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MATHEMATICAL MODELING OF CITRUS GROVES INFECTED BY HUANGLONGBING

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Abstract. Huanglongbing (citrus greening) is a bacterial disease that is significantly impacting the citrus industry in Florida and poses a risk to the remaining citrus-producing regions of the United States. A mathematical model of a grove infected by citrus greening is developed. An equilibrium stability analysis is presented. The basic reproductive number and its relation to the persistence of the disease is discussed. A numerical study is performed to illustrate the theoretical findings.

1. **Introduction.** *Huanglongbing* (HLB) is a vector-transmitted bacterial infection that is currently affecting all types of citrus in the state of Florida. In Chinese, the name means "yellow dragon disease" [11], but in the United States it is frequently referred to as *citrus greening* disease. Symptoms of the disease include stunted growth and poor flowering of citrus trees as well as blotchy mottling and yellowing of their leaves. In addition, the fruit that is produced by infected trees is misshapen and smaller than normal, with a green tint and a bitter taste [11], making the fruit inedible. The disease has severely affected the citrus industry in Florida, the nation's largest citrus producer and the second largest producer of orange juice in the world. The presence of HLB has also been detected in other southeastern states including Texas [15], and all citrus producing regions of the United States are considered to be at risk. Because of its impact on many sectors of the Florida economy and the implications for the citrus industry nationwide, citrus greening has become an important issue to study.

HLB was first discovered in China in the late 1800s [8]. Different strains of the disease have also created problems in the past in both Africa, around 1930, and in Brazil, beginning in 2004 [8]. It is believed that the vectors that transmit the disease arrived in southern Florida in 1998 [11], and citrus greening began spreading north throughout the state in 2000 [9]. However, it was not until 2005 that symptomatic trees were first detected [8]. Since then, the disease has been spotted in almost every county as far north as Alachua. The disease has even affected some citrus in the Florida panhandle, although so far only at large discount stores that handle and ship citrus trees [11].

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An insect known as the Asian citrus psyllid, *Diaphorina citri*, carries the organism that causes citrus greening, *Candidatus Liberibacter asiaticus* (Las) [13, 8]. The psyllids have a life cycle that lasts 45 days on average, including five nymphal stages that together last 15 days. The bacteria are carried in the saliva of a psyllid; when an infected psyllid feeds on the leaves of a tree, it passes the disease to the phloem within the veins of that tree. Similarly, a healthy psyllid can acquire the infection by feeding on an infected tree. These transmissions are most likely to occur during the flush, which happens in the state of Florida in the spring and fall [11]. Nymphs are not able to transmit the disease while they are in the first three life stages, but after this point they can pass along the disease in as little as one day after becoming infected [18]. It is also believed that the disease can be spread among the psyllid population transovarially; a single psyllid will lay around 800 eggs during its lifetime [11].

The psyllids are the primary source of the spread of the disease. Although psyllids are generally sedentary as nymphs [9], a mature psyllid can fly over a mile [11], and high winds can increase the distance a psyllid can travel [16]. Although large commercial groves generally have more than a few miles between them, there are many abandoned groves and residential citrus trees between them that facilitate the spread of the disease [8]. Ornamental trees, such as *Murraya paniculata* (orange jasmine) are very popular in the state of Florida and serve as another habitat from which the psyllids can spread out [11]. In addition, many discount stores ship both infected plants and fruit throughout the state, enabling the disease to proliferate quickly. For these reasons the disease has traveled about 30 miles a year in Florida but only 12 miles a year in Brazil [9]. Grafting together healthy and infected trees also contributes to the spread [11].

With all of these factors contributing to the advancement of citrus greening, it is very difficult to keep the disease under control. However, because of the importance of citrus crops to the state's economy, several attempts have been made to implement various control strategies. One option is to rogue (or remove) infected trees. This helps to prevent more psyllids from acquiring the disease. Unfortunately, because a tree may be infected for a couple years before any symptoms are detected, it is difficult to know which trees must be rogued [8]. Especially in commercial groves, farmers are reluctant to eliminate a healthy-looking tree that is still capable of producing good fruit. Once a tree is rogued, another can be replanted in its place. However, it is possible that the remnants of tree roots in the soil may serve as a reservoir for the disease, as it has been observed that recently planted trees are more susceptible, creating another setback to this control strategy [16]. Another method is to treat unhealthy trees with antibiotics. This can be very effective, but it is also very costly. It is also not a permanent fix; symptoms will return about a year after being treated [11]. With either control strategy, it is important to know which asymptomatic trees are actually infected with the disease. One way is to test plant tissue with the polymerase chain reaction technique (PCR). However, it is not feasible for every single tree in a grove to be tested this way. Also, the disease is not evenly distributed within a given tree, and thus the test may not always produce accurate results. Many farmers use scouts to visually inspect trees for psyllids, but it is generally difficult to see the miniscule insects that are only three to four millimeters in diameter [11].

