21}}{\xi_{12}}} \right), 0, 0, 0, 0 \right), \tag{3.3a}$$

$$E_{C,13} = \left(\frac{KK_{13}}{K_{13} + K\frac{\xi_{31}}{\xi_{13}}}, 0, \frac{\xi_{31}}{\xi_{13}} \left(\frac{KK_{13}}{K_{13} + K\frac{\xi_{31}}{\xi_{13}}} \right), 0, 0, 0 \right), \tag{3.3b}$$

$$E_{C,23} = \left(0, \frac{KK_{23}}{K_{23} + K\frac{\xi_{32}}{\xi_{23}}}, \frac{\xi_{32}}{\xi_{23}} \left(\frac{KK_{23}}{K_{23} + K\frac{\xi_{32}}{\xi_{23}}} \right), 0, 0, 0 \right). \tag{3.3c}$$

The feasibility of these equilibria is determined by the ratio ξ_{ji}/ξ_{ij}, where i and j represent the two persisting species. As described by Bokil and Manore [1], there are four cases:

1. $\xi_{ij} > 0, \xi_{ji} > 0$: Intraspecific crowding is stronger than interspecific competition for both species. The equilibrium is feasible.

2. $\xi_{ij} < 0, \xi_{ji} > 0$: Species i is inhibited most by interspecific competition, whereas species j is inhibited most by intraspecific crowding. Species i "loses" the competitive interaction and the equilibrium is not biologically feasible.

3. $\xi_{ij} > 0, \xi_{ji} < 0$: Species i is inhibited most by intraspecific crowding, whereas species j is inhibited most by intraspecific competition. Species j "loses" the competitive interaction and the equilibrium is not biologically feasible.

4. $\xi_{ij} < 0, \xi_{ji} < 0$: Interspecific competition is stronger than intraspecific crowding for both species. The equilibrium is feasible.

3.5 Two Host Infected Equilibria

Theorem 1. *The basic reproduction number for the two host equilibrium with populations of species i and j is*

$$\mathcal{R}_{0,ij} = \frac{\beta}{b}(S_i^{C,ij} + S_j^{C,ij}) = \frac{\beta}{b} S_i^{C,ij} \left(1 + \frac{\xi_{ji}}{\xi_{ij}} \right).$$

Proof. The proof follows from [1]. □

We can then write the two host infected equilibria as

$$E_{I,12} = \left(\frac{b}{\beta\left(1+\frac{\xi_{21}}{\xi_{12}}\right)}, \frac{\xi_{21}}{\xi_{12}}\frac{b}{\beta\left(1+\frac{\xi_{21}}{\xi_{12}}\right)}, 0, \frac{b(\mathcal{R}_{0,12}-1)}{\beta\left(1+\frac{\xi_{21}}{\xi_{12}}\right)}, \frac{\xi_{21}}{\xi_{12}}\frac{b(\mathcal{R}_{0,12}-1)}{\beta\left(1+\frac{\xi_{21}}{\xi_{12}}\right)}, 0 \right), \quad (3.4a)$$

$$E_{I,13} = \left(\frac{b}{\beta\left(1+\frac{\xi_{31}}{\xi_{13}}\right)}, 0, \frac{\xi_{31}}{\xi_{13}}\frac{b}{\beta\left(1+\frac{\xi_{31}}{\xi_{13}}\right)}, \frac{b(\mathcal{R}_{0,13}-1)}{\beta\left(1+\frac{\xi_{31}}{\xi_{13}}\right)}, 0, \frac{\xi_{31}}{\xi_{13}}\frac{b(\mathcal{R}_{0,13}-1)}{\beta\left(1+\frac{\xi_{31}}{\xi_{13}}\right)} \right), \quad (3.4b)$$

$$E_{I,23} = \left(0, \frac{b}{\beta\left(1+\frac{\xi_{32}}{\xi_{23}}\right)}, \frac{\xi_{32}}{\xi_{23}}\frac{b}{\beta\left(1+\frac{\xi_{32}}{\xi_{23}}\right)}, 0, \frac{b(\mathcal{R}_{0,23}-1)}{\beta\left(1+\frac{\xi_{32}}{\xi_{23}}\right)}, \frac{\xi_{32}}{\xi_{23}}\frac{b(\mathcal{R}_{0,23}-1)}{\beta\left(1+\frac{\xi_{32}}{\xi_{23}}\right)} \right). \quad (3.4c)$$

Therefore, feasibility of $E_{I,ij}$ requires $\mathcal{R}_{0,ij} > 1$ and $\xi_{ji}/\xi_{ij} > 0$, for $i, j = 1, 2, 3$.

3.6 Coexistence Disease-Free Equilibrium

We will denote the coexistence disease-free equilibrium as

$$E_C = E_{C,123} = (N_1^C, N_2^C, N_3^C, 0, 0, 0).$$

The values of N_1^C, N_2^C, and N_3^C can be determined analogously to the method used by Chi et al. [2]. From (2.1) and (3.1), this equilibrium must satisfy

$$\frac{N_1^C}{K_{i1}} + \frac{N_2^C}{K_{i2}} + \frac{N_3^C}{K_{i3}} = 1, \quad (3.5)$$

for $i = 1, 2, 3$, since each species' birth rate equals its death rate. Let

$$M = \begin{bmatrix} \frac{1}{K} & \frac{1}{K_{12}} & \frac{1}{K_{13}} \\ \frac{1}{K_{21}} & \frac{1}{K} & \frac{1}{K_{23}} \\ \frac{1}{K_{31}} & \frac{1}{K_{32}} & \frac{1}{K} \end{bmatrix}, \quad \Delta = \det M,$$

and Δ_i for $i = 1, 2, 3$ be the determinant of the matrix formed by replacing the i-th column of M with a vector of ones. Then, using (2.2), it can be shown that

$$\Delta_1 = \xi_{12}\xi_{23} - \xi_{23}\xi_{32} + \xi_{32}\xi_{13} \quad (3.6a)$$

$$\Delta_2 = \xi_{23}\xi_{31} - \xi_{31}\xi_{13} + \xi_{13}\xi_{21} \quad (3.6b)$$

$$\Delta_3 = \xi_{31}\xi_{12} - \xi_{12}\xi_{21} + \xi_{21}\xi_{32} \quad (3.6c)$$

$$\Delta = \frac{\Delta_1 + \Delta_2 + \Delta_3}{K} - \xi_{13}\xi_{32}\xi_{21} - \xi_{12}\xi_{23}\xi_{31} \quad (3.6d)$$

From Cramer's rule, it follows that

$$(N_1^C, N_2^C, N_3^C, 0, 0, 0) = \left(\frac{\Delta_1}{\Delta}, \frac{\Delta_2}{\Delta}, \frac{\Delta_3}{\Delta}, 0, 0, 0 \right). \quad (3.7)$$

Making an assumption analogous to that made by Chi et al. [2], we have

$$0 < \frac{1}{K_{12}} < \frac{1}{K} < \frac{1}{K_{13}}, \quad 0 < \frac{1}{K_{23}} < \frac{1}{K} < \frac{1}{K_{21}}, \text{ and } \quad 0 < \frac{1}{K_{31}} < \frac{1}{K} < \frac{1}{K_{32}}.$$

Rewritten in terms of the constants ξ, this is

(A5) $\xi_{12}, \xi_{23}, \xi_{31} > 0$ and $\xi_{13}, \xi_{21}, \xi_{32} < 0$.

This assumption guarantees that $\Delta > 0$ and $\Delta_i > 0$ for $i = 1, 2, 3$, so the equilibrium E_C is feasible.

3.7 Infected Coexistence Equilibrium

Denote this equilibrium

$$E_I = E_{I,123} = (S_1^I, S_2^I, S_3^I, I_1^I, I_2^I, I_3^I).$$

Theorem 2. *Let $\Delta_S = \Delta_1 + \Delta_2 + \Delta_3$, where the Δ_i are defined as in* (3.6)*. The basic reproduction number of the infection among 3 species is*

$$\mathcal{R}_{0,3} = \frac{\beta}{b} \frac{\Delta_S}{\Delta}.$$

Proof. We use the next-generation matrix approach to compute $R_{0,3}$ [12]. Let $X = \begin{bmatrix} I_1 & I_2 & I_3 \end{bmatrix}^T$. Then the system (2.1) can be written as

$$\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X) = \begin{bmatrix} \beta S_1(I_1 + I_2 + I_3) \\ \beta S_2(I_1 + I_2 + I_3) \\ \beta S_3(I_1 + I_2 + I_3) \end{bmatrix} - \begin{bmatrix} bI_1 \\ bI_2 \\ bI_3 \end{bmatrix},$$

where $\mathcal{F}(X)$ represents new infections and $\mathcal{V}(X)$ represents all other dynamics. Let F and V be the Jacobians of $\mathcal{F}$ and $\mathcal{V}$ respectively evaluated at the coexistence disease-free equilibrium E_C. Then

$$F = \begin{bmatrix} \beta N_1^C & \beta N_1^C & \beta N_1^C \\ \beta N_2^C & \beta N_2^C & \beta N_2^C \\ \beta N_3^C & \beta N_3^C & \beta N_3^C \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} b & 0 & 0 \\ 0 & b & 0 \\ 0 & 0 & b \end{bmatrix}.$$

Then $\mathcal{R}_{0,3}$ is the spectral radius of the matrix

$$FV^{-1} = \frac{\beta}{b} \begin{bmatrix} N_1^C & N_1^C & N_1^C \\ N_2^C & N_2^C & N_2^C \\ N_3^C & N_3^C & N_3^C \end{bmatrix}.$$

A routine computation shows the eigenvalues of FV^{-1} are $0, 0$ and $\frac{\beta}{b}(N_1^C + N_2^C + N_3^C)$. Then from (3.6),

$$\mathcal{R}_{0,3} = \frac{\beta}{b} \frac{\Delta_S}{\Delta}.$$

□

Theorem 3. *The infected coexistence equilibrium has components* $S_i^I = \frac{b}{\beta}\frac{\Delta_i}{\Delta_S}$ *and* $I_i^I = S_i^I(\mathcal{R}_{0,3} - 1)$ *for* $i = 1, 2, 3$.

Proof. Since $dS_i = 0$ and $dI_i = 0$ at the equilibrium, (2.1) gives

$$S_i^I\left[a\left(1 - \sum_{j=1}^{3}\frac{N_j^I}{\theta_{ij}}\right) - b\right] + aI_i^I\left(1 - \sum_{j=1}^{3}\frac{N_j^I}{\theta_{ij}}\right) - \beta S_i^I\sum_{j=1}^{3}I_j^I = 0, \tag{3.8}$$

$$\beta S_i^I\sum_{j=1}^{3}I_j^I - bI_i^I = 0. \tag{3.9}$$

for $i = 1, 2, 3$. Let

$$\gamma = \beta\sum_{j=1}^{3}I_j^I. \tag{3.10}$$

and assume $\gamma \neq 0$. Thus from (3.9),

$$S_i^I = \frac{bI_i^I}{\gamma}. \tag{3.11}$$

Substituting (3.11) into (3.8) and using $N_i^I = S_i^I + I_i^I$ yields

$$\frac{bI_i^I}{\gamma}\left[a\left(1 - \sum_{j=1}^{3}\left(\frac{bI_j^I}{\theta_{ij}\gamma} + \frac{I_j^I}{\theta_{ij}}\right)\right) - b\right] + aI_i^I\left[1 - \sum_{j=1}^{3}\left(\frac{bI_j^I}{\theta_{ij}\gamma} + \frac{I_j^I}{\theta_{ij}}\right)\right] - bI_i^I = 0.$$