There are a few control strategies that are more realistic and easier to implement, though less effective. These include spraying insecticides over entire groves as well

as eliminating grafting [11]. It is also important for citrus stock to be inspected and screened before it is transported or sold, and it is very beneficial for nurseries that grow citrus to be screened-in [8]. A quarantine that began June 17, 2010, is designed to prevent the transportation of citrus throughout the state of Florida [2].

Citrus greening has become very wide-spread and is difficult to eradicate. Because citrus is such an important part of Florida's economy, this disease is an issue of great concern. A recent study by the University of Florida's Institute of Food and Agricultural Sciences estimates that from 2006 to 2011 citrus greening has caused $3.63 billion in lost revenue and over 6,000 lost jobs in the state. The five-year production level for orange juice is estimated to be 1.7 billion gallons less than projected [12]. If the disease continues, eventually there will not be enough citrus produced for the juice plants to operate; many plants have already begun to import extra citrus from Brazil in order to keep up their production levels [16]. Unfortunately, many believe that the damage may be irreversible in the state of Florida. However, HLB has also been detected in Georgia, South Carolina, and Louisiana. In January 2012, the Texas Department of Agriculture and USDA confirmed the detection of the disease in a commercial grove in Texas [15]. Recently, HLB-infected trees and the asian citrus psyllids were found in both Arizona and California, and the psyllids alone were discovered in Hawaii, Mississippi and Alabama.

It is clear that research on the progression of the disease in Florida could be instrumental in preventing the same outbreak in other states.

In this paper, we develop a model for the spread of citrus greening disease within a single grove of citrus trees. The model represents healthy and infected individuals in both the tree and vector populations, and includes the control method of roguing and replanting. The unique aspect of this model is that it allows for a newly planted tree to become infected immediately due to the possibility that the soil may be a reservoir for the disease; this feature has not been considered in other models with roguing and replanting. We will calculate the basic reproductive number, R_0, a threshold below which the disease can be eradicated but above which the disease will persist. Extinction and persistence results will be proved, and numerical simulations will suggest other results. Finally, we will consider a modification to our model that can result in bistability given the appropriate parameters.

2. **A mathematical model and main results.**

2.1. **Model formulation.**

We subdivide the citrus tree population into four stages. S denotes susceptible trees and R represents dead trees. Due to the observed delay in the appearance of symptoms of citrus greening we split the infected trees I into an asymptomatic (latent) stage, I_1, and a symptomatic stage, I_2. For the psyllid vector population we let V_- and V_+ represent the uninfected/susceptible and infected psyllids, respectively. We assume that the grove is subject to a rogue and replant disease management strategy. In the model, symptomatic and dead trees are rogued at a rate ρ and the corresponding spots are replanted with new trees. We assume that a proportion $f \in [0, 1]$ of the newly planted trees will be healthy and a proportion $1 - f$ will become infected immediately. A schematic for the model is provided in Figure 1.

We assume that infected trees progress from the latent state to the symptomatic state at the rate γ. The parameter α represents the disease-associated mortality of the symptomatic trees; the natural death rate of the trees is neglected. The mortality rate of the psyllids is given by μ which is assumed to be the same for

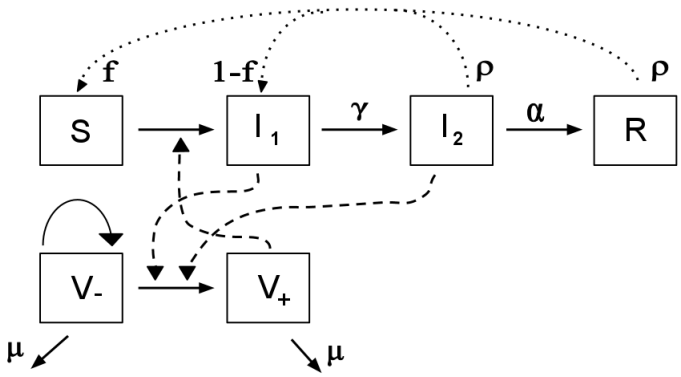


FIGURE 1. Flow diagram for the grove-scale citrus greening model (1)-(6)

uninfected and infected psyllids. With the above assumptions, the model takes the following form:

$$\dot{I}_1 = \beta \frac{S}{N}\frac{V_+}{V} - \gamma I_1 + \rho(1-f)(I_2+R), \tag{1}$$

$$\dot{I}_2 = \gamma I_1 - (\alpha+\rho) I_2, \tag{2}$$

$$\dot{R} = \alpha I_2 - \rho R, \tag{3}$$

$$\dot{V}_+ = \beta \frac{V_-}{V}\frac{I_1+I_2}{N} - \mu V_+, \tag{4}$$

$$\dot{S} = b\left(1-\frac{N}{N_0}\right) - \beta\frac{S}{N}\frac{V_+}{V} + \rho f(I_2+R), \tag{5}$$

$$\dot{V}_- = rV\left(1-\frac{V}{K}\right) - \beta\frac{V_-}{V}\frac{I_1+I_2}{N} - \mu V_-. \tag{6}$$

The total number of trees is given by $N = S + I_1 + I_2 + R$ while N_0 represents the number of spaces in the grove. We will assume throughout that $N \leq N_0$. The trees are assumed to be replanted at a rate that is proportional, with constant b, to the number of empty spaces in the grove. The total psyllid population is represented by $V = V_- + V_+$ with logistic growth rate r and carrying capacity K. We assume that there is a fixed number of contacts per unit time between trees and psyllids. The biting rate β, given as units/time, accounts for the proportion of bites that result in successful transmission. For example, the infection rate of trees is therefore given by the product of the biting rate and the probability that a bite involves a healthy tree and an infected psyllid.

2.2. **Equilibria.** We begin our analysis of the model by making the following immediate observations. First, due to the specific form of equations (1)-(6), the model is well-posed; that is, all positive solutions are well-defined and remain positive in forward time. Second, adding equations (1)-(3) and (5), we find that the total tree population is governed by

$$\dot{N} = b\left(1-\frac{N}{N_0}\right).$$

Similarly, when we add equations (4) and (6), the total vector population satisfies

$$\dot{V} = rV\left(1 - \frac{V}{K}\right) - \mu V.$$

Consequently, all positive solutions of (1)-(6) are bounded and can be extended to all $t \in (0, +\infty)$. Additionally, $N(t) \to N_0$ and $V(t) \to V^* = (1 - \frac{\mu}{r})K$ (here, we assume that $\mu < r$ so that $V^* > 0$) exponentially fast as $t \to +\infty$.

Now, we look for steady states $(I_1^*, I_2^*, R^*, V_+^*, S^*, V_-^*)$ of system (1)-(6). Clearly, $N = N_0$ and $V = V^*$ at equilibrium. Trivially, $x_0 = (0, 0, 0, 0, N_0, V^*)$ is an equilibrium which we designate as the disease-free equilibrium (DFE) . To find endemic equilibria, we set (1)-(6) equal to zero and obtain

$$I_2 = \frac{\gamma}{\alpha + \rho} I_1, \quad R = \frac{\alpha}{\rho} I_2, \quad V^+ = \frac{\beta V^*(I_1 + I_2)}{\mu N_0 V^* + \beta(I_1 + I_2)}.$$

Thus we can express each infected state in terms of I_1. Then if $I_1 = 0$ we see that $I_2 = R = V_+ = 0$ and we are in the case of the disease-free equilibrium. Hence there exists an endemic equilibrium if and only if $I_1 > 0$. If $I_1 \neq 0$, (5) yields

$$S = \frac{N_0 f \gamma}{\beta} I_1 + \frac{N_0^2 f \mu V^*}{\beta^2(\frac{1}{\gamma} + \frac{1}{\alpha+\rho})}.$$

Then recalling that $N_0 = S + I_1 + I_2 + R$ we easily compute

$$N_0 = \frac{N_0 f \gamma}{\beta} I_1 + \frac{N_0^2 f \mu V^*}{\beta^2(\frac{1}{\gamma} + \frac{1}{\alpha+\rho})} + \left(1 + \frac{\gamma}{\rho}\right) I_1.$$

Therefore the condition for existence of a positive (endemic) equilibrium is:

$$I_1 > 0 \quad \Leftrightarrow \quad \frac{N_0^2 f \mu V^*}{\beta^2(\frac{1}{\gamma} + \frac{1}{\alpha+\rho})} < N_0 \quad \Leftrightarrow \quad \frac{\beta^2(\frac{1}{\gamma} + \frac{1}{\alpha+\rho})}{N_0 V^* f \mu} > 1. \tag{7}$$

The last inequality is closely related to the basic reproductive number R_0 which we discuss in the following section.

2.3. **The basic reproductive number R_0.** The basic reproductive number R_0 represents the average number of secondary infections that result from the introduction of a single infected agent (a tree or a vector) into a susceptible population. There are various approaches for calculating R_0 leading to some ambiguity in its definition. For instance, van den Dreissche and Watmough define R_0 to be the spectral radius of the next generation matrix, FV^{-1} [17]. In Part 1 of the Appendix we calculate FV^{-1} for our system (1)-(6) and find

$$R_0 = \frac{1 - f}{2} + \sqrt{\frac{(1 - f)^2}{4} + \frac{\beta^2}{\mu V^* N_0}\left(\frac{1}{\gamma} + \frac{1}{\alpha + \rho}\right)}.$$

We prove below in Theorem 2.1 the existence of an equivalent threshold condition involving the quantity

$$T_0 = \frac{\beta^2}{\mu V^* N_0}\left(\frac{1}{\gamma} + \frac{1}{\alpha + \rho}\right) + 1 - f.$$

The quantity T_0 provides the following biological interpretation. Suppose that a single infected tree in stage I_1 is introduced into a completely susceptible grove.