Divide by $I_i^I \neq 0$ and multiply both sides by γ^2 to get

$$ab\gamma - ab\sum_{j=1}^{3}\left(\frac{bI_j^I}{\theta_{ij}} + \frac{\gamma I_j^I}{\theta_{ij}}\right) - b^2\gamma + a\gamma^2 - a\gamma\sum_{j=1}^{3}\left(\frac{bI_i^I}{\theta_{ij}} + \frac{I_j^I\gamma}{\theta_{ij}}\right) - b\gamma^2 = 0,$$

$$\Rightarrow (a-b)(b+\gamma)\gamma - a(b+\gamma)\sum_{j=1}^{3}\frac{(b+\gamma)I_j^I}{\theta_{ij}} = 0.$$

Since $b + \gamma \neq 0$, it follows that

$$r\gamma - a(b+\gamma)\sum_{j=1}^{3}\frac{I_j^I}{\theta_{ij}} = 0.$$

Use $\frac{1}{\theta_{ij}} = \frac{r}{aK_{ij}}$ to get

$$\gamma - (b+\gamma)\sum_{j=1}^{3}\frac{I_j^I}{K_{ij}} = 0, \qquad i = 1, 2, 3 \tag{3.12}$$

and subtract this from the equation (3.12) corresponding to $i = l$ to obtain

$$\sum_{j=1}^{3} I_j^I \left(\frac{1}{K_{ij}} - \frac{1}{K_{lj}} \right) = 0.$$

For $i = 1$ and $l = 2$, we have

$$I_1^I \left(\frac{1}{K} - \frac{1}{K_{21}} \right) + I_2^I \left(\frac{1}{K_{12}} - \frac{1}{K} \right) + I_3^I \left(\frac{1}{K_{13}} - \frac{1}{K_{23}} \right) = 0.$$

Since $\xi_{ij} = \frac{1}{K} - \frac{1}{K_{ij}}$, we get

$$\xi_{21} I_1^I - \xi_{12} I_2^I + (\xi_{23} - \xi_{13}) I_3^I = 0. \tag{3.13}$$

Similarly, for $i = 1$ and $l = 3$,

$$\xi_{31} I_1^I + (\xi_{32} - \xi_{12}) I_2^I - \xi_{13} I_3^I = 0. \tag{3.14}$$

From (3.13) and (3.14), we have

$$I_2^I \left(\frac{\xi_{12}\xi_{31} - \xi_{12}\xi_{21} + \xi_{32}\xi_{21}}{\xi_{31}\xi_{21}} \right) = I_3^I \left(\frac{\xi_{13}\xi_{21} - \xi_{13}\xi_{31} + \xi_{23}\xi_{31}}{\xi_{31}\xi_{21}} \right).$$

Thus, $I_2^I = \frac{\Delta_2}{\Delta_3} I_3^I$. It can be shown analogously that

$$I_j^I = \frac{\Delta_j}{\Delta_k} I_k^I, \tag{3.15}$$

where $k = 1, 2, 3$ and $j = 1, 2, 3$. Setting $\Delta_S = \Delta_1 + \Delta_2 + \Delta_3$ and substituting (3.15) into (3.12) for $i = 1$, we can obtain

$$\beta \Delta_S \left(\frac{I_1^I}{\Delta_1} \right) - \left[b + \beta \Delta_S \left(\frac{I_1^I}{\Delta_1} \right) \right] \left(\frac{I_1^I}{\Delta_1} \right) \left(\frac{\Delta_1}{K} + \frac{\Delta_2}{K_{12}} + \frac{\Delta_3}{K_{13}} \right) = 0.$$

Setting $\delta_1 = \frac{\Delta_1}{K} + \frac{\Delta_2}{K_{12}} + \frac{\Delta_3}{K_{13}}$, it follows that

$$\beta \Delta_S - b\delta_1 - \frac{\beta \delta_1 I_1^I \Delta_S}{\Delta_1} = 0.$$

Then

$$I_1^I = \frac{b}{\beta} \frac{\Delta_1}{\Delta_S} \left(\frac{\beta}{b} \frac{\Delta_S}{\delta_1} - 1 \right). \tag{3.16}$$

Similarly, for $i = 2, 3$, we obtain

$$I_i^I = \frac{b}{\beta} \frac{\Delta_i}{\Delta_S} \left(\frac{\beta}{b} \frac{\Delta_S}{\delta_i} - 1 \right) \quad \text{with} \quad \delta_i = \frac{\Delta_i}{K} + \frac{\Delta_j}{K_{ij}} + \frac{\Delta_l}{K_{il}},$$

and $j, l = 1, 2, 3$ where $i \neq j, l$.