The average number of secondary infections resulting from psyllid contact during the I_1 and I_2 stages of the tree are, respectively,

$$\frac{\beta^2}{V^*N_0\mu\gamma} \text{ and } \frac{\beta^2}{V^*N_0\mu(\alpha+\rho)}.$$

The tree will necessarily be rogued in either the I_2 or R stage and will on average produce $1-f$ newly infected I_1 trees. Thus the expected number of secondary infections is exactly T_0. In Theorem 2.1, we prove the equivalence of T_0 and R_0 for the local stability of the DFE.

Theorem 2.1. *(i) $R_0 < 1$ if and only if $T_0 < 1$.*
(ii) $T_0 < 1$ if and only if all eigenvalues of the Jacobian matrix of system (1)-(6) evaluated at DFE have negative real parts.

Proof. It is shown in Part 1 of the Appendix that R_0 is the largest positive root of

$$p(\lambda) = \lambda^2 - (1-f)\lambda - \frac{\beta^2}{\mu V^* N_0}\left(\frac{1}{\gamma} + \frac{1}{\alpha+\rho}\right).$$

The leading coefficient of $p(\lambda)$ is positive and therefore $R_0 < 1$ if and only if

$$p(1) = f - \frac{\beta^2}{\mu V^* N_0}\left(\frac{1}{\gamma} + \frac{1}{\alpha+\rho}\right) > 0.$$

Then $p(1) > 0$ is clearly equivalent to $T_0 < 1$ and the first assertion follows.

To prove the second assertion, we compute the Jacobian matrix of system (1)-(6) at the DFE:

$$J(x_0) = \begin{bmatrix} -\gamma & \rho(1-f) & \rho(1-f) & \frac{\beta}{V^*} & 0 & 0 \\ \gamma & -(\alpha+\rho) & 0 & 0 & 0 & 0 \\ 0 & \alpha & -\rho & 0 & 0 & 0 \\ \frac{\beta}{N_0} & \frac{\beta}{N_0} & 0 & -\mu & 0 & 0 \\ -\frac{b}{N_0} & -\frac{b}{N_0}+\rho f & -\frac{b}{N_0}+\rho f & -\frac{\beta}{V^*} & -\frac{\beta}{N_0} & 0 \\ -\frac{\beta}{N_0} & -\frac{\beta}{N_0} & 0 & 2\mu - r & 0 & \mu - r \end{bmatrix}.$$

We see that $J(x_0)$ has a block triangular form and therefore $-\frac{\beta}{N_0}$ and $\mu - r$ are eigenvalues of $J(x_0)$. It follows from our assumption $\mu < r$ that these two eigenvalues are negative. We let $\tilde{J}(x_0)$ be the upper left 4×4 submatrix of $J(x_0)$. Then the remaining eigenvalues are determined by the characteristic equation of $\tilde{J}(x_0)$:

$$p(\lambda) = (\lambda+\mu)(\lambda+\alpha+\rho)\Big((\lambda+\rho)(\lambda+\gamma) - \gamma\rho(1-f)\Big) - \frac{\beta^2}{V^*N_0}(\lambda+\rho)(\lambda+\alpha+\rho+\gamma).$$

Any root λ of $p(\lambda)$ with $\mathrm{Re}(\lambda) \geq 0$ is also a root of $q(\lambda)$ defined by

$$\begin{aligned} q(\lambda) &= \frac{p(\lambda)}{(\lambda+\mu)(\lambda+\rho)(\lambda+\gamma)(\lambda+\alpha+\rho)} \\ &= 1 - \frac{\beta^2(\lambda+\alpha+\rho+\gamma)}{V^*N_0(\lambda+\mu)(\lambda+\gamma)(\lambda+\alpha+\rho)} - \frac{\gamma\rho(1-f)}{(\lambda+\rho)(\lambda+\gamma)}. \end{aligned}$$

We observe that $q(\lambda)$ is monotone increasing in λ when $\lambda > 0$. Therefore since the leading coefficient of $p(\lambda)$ is positive we know that $p(\lambda)$ has no positive real roots if and only if $q(0) > 0$. Therefore all real eigenvalues of $J(x_0)$ are negative if and only if $q(0) > 0$, which is equivalent to

$$\frac{\beta^2}{V^*N_0\mu}\left(\frac{1}{\gamma} + \frac{1}{\alpha+\rho}\right) + 1 - f < 1;$$

that is, $T_0 < 1$. To prove that all complex eigenvalues of $J(x_0)$ have negative real parts we define $G(\lambda) = 1 - q(\lambda)$ and suppose $p(\lambda) = 0$ with $\mathrm{Re}(\lambda) \geq 0$ and $\mathrm{Im}(\lambda) \neq 0$. Then $G(\lambda) = 1$ where

$$G(\lambda) = \frac{\beta^2}{V^* N_0(\lambda+\mu)(\lambda+\gamma)} + \frac{\beta^2\gamma}{V^* N_0(\lambda+\mu)(\lambda+\gamma)(\lambda+\alpha+\rho)} + \frac{\gamma\rho(1-f)}{(\lambda+\rho)(\lambda+\gamma)}.$$