Note that from (3.11) and (3.15), we have

$$S_i^I = \frac{b}{\beta} \frac{\Delta_i}{\Delta_S}, \tag{3.17}$$

for $i = 1, 2, 3$. Thus, (3.16) can be written as

$$I_1^I = S_1^I \left(\frac{\beta}{b} \frac{\Delta_S}{\delta_1} - 1 \right). \tag{3.18}$$

From (3.7) and (3.5) for $i = 1$, we know $\Delta_i = \Delta N_i^C$ and $\frac{N_1^C}{K} + \frac{N_2^C}{K_{12}} + \frac{N_3^C}{K_{13}} = 1$. Thus,

$$\delta_1 = \frac{\Delta N_1^C}{K} + \frac{\Delta N_2^C}{K_{12}} + \frac{\Delta N_3^C}{K_{13}} = \Delta. \tag{3.19}$$

It follows that

$$\frac{\Delta_S}{\delta_1} = N_1^C + N_2^C + N_3^C,$$

and therefore (3.18) can be written as $I_1^I = S_1^I \left(\mathcal{R}_{0,3} - 1 \right)$. This can be proved similarly for the general case, and so for $i = 1, 2, 3$,

$$I_i^I = S_i^I (\mathcal{R}_{0,3} - 1).$$

□

4 Local Stability Analysis

Trivial Equilibrium

The Jacobian of the system (2.1) evaluated at E_0 is

$$\mathcal{J}(E_0) = \begin{bmatrix} r & 0 & 0 & a & 0 & 0 \\ 0 & r & 0 & 0 & a & 0 \\ 0 & 0 & r & 0 & 0 & a \\ 0 & 0 & 0 & -b & 0 & 0 \\ 0 & 0 & 0 & 0 & -b & 0 \\ 0 & 0 & 0 & 0 & 0 & -b \end{bmatrix}$$

The eigenvalues of $\mathcal{J}(E_0)$ are r and $-b$, each with algebraic multiplicity 3. Since $r > 0$, this equilibrium is always unstable.

4.1 One Host Equilibria

One Host Disease-Free Equilibria

The Jacobian evaluated at, for example, $E_{C,1}$, is

$$\mathcal{J}(E_{C,1}) = \begin{bmatrix} -r & -\frac{rK}{K_{12}} & -\frac{rK}{K_{13}} & 2b-a-\beta K & -\frac{rK}{K_{12}}-\beta K & -\frac{rK}{K_{13}}-\beta K \\ 0 & rK\xi_{21} & 0 & 0 & -\frac{rK}{K_{21}}+a & 0 \\ 0 & 0 & rK\xi_{31} & 0 & 0 & -\frac{rK}{K_{31}}+a \\ 0 & 0 & 0 & \beta K-b & \beta K & \beta K \\ 0 & 0 & 0 & 0 & -b & 0 \\ 0 & 0 & 0 & 0 & 0 & -b \end{bmatrix}.$$

$\mathcal{J}(E_{C,2})$ and $\mathcal{J}(E_{C,3})$ are similar. For $i = 1, 2, 3$, the equilibrium $E_{C,i}$ has eigenvalues $-r, -b, -b, \beta K - b, rK\xi_{ji}$, and $rK\xi_{ki}$, where j and k represent the absent species.

Thus, stability requires $\xi_{ji} < 0, \xi_{ki} < 0$, and $K < \frac{b}{\beta}$. From the basic reproduction number (3.2), these conditions are that $\mathcal{R}_{0,1} < 1$ and that species i inhibits the other species more than it inhibits itself, preventing them from invading the system.

One Host Infected Equilibria

The Jacobian at $E_{I,1}$ is

$$\mathcal{J}(E_{I,1}) = \begin{bmatrix} 2b-a-\beta K & -\frac{rK}{K_{12}} & -\frac{rK}{K_{13}} & -r & -\frac{rK}{K_{12}}-b & -\frac{rK}{K_{13}}-b \\ 0 & a-\beta K-\frac{rK}{K_{21}} & 0 & 0 & -\frac{rK}{K_{21}}+a & 0 \\ 0 & 0 & a-\beta K-\frac{rK}{K_{31}} & 0 & 0 & -\frac{rK}{K_{31}}+a \\ \beta K-b & 0 & 0 & 0 & b & b \\ 0 & \beta K-b & 0 & 0 & -b & 0 \\ 0 & 0 & \beta K-b & 0 & 0 & -b \end{bmatrix}.$$

which has eigenvalues $-r, -\beta K, -\beta K, b-\beta K, rK\xi_{21}$, and $rK\xi_{31}$. The equilibria $E_{I,2}$ and $E_{I,3}$ are similar.

Thus, as in the disease-free case, species i must inhibit the other species more than itself (implying $\xi_{ji}, \xi_{ki} < 0$). The requirement $R_{0,1} > 1$ implied by the feasibility of the equilibria is now also a stability requirement.

4.2 Two Host Equilibria

Consider the equilibrium of species 1 and 2. For any equilibrium with nonzero populations of species i, (3.5) holds. Thus, the characteristic polynomial of the Jacobian evaluated at $E_e = (S_1^e, S_2^e, S_3^e, I_1^e, I_2^e, I_3^e)$ is

$$P(x) = (x+\eta)^2(x+\epsilon)\left(x^3 + \delta_1 x^2 + \delta_2 x + \delta_3\right)$$

where

$$\begin{aligned}
\eta &= b + \beta(I_1^e + I_2^e) \\
\epsilon &= b + \beta(I_1^e + I_2^e) - \beta(S_1^e + S_2^e) \\
\delta_1 &= r\left[N_1\left(\frac{1}{K} + \frac{1}{K_{31}}\right) + N_2\left(\frac{1}{K} + \frac{1}{K_{32}}\right) - 1\right] \\
\delta_2 &= r^2\left[N_1N_2\left(\frac{1}{K^2} - \frac{1}{K_{12}K_{21}}\right) + \frac{N_1+N_2}{K}\left(\frac{N_1}{K_{31}} + \frac{N_2}{K_{32}} - 1\right)\right] \\
\delta_3 &= r^3N_1N_2\left(\frac{1}{K^2} - \frac{1}{K_{12}K_{21}}\right)\left(\frac{N_1}{K_{31}} + \frac{N_2}{K_{32}} - 1\right)
\end{aligned}$$

The eigenvalue $-\eta$ is clearly negative. In the disease-free case, we can use Theorem 1 to rewrite the eigenvalue $-\epsilon$ as $b(\mathcal{R}_{0,12} - 1)$. In the infected case, Theorem 1 and (3.4a) yield $-\epsilon = b(1-\mathcal{R}_{0,12})$. By extension to the other two-host equilibria, it follows that $\mathcal{R}_{0,ij} < 1$ is required for stability of the disease-free equlibria with species i and j, and $\mathcal{R}_{0,ij} > 1$ is required for stability of the analogous infected equilibria.

The quantity $x^3 + \delta_1 x^2 + \delta_2 x + \delta_3$ is the characteristic polynomial of

$$\tilde{\mathcal{A}} = -r\begin{bmatrix} \frac{N_1^e}{K} & \frac{N_1^e}{K_{12}} & \frac{N_1^e}{K_{13}} \\ \frac{N_2^e}{K_{21}} & \frac{N_2^e}{K} & \frac{N_2^e}{K_{23}} \\ 0 & 0 & \frac{N_1^e}{K_{31}} + \frac{N_2^e}{K_{32}} - 1 \end{bmatrix}.$$

Let $\tilde{\mathcal{A}}_1$ be the 2×2 matrix formed by removing the last row and column from $\tilde{\mathcal{A}}$. Since the bottom rightmost element of $\tilde{\mathcal{A}}$ is one of its eigenvalues, we can factor the original cubic polynomial into $(x + \gamma)(x^2 + \alpha_1 x + \alpha_2)$ where

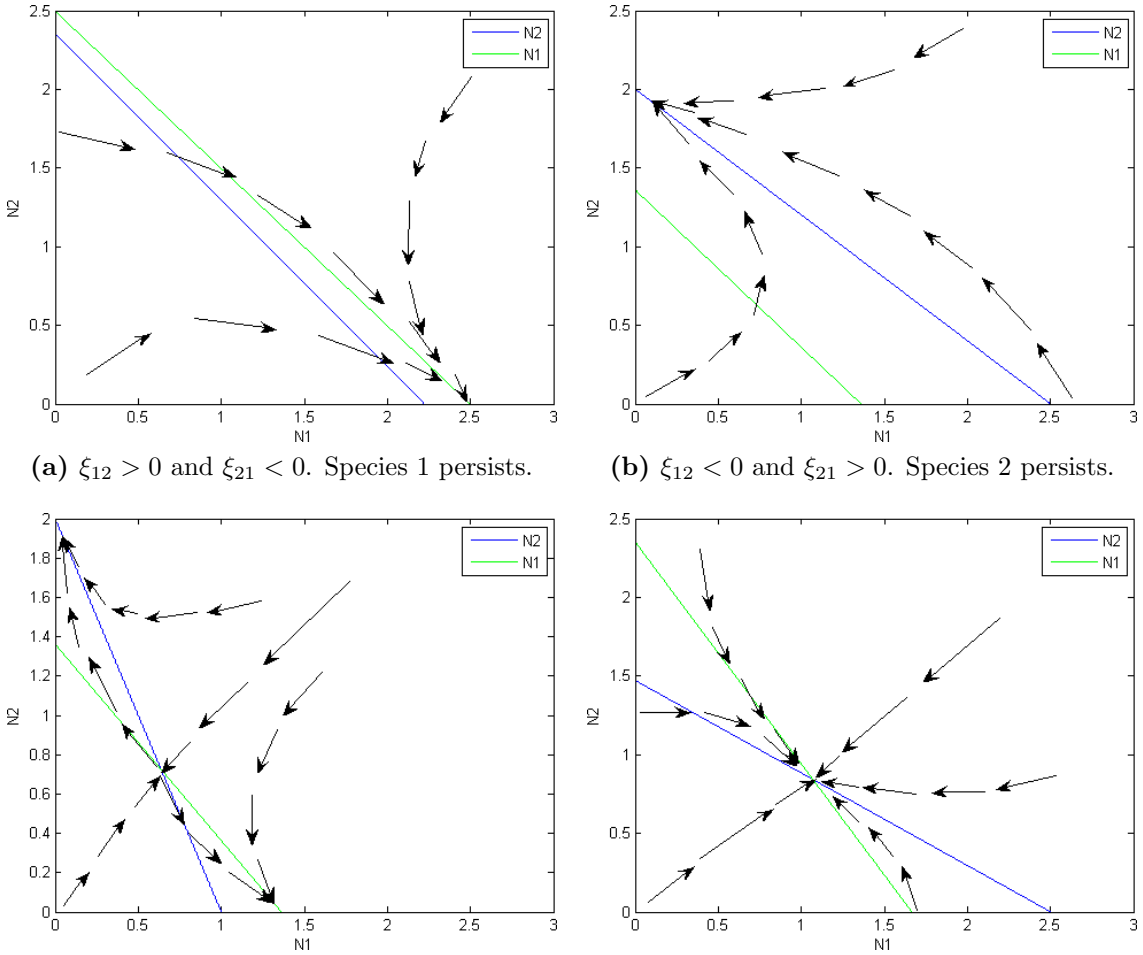
$$\begin{aligned}
\gamma &= -r\left(1 - \frac{N_1^e}{K_{31}} - \frac{N_2^e}{K_{32}}\right) = -\left[\frac{1}{N_3}\frac{dN_3}{dt}\right]_{E_{C,12}} \\
\alpha_1 &= \frac{r}{K}(N_1^e + N_2^e) = \left[-\mathrm{tr}(\tilde{\mathcal{A}}_1)\right]_{E_{C,12}} \\
\alpha_2 &= r^2N_1^eN_2^e\left(\frac{\xi_{12}}{K} + \frac{\xi_{21}}{K_{12}}\right) = \left[\det(\tilde{\mathcal{A}}_1)\right]_{E_{C,12}}
\end{aligned}$$

To simplify notation, define

$$\kappa_{ij} = \left[\frac{1}{N_l}\frac{dN_l}{dt}\right]_{E_{C,ij}}$$

to be the per capita growth rate of species l at the coexistence disease-free equilibrium of species i and j.