We claim $|G(\lambda)| < G(\mathrm{Re}(\lambda))$. Indeed,

$$\begin{aligned}
&|G(\lambda)| \leq \\
&\frac{\beta^2}{V^* N_0|\lambda+\mu||\lambda+\gamma|} + \frac{\beta^2\gamma}{V^* N_0|\lambda+\mu||\lambda+\gamma||\lambda+\alpha+\rho|} + \frac{\gamma\rho(1-f)}{|\lambda+\rho||\lambda+\gamma|} \\
&< \frac{\beta^2}{V^* N_0(\mathrm{Re}(\lambda)+\mu)(\mathrm{Re}(\lambda)+\gamma)} + \frac{\gamma\rho(1-f)}{(\mathrm{Re}(\lambda)+\rho)(\mathrm{Re}(\lambda)+\gamma)} \\
&+ \frac{\beta^2\gamma}{V^* N_0(\mathrm{Re}(\lambda)+\mu)(\mathrm{Re}(\lambda)+\gamma)(\mathrm{Re}(\lambda)+\alpha+\rho)} \\
&= G(\mathrm{Re}(\lambda)),
\end{aligned}$$

where the second inequality is strict since $\mathrm{Im}(\lambda) \neq 0$. Therefore we have $T_0 < 1 \Leftrightarrow q(0) > 0 \Leftrightarrow G(0) < 1$, which implies $G(\mathrm{Re}(\lambda)) < 1$ since G is decreasing in λ. Then $|G(\lambda)| < G(\mathrm{Re}(\lambda)) < 1$ gives a contradiction. □

2.4. **Persistence of the disease when $R_0 > 1$.** Theorem 2.1 states that if $R_0 < 1$ the DFE is locally asymptotically stable while $R_0 > 1$ indicates that the DFE is unstable. In Theorem 2.4 in Section 2.5, we prove a global stability result for the case $R_0 \leq 1$. We were not able to analytically establish the local stability of the endemic equilibrium in the general case but simulations suggest that it is stable whenever it exists. In Figure 2, we present a numerical simulation of system (1)-(6) with $f = 0.5$, $r = 1.5$, $b = N_0 = \alpha = \rho = \gamma = \mu = \beta = V^* = 1$ and initial condition $(I_1(0), I_2(0), R(0), V_+(0), S(0), V_-(0)) = (0.1, 0, 0, 0, 0.9, 1)$. With these hypothesized parameter values $R_0 = 1.5$ and the endemic equilibrium is $(I_1^*, I_2^*, R^*, V_+^*, S^*, V_-^*) = (0.267, 0.133, 0.133, 0.286, 0.467, 0.714)$. We see from the simulation that the solution converges to the endemic equilibrium.

In the case where $f = 0$ the system is more tractable. In the limiting case $N = N_0$ and $V = V^*$, the equations of the model are given by:

$$\dot{I}_1 = \beta\frac{S}{N_0}\frac{V_+}{V^*} - \gamma I_1 + \rho(I_2 + R), \tag{8}$$

$$\dot{I}_2 = \gamma I_1 - (\alpha+\rho)I_2, \tag{9}$$

$$\dot{R} = \alpha I_2 - \rho R, \tag{10}$$

$$\dot{V}_+ = \beta\frac{V_-}{V^*}\frac{I_1+I_2}{N_0} - \mu V_+, \tag{11}$$

$$\dot{S} = -\beta\frac{S}{N_0}\frac{V_+}{V^*}, \tag{12}$$

$$\dot{V}_- = V^*\mu - \beta\frac{V_-}{V^*}\frac{I_1+I_2}{N_0} - \mu V_-. \tag{13}$$

This system has the unique endemic equilibrium

$$(I_1^*, I_2^*, R^*, V_+^*, S^*, V_-^*) = \left(\frac{\rho N_0}{\gamma+\rho}, \frac{\gamma\rho N_0}{(\alpha+\rho)(\gamma+\rho)}, \frac{\alpha\gamma N_0}{(\alpha+\rho)(\gamma+\rho)}, V_+^*, 0, V_-^*\right)$$