Then in order for the eigenvalue $-\gamma = \kappa_{12}$ to be negative, we require the growth rate of species 3 to be negative at the disease-free equilibrium of species 1 and 2, indicating that it cannot invade the system.



(a) $\xi_{12} > 0$ and $\xi_{21} < 0$. Species 1 persists.

(b) $\xi_{12} < 0$ and $\xi_{21} > 0$. Species 2 persists.

(c) $\xi_{12} < 0$ and $\xi_{21} < 0$. Coexistence is feasible but unstable.

(d) $\xi_{12} > 0$ and $\xi_{21} > 0$. Coexistence is feasible and stable.

Figure 3: Phase planes showing possible competitive interactions between two species in the absence of disease. Here, species 1 and 2 are shown.

By the trace-determinant theorem, we need $\alpha_1 > 0$ and $\alpha_2 > 0$ for stability. It is obvious that α_1 is positive. Since $\xi_{12}/\xi_{21} > 0$ for feasibility and stability of $E_{C,12}$ and $E_{I,12}$ (cf. Section 3.4), the only way to have $\alpha_2 > 0$ is to have $\xi_{12} > 0$ and $\xi_{21} > 0$. This indicates that species 1 and 2 are affected more by intraspecific crowding than by interspecific competition.

These results can be extended to the other 2-host equilibria. Thus conditions for stability of the disease-free equilibrium are $\mathcal{R}_{0,ij} < 1$, $\xi_{ij} > 0$, $\xi_{ji} > 0$, and $\kappa_{ij} < 0$.

4.3 Coexistence Equilibria

Since (3.5) holds for any equilibrium with nonzero populations of species i, the Jacobian of the system (2.1) computed at a coexistence equilibrium $E_e = E_C, E_I$ can be

written as the block matrix

$$\mathcal{J}(E_e) = \begin{bmatrix} \mathcal{A}(E_e) & \mathcal{B}(E_e) \\ \mathcal{C}(E_e) & \mathcal{D}(E_e) \end{bmatrix}, \tag{4.2}$$

where the 3×3 sub-blocks are

$$\begin{aligned} \mathcal{A}(E_e) &= (A_{ij}) - \operatorname{diag}(b + \mathcal{I}(E_e)), \\ \mathcal{B}(E_e) &= (A_{ij}) - (\beta S_i^e), \\ \mathcal{C}(E_e) &= \operatorname{diag}(\mathcal{I}(E_e)), \\ \mathcal{D}(E_e) &= (\beta S_i^e) - \operatorname{diag}(b), \end{aligned}$$

with the definitions

$$\begin{aligned} \mathcal{I}(E_e) &= \beta(I_1^e + I_2^e + I_3^e), \\ A_{ij} &= -\frac{rN_i^e}{K_{ij}} + b\delta_{ij}, \end{aligned}$$

where δ_{ij} is the Kronecker delta function

$$\delta_{ij} = \begin{cases} 1 & i = j \\ 0 & i \neq j \end{cases}.$$

A routine computation shows the characteristic polynomial of $\mathcal{J}(E_e)$ to be

$$P(x) = (x+\eta)^2(x+\epsilon)\left(x^3 + \delta_1 x^2 + \delta_2 x + \delta_3\right), \tag{4.3}$$

where

$$\eta = b + \beta(I_1^e + I_2^e + I_3^e), \tag{4.4a}$$

$$\epsilon = b + \beta(I_1^e + I_2^e + I_3^e) - \beta(S_1^e + S_2^e + S_3^e), \tag{4.4b}$$

$$\delta_1 = \frac{r}{K}(N_1^e + N_2^e + N_3^e), \tag{4.4c}$$

$$\delta_2 = r^2\left[\frac{1}{K^2}\left(N_1^eN_2^e + N_1^eN_3^e + N_2^eN_3^e\right) - \frac{N_1^eN_2^e}{K_{12}K_{21}} - \frac{N_1^eN_3^e}{K_{13}K_{31}} - \frac{N_2^eN_3^e}{K_{23}K_{32}}\right], \tag{4.4d}$$

$$\delta_3 = r^3\Delta N_1^eN_2^eN_3^e. \tag{4.4e}$$

The eigenvalue $-\eta$ is clearly negative. In the infected case, it follows directly from Theorems 2 and 3 that the eigenvalue $-\epsilon$ is $b(1-\mathcal{R}_{0,3})$, so stability requires $\mathcal{R}_{0,3} > 1$. In the disease-free case, $-\epsilon = b(\mathcal{R}_{0,3} - 1)$, so stability requires $\mathcal{R}_{0,3} < 1$.

We then consider $x^3 + \delta_1 x^2 + \delta_2 x + \delta_3$. This is the characteristic polynomial of the matrix

$$\mathcal{Q} = \begin{bmatrix} -\frac{rN_1^e}{K} & -\frac{rN_1^e}{K_{12}} & -\frac{rN_1^e}{K_{13}} \\ -\frac{rN_2^e}{K_{21}} & -\frac{rN_2^e}{K} & -\frac{rN_2^e}{K_{23}} \\ -\frac{rN_3^e}{K_{31}} & -\frac{rN_3^e}{K_{32}} & -\frac{rN_3^e}{K} \end{bmatrix} = -r\operatorname{diag}(N_1^e, N_2^e, N_3^e)M.$$

This matrix has an eigenvalue $\lambda_1 = -r$ with corresponding eigenvector $\begin{bmatrix} N_1^e & N_2^e & N_3^e \end{bmatrix}^T$, since

$$-r \begin{bmatrix} N_1^e & 0 & 0 \\ 0 & N_2^e & 0 \\ 0 & 0 & N_3^e \end{bmatrix} \begin{bmatrix} \frac{1}{K} & \frac{1}{K_{12}} & \frac{1}{K_{13}} \\ \frac{1}{K_{21}} & \frac{1}{K} & \frac{1}{K_{23}} \\ \frac{1}{K_{31}} & \frac{1}{K_{32}} & \frac{1}{K} \end{bmatrix} \begin{bmatrix} N_1^e \\ N_2^e \\ N_3^e \end{bmatrix} = -r \begin{bmatrix} N_1^e & 0 & 0 \\ 0 & N_2^e & 0 \\ 0 & 0 & N_3^e \end{bmatrix} \begin{bmatrix} 1 \\ 1 \\ 1 \end{bmatrix} = -r \begin{bmatrix} N_1^e \\ N_2^e \\ N_3^e \end{bmatrix}.$$

This follows as a consequence of Lemma 1 and (3.5).

To find the remaining eigenvalues λ_2 and λ_3, note that $\lambda_1\lambda_2\lambda_3 = -r^3\Delta N_1^e N_2^e N_3^e$ and $\lambda_1 + \lambda_2 + \lambda_3 = -\frac{r}{K}(N_1^e + N_2^e + N_3^e)$. Thus we have

$$\lambda_2\lambda_3 = r^2\Delta N_1^e N_2^e N_3^e$$

and

$$\lambda_2 + \lambda_3 = -\frac{r}{K}(N_1^e + N_2^e + N_3^e - K),$$

giving

$$\lambda_2, \lambda_3 = \frac{1}{2}\left[\frac{r}{K}(K - N_1^e - N_2^e - N_3^e) \pm \sqrt{\frac{r^2}{K^2}\left(N_1^e + N_2^e + N_3^e - K\right)^2 - 4r^2\Delta N_1^e N_2^e N_3^e}\right]. \tag{4.5}$$

From Lemma 1 and (3.7), note that the discriminant of (4.5) is

$$\begin{aligned} &\frac{r^2}{\Delta^2}\left[\left(\frac{\Delta_1 + \Delta_2 + \Delta_3}{K} - \Delta\right)^2 - 4\Delta_1\Delta_2\Delta_3\right] \\ &= \frac{r^2}{\Delta^2}\big[\xi_{13}^2\xi_{32}^2\xi_{21}^2 + 2\xi_{13}\xi_{32}\xi_{21}\xi_{12}\xi_{23}\xi_{31} + \xi_{12}^2\xi_{23}^2\xi_{31}^2 \\ &\quad - 4\left(\xi_{12}^2\xi_{23}^2\xi_{31}^2 + \xi_{13}^2\xi_{32}^2\xi_{21}^2 + G(\xi_{12}, \xi_{13}, \xi_{21}, \xi_{23}, \xi_{31}, \xi_{32})\right)\big], \end{aligned}$$

where G is a homogenous polynomial of ξ_{ij}. Making the assumption (A5) guarantees $G > 0$. Since we assume $K > 1$, the discriminant is negative, so the stability of the equilibrium depends on

$$\mathrm{Re}(\lambda_2) = \mathrm{Re}(\lambda_3) = -\frac{r}{2\Delta}(\xi_{13}\xi_{32}\xi_{21} + \xi_{12}\xi_{23}\xi_{31}).$$

Thus, under these assumptions, we have local asymptotic stability for $\xi_{13}\xi_{32}\xi_{21} + \xi_{12}\xi_{23}\xi_{31} > 0$ and a saddle point with one-dimensional stable manifold if $\xi_{13}\xi_{32}\xi_{21} + \xi_{12}\xi_{23}\xi_{31} < 0$. A Hopf bifurcation occurs at $\xi_{13}\xi_{32}\xi_{21} + \xi_{12}\xi_{23}\xi_{31} = 0$.

5 Summary of Results

A review of the biological quantities used in our analysis is given in Table 1. Our results from the feasibility analysis in Section 3 and local stability analysis in Section 4 are then summarized in Table 2.

Parameter	Definition
ξ_{ij}	The relative strength of intraspecific crowding versus interspecific competition of species j on i.
$\mathcal{R}_{0,1}$	The basic reproduction number of the disease affecting one species in isolation.
$\mathcal{R}_{0,ij}$	The basic reproduction number of the disease affecting two species i and j in coexistence
$\mathcal{R}_{0,3}$	The basic reproduction number of the disease affecting all three species.
κ_{ij}	The per capita growth rate of species l at the disease-free equilibrium of species i and j

Table 1: Quantities used to define conditions for equilibrium feasibility and stability.

Equilibrium	Feasibility	Stability
Trivial	always	never
1-host DFE	always	$\xi_{mi}, \xi_{ni} < 0$, $\mathcal{R}_{0,1} < 1$
1-host Infected	$\mathcal{R}_{0,1} > 1$	$\xi_{mi}, \xi_{ni} < 0$, $\mathcal{R}_{0,1} > 1$
2-host DFE	$\xi_{ij}/\xi_{ji} > 0$	$\xi_{ij}, \xi_{ji} > 0$, $\kappa_{ij} < 0$, $\mathcal{R}_{0,ij} < 1$
2-host Infected	$\xi_{ij}/\xi_{ji} > 0$, $\mathcal{R}_{0,ij} > 1$	$\xi_{ij}, \xi_{ji} > 0$, $\kappa_{ij} < 0$, $\mathcal{R}_{0,ij} > 1$
3-host DFE	$\xi_{12}, \xi_{23}, \xi_{31} > 0$, $\xi_{13}, \xi_{32}, \xi_{21} < 0$	$\xi_{12}\xi_{23}\xi_{31} + \xi_{13}\xi_{32}\xi_{21} > 0$, $\mathcal{R}_{0,3} < 1$
3-host Infected	$\xi_{12}, \xi_{23}, \xi_{31} > 0$, $\xi_{13}, \xi_{32}, \xi_{21} < 0$, $\mathcal{R}_{0,3} > 1$	$\xi_{12}\xi_{23}\xi_{31} + \xi_{13}\xi_{32}\xi_{21} > 0$, $\mathcal{R}_{0,3} > 1$

Table 2: Summary of conditions for feasibility and stability. In the one-host case, m and n refer to the absent species and i to the persisting species. In the two-host case, i and j refer to the persisting species and k to the absent species.