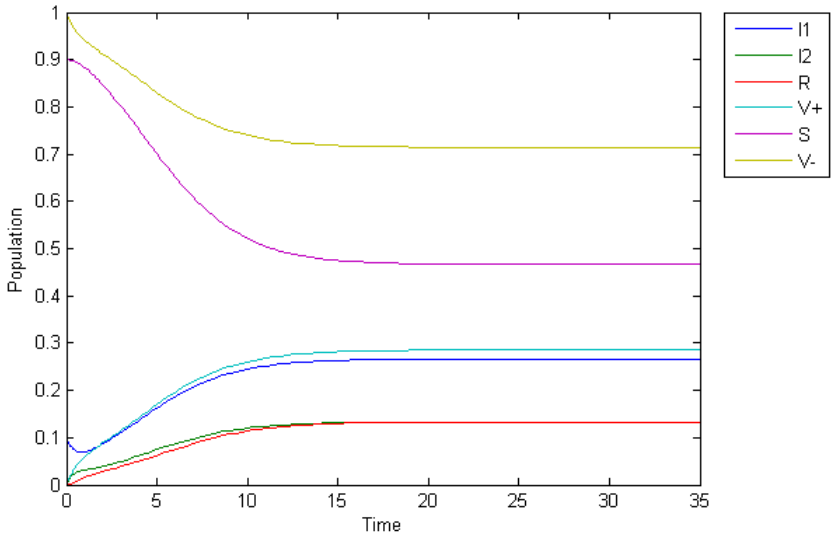


FIGURE 2. Simulation of system (1)-(6) using MATLAB ode45 solver with $f = 0.5$, $r = 1.5$, $b = N_0 = \alpha = \rho = \gamma = \mu = \beta = V^* = 1$, and initial condition $(I_1(0), I_2(0), R(0), V_+(0), S(0), V_-(0)) = (0.1, 0, 0, 0, 0.9, 1)$.

where V_+^* satisfies

$$V_+^* = \frac{\frac{\beta}{N_0}(I_1^* + I_2^*)}{\mu + \frac{\beta}{N_0 V^*}(I_1^* + I_2^*)} < V^*,$$

and $V_-^* = V^* - V_+^* > 0$. In this case we are able to prove the following theorem concerning global asymptotic stability of the endemic equilibrium.

Theorem 2.2. *All positive solutions of the system (8)-(13) with $S(0) < N_0$ converge to the endemic equilibrium $(I_1^*, I_2^*, R^*, V_+^*, S^*, V_-^*)$.*

Proof. First we observe that $S(t)$ is decreasing and bounded below by 0. So $S(t) \to S_\infty$ as $t \to \infty$ for some $0 \leq S_\infty < N_0$. Now let $M(t) = I_1(t) + I_2(t) + R(t)$. Since the total population is N_0 we have that $M(t) \to M_\infty$ as $t \to \infty$ for $M_\infty = N_0 - S_\infty > 0$. Hence $I_1(t) = M_\infty + h(t) - I_2 - R$ where $h(t) \to 0$ as $t \to \infty$. Then we consider the subsystem:

$$\dot{I}_2 = \gamma(M_\infty + h(t) - I_2 - R) - (\alpha + \rho)I_2, \tag{14}$$

$$\dot{R} = \alpha I_2 - \rho R. \tag{15}$$

Letting $\tilde{I}_2 = \frac{\gamma\rho}{(\alpha+\rho)(\gamma+\rho)} M_\infty$ and $\tilde{R} = \frac{\alpha\gamma}{(\alpha+\rho)(\gamma+\rho)} M_\infty$, we observe that

$$0 = \gamma(M_\infty - \tilde{I}_2 - \tilde{R}) - (\alpha + \rho)\tilde{I}_2,$$

$$0 = \alpha\tilde{I}_2 - \rho\tilde{R}.$$

We then perform a shift by defining $i_2 = I_2 - \tilde{I}_2$ and $r = R - \tilde{R}$. Then (14)-(15) can be written as

$$\dot{x} = Ax + f(t)$$

where

$$x = \begin{bmatrix} i_2 \\ r \end{bmatrix}, \qquad A = \begin{bmatrix} -(\gamma+\alpha+\rho) & -\gamma \\ \alpha & -\rho \end{bmatrix}, \qquad f(t) = \begin{bmatrix} \gamma h(t) \\ 0 \end{bmatrix}.$$

Using the variation of parameters, we find that

$$x(t) = \int_0^t e^{(t-s)A} f(s)ds + e^{tA}x_0.$$

Note that the matrix A is Hurwitz since both eigenvalues have negative real parts, so $e^{tA} \to 0$ as $t \to \infty$. This also means that there exist $C > 0$, $\alpha > 0$ such that $|e^{(t-s)A}f(s)| \leq Ce^{-\alpha(t-s)}|f(s)|$ for all $t \geq s \geq 0$. So for any given $\epsilon > 0$, there exists $T > 0$ such that $Ce^{-\alpha t}\int_0^\tau e^{\alpha s}|f(s)|ds < \frac{\epsilon}{2}$ for all $t \geq T$. Also, there exists $\tau > 0$ such that $|f(t)| < \frac{\epsilon\alpha}{2C}$ for all $t \geq \tau$. So

$$\begin{aligned} \left|\int_0^t e^{(t-s)A} f(s)ds\right| &\leq \int_0^t Ce^{-\alpha(t-s)}|f(s)|ds \\ &= \int_0^\tau Ce^{-\alpha(t-s)}|f(s)|ds + \int_\tau^t Ce^{-\alpha(t-s)}|f(s)|ds \\ &\leq Ce^{-\alpha t}\int_0^\tau e^{\alpha s}|f(s)|ds + \frac{\epsilon\alpha}{2}\int_\tau^t e^{-\alpha(t-s)}ds \\ &< \frac{\epsilon}{2} + \frac{\epsilon}{2}(1 - e^{-\alpha(t-\tau)}) \text{ for all } t \geq T \\ &< \epsilon. \end{aligned}$$

Thus $x(t) \to 0$ as $t \to \infty$. Hence $I_2 \to \tilde{I}_2$ and $R \to \tilde{R}$ as $t \to \infty$. Therefore $I_1 \to \frac{\rho}{\gamma+\rho}M_\infty$ which we define as $\tilde{I}_1$. Now $\tilde{I}_1 + \tilde{I}_2 > 0$ and we have

$$I_1(t) + I_2(t) = \tilde{I}_1 + \tilde{I}_2 + g(t)$$

for some $g(t) \to 0$ as $t \to \infty$. We can then express (11) as

$$\dot{V}_+ = \frac{\beta}{N_0}(\tilde{I}_1 + \tilde{I}_2 + g(t)) - \left(\frac{\beta}{N_0 V^*}(\tilde{I}_1 + \tilde{I}_2 + g(t)) + \mu\right)V_+ = g_1(t) - g_2(t)V_+$$

where the functions $g_1(t)$ and $g_2(t)$ clearly approach the positive limits $\frac{\beta}{N_0}(\tilde{I}_1 + \tilde{I}_2)$ and $\frac{\beta}{N_0 V^*}(\tilde{I}_1 + \tilde{I}_2) + \mu$, respectively, as $t \to \infty$. It follows that V_+ has the limit

$$\tilde{V}_+ = \frac{\frac{\beta}{N_0}(\tilde{I}_1 + \tilde{I}_2)}{\mu + \frac{\beta}{N_0 V^*}(\tilde{I}_1 + \tilde{I}_2)}.$$

Hence $\tilde{V}_+ > 0$ and so there exists $\tau > 0$ and $\xi > 0$ such that for all $t > \tau$

$$\dot{S} \leq -\frac{\beta}{N_0 V^*}S\xi,$$

and therefore

$$S(t) \leq S(\tau)e^{\frac{-\beta\xi}{N_0 V^*}(t-\tau)},$$

where the right side converges to 0 as $t \to \infty$. Therefore $S_\infty = 0$ which implies that $\tilde{I}_1 = I_1^*$, $\tilde{I}_2 = I_2^*$, $\tilde{R} = R^*$, and $\tilde{V}_+ = V_+^*$. □

We now return to the general model and present a proof for strong uniform persistence of the disease in the case $R_0 > 1$. Taking $N = N_0$ and $V = V^*$ we consider the limiting system of the infected components:

$$\begin{aligned}
\dot{I}_1 &= \beta\left(1 - \frac{I_1 + I_2 + R}{N_0}\right)\frac{V_+}{V^*} - \gamma I_1 + \rho(1-f)(I_2 + R), && (16)\\
\dot{I}_2 &= \gamma I_1 - (\alpha + \rho)I_2, && (17)\\
\dot{R} &= \alpha I_2 - \rho R, && (18)\\
\dot{V}_+ &= \beta\left(1 - \frac{V_+}{V^*}\right)\frac{I_1 + I_2}{N_0} - \mu V_+, && (19)
\end{aligned}$$

with solutions restricted to the feasible region $\Sigma = \{(I_1, I_2, R, V^+) : 0 \le I_1 + I_2 + R \le N_0, 0 \le V_+ \le V^*\}$, a positively invariant subset of $\mathbb{R}^4_+$.

Theorem 2.3. *Suppose $R_0 > 1$. Then there exists $\epsilon_0 > 0$ such that $\liminf_{t\to\infty} d(x(t), \partial\mathbb{R}^4_+) > \epsilon_0$ for all positive solutions $x(t)$ of (16)-(19).*

Proof. It suffices to consider $0 < f \le 1$ because the result follows from Theorem 2.2 when $f = 0$. We first prove that there exists $\epsilon_1 > 0$ such that $\limsup_{t\to\infty} I_1(t) + I_2(t) + R(t) \ge \epsilon_1$ for all positive solutions of (16)-(19). Consider the matrix

$$A(\epsilon) = \begin{bmatrix} -\gamma & \rho(1-f) & \rho(1-f) & \frac{\beta}{V^*}(1 - \frac{\epsilon}{N_0}) \\ \gamma & -(\alpha+\rho) & 0 & 0 \\ 0 & \alpha & -\rho & 0 \\ \frac{\beta}{N_0}(1 - \frac{\beta\epsilon}{V^* N_0 \mu}) & \frac{\beta}{N_0}(1 - \frac{\beta\epsilon}{V^* N_0 \mu}) & 0 & -\mu \end{bmatrix}$$

for $\epsilon > 0$. Observe that as $\epsilon \to 0$, $A(\epsilon) \to \tilde{J}(x_0)$ where $\tilde{J}(x_0)$ is the upper left four by four submatrix contained in the Jacobian matrix of the system (1)-(6). Thus by continuity of eigenvalues, the eigenvalues of A converge to the eigenvalues of $\tilde{J}(x_0)$. Since $R_0 > 1$ by Theorem 2.1 there exists an eigenvalue of $\tilde{J}(x_0)$ that has positive real part. Therefore there exists $\epsilon_1 > 0$ such that $A(\epsilon_1)$ has an eigenvalue with positive real part.