We conclude this section with a further interpretation of the Δ_i, which we can relate to the stability of the 2-species coexistence disease-free equilibrium that does not include species i. Consider the per capita growth rate of species 3 at the equilibrium $E_{C,12}$. From (2.1), this is

$$\kappa_{12} = \frac{1}{N_3}\frac{dN_3}{dt}\bigg|_{E_{C,12}} = r\left(1 - \frac{N_1^{C,12}}{K_{31}} - \frac{N_2^{C,12}}{K_{32}}\right),$$

which, substituting in the equilibrium values given in (3.3a), can be shown to be

$$\kappa_{12} = r\Delta_3 \frac{N_1^{C,12}}{\xi_{12}} = r\Delta_3 \frac{K}{\xi_{12} + \xi_{21} - K\xi_{12}\xi_{21}}.$$

Therefore,

$$\Delta_3 = \frac{\kappa_{12}}{rK}\left(\xi_{12} + \xi_{21} - K\xi_{12}\xi_{21}\right).$$

In Section 4.2 we proved that stability of $E_{C,12}$ requires $\xi_{12}, \xi_{21} > 0$. Since

$$N_1^{C,12} = \frac{K\xi_{12}}{\xi_{12} + \xi_{21} - K\xi_{12}\xi_{21}}, \quad N_2^{C,12} = \frac{K\xi_{21}}{\xi_{12} + \xi_{21} - K\xi_{12}\xi_{21}},$$

and $E_{C,12}$ is assumed feasible, then $\xi_{12} + \xi_{21} - K\xi_{12}\xi_{21}$ has the same sign as ξ_{12} and ξ_{21}. Note that the denominator can be rewritten as

$$K\left(\frac{1}{K^2} - \frac{1}{K_{12}K_{21}}\right),$$

so it is a measure of the strength of crowding versus the strength of competition in species 1 and 2.

Therefore, Δ_3 contains information about the competitive aspects of stability of this equilibrium (there is no dependence on $\mathcal{R}_{0,12}$ so disease dynamics are not included). $\Delta_3 < 0$ implies either stability or instability due to both the condition on interspecific competition between species 1 and 2 and the condition on the per capita growth rate of species 3 being unsatisfied. $\Delta_3 > 0$ implies exactly one of these conditions is unsatisfied, resulting in an unstable equilibrium. A similar interpretation can be made for Δ_1 and Δ_2.

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References

[1] V. A. Bokil and C. A. Manore. Coexistence of competing species with a directly transmitted pathogen. Technical Report ORST-MATH 10-05, Oregon State University, `http://hdl.handle.net/1957/18639`, 2010.

[2] C.-W. Chi, S.-B. Hsu, and L.-I. Wu. On the asymmetric May-Leonard model of three competing species. *SIAM J. Appl. Math.*, 58(1):211–226, 1998.

[3] L. Gilbert, R. Norman, K. M. Laurenson, H. W. Reid, and P. J. Hudson. Disease persistence and apparent competition in a three-host community: an empirical and analytical study of large-scale, wild populations. *J. Anim. Ecol.*, 70(6):1053–1061, 2001.

[4] M. Hatcher, J. Dick, and A. Dunn. How parasites affect interactions between competitors and predators. *Ecol. Lett.*, 9(11):1253–1271, 2006.

[5] R. May and W. Leonard. Nonlinear aspects of competition between three species. *SIAM J. Appl. Math.*, 29(2):243–253, 1975.

[6] H. McCallum, N. Barlow, and J. Hone. How should pathogen transmission be modelled? *Trends Ecol. Evol.*, 16(6):295–300, June 2001.

[7] S. M. Moore, C. A. Manore, V. A. Bokil, E. T. Borer, and P. R. Hosseini. Spatiotemporal model of barley and cereal yellow dwarf virus transmission dynamics with seasonality and plant competition. *B. Math. Biol.*, pages 1–24, 2011.

[8] P. Nummi. Alien species in Finland. Technical report, Ministry of the Environment, 2000. Case studies by Arto Kurtto, Jyrki Tomminen, Erkki Leppkoski, and Petri Nummi.

[9] S. P. Rushton, P. W. W. Lurz, J. Gurnell, P. Nettleton, C. Bruemmer, M. D. F. Shirley, and A. W. Sainsbury. Disease threats posed by alien species: the role of a poxvirus in the decline of the native red squirrel in Britain. *Epidemiol. Infect.*, 134(3):521–533, Oct. 2006.

[10] P. Schuster, K. Sigmund, and R. Wolff. On ω-limits for competition between three species. *SIAM J. Appl. Math.*, 37(1):49–54, 1979.

[11] D. M. Tompkins, A. R. White, and M. Boots. Ecological replacement of native red squirrels by invasive greys driven by disease. *Ecol. Lett.*, 6(3):189–196, 2003.

[12] P. Van den Driessche and J. Watmough. Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.*, 180(1):29–48, 2002.