By way of contradiction, suppose $X(t) = (I_1, I_2, R, V_+)(t)$ is a positive solution and $\limsup_{t\to\infty} I_1(t) + I_2(t) + R(t) < \epsilon_1$. We see from (19) that $\limsup_{t\to\infty} V_+(t) < \frac{\beta\epsilon_1}{V^* N_0 \mu}$. Thus there exists $\tau > 0$ such that for all $t > \tau$

$$\begin{aligned}
\dot{I}_1 &\ge \frac{\beta}{V^*}\left(1 - \frac{\epsilon_1}{N_0}\right)V_+ - \gamma I_1 + \rho(1-f)(I_2 + R),\\
\dot{I}_2 &\ge \gamma I_1 - (\alpha + \rho)I_2,\\
\dot{R} &\ge \alpha I_2 - \rho R,\\
\dot{V}_+ &\ge \frac{\beta}{N_0}\left(1 - \frac{\beta\epsilon_1}{N_0 \mu V^*}\right)(I_1 + I_2) - \mu V_+.
\end{aligned}$$

We define $s = t - \tau$ and let $Y(s) = (i_1, i_2, r, v_+)(s) \in \mathring{\mathbb{R}}^4_+$ be the solution of $\dot{Y} = A(\epsilon_1)Y$ with initial condition $Y(0) = X(\tau)$. Applying Kamke's Theorem (Theorem B.1 in [14]), we conclude that $X(s) \ge Y(s)$ for all $s \ge 0$.

Let λ be the eigenvalue of $A(\epsilon_1)$ with the largest real part. Since $A(\epsilon_1)$ is quasi-positive, by the Perron-Frobenius Theorem there exists a nonnegative eigenvector v of $A(\epsilon_1)$ with eigenvalue λ. Choose k sufficiently small so that $kv \le Y(0)$ entrywise. Then for $Z(s) = ke^{\lambda s}v$ we have $\dot{Z} = A(\epsilon_1)Z$ and $Z(0) \le Y(0)$, which implies

that $Z(s) \leq Y(s)$ for all $s \geq 0$. Then there exists a component $Z_i(s)$ such that $\lim_{s\to\infty} Z_i(s) = \infty$. Therefore $\lim_{s\to\infty} Y_i(s) = \infty$ implying that $\lim_{s\to\infty} X_i(s) = \infty$, a contradiction. Therefore $\limsup_{t\to\infty} I_1(t) + I_2(t) + R(t) \geq \epsilon_1$ for all positive solutions $x(t)$ of (16)-(19).

Now let M be the maximal invariant set in $\partial\mathbb{R}^4_+ \cap \Sigma$. Suppose $\tilde{x} \in M$. Let $x(t) = (I_1, I_2, R, V_+)(t)$ be the solution to (16)-(19) with $x(0) = \tilde{x}$. Suppose that $V_+(t_1) > 0$ for some $t_1 \geq 0$. Since $\tilde{x} \in \partial\mathbb{R}^4_+$ there exists an $i \in \{1, 2, 3\}$ such that $x_i(t_1) = 0$. Suppose $I_1(t) = 0$ for all $t \geq t_1$. Then

$$\dot{I}_2 + \dot{R} = -\rho(I_2 + R)$$

so there exists $t_2 > t_1$ such that $(I_2 + R)(t_2) < N_0$. Then $\dot{I}_1(t_2) > 0$ and continuity implies that there exists $t_3 > t_2$ such that $I_1(t_3) > 0$. Then if $I_2(t_3) = 0$ we would have $\dot{I}_2(t_3) = \gamma I_1(t_3) > 0$ so $I_2(t)$ becomes positive. Similarly, $R(t)$ eventually becomes positive. But then there exists $t_4 > t_3$ such that $x(t_4) \in \mathring{\mathbb{R}}^4_+$ which contradicts the invariance of M. So $V_+(t) = 0$ for all $t \geq 0$. Then $\dot{V}_+(t) = 0$ implies that $I_1(t) = I_2(t) = 0$ for all $t \geq 0$. If $f = 1$ we conclude that $M = \{(0, 0, R, 0) : 0 \leq R \leq N_0\}$. If $f < 1$ equation (16) implies further that $R(t) = 0$ for all $t \geq 0$ and hence M is the origin. We show next that M is a uniform repeller in either case.

Let $f < 1$ and $\epsilon < \frac{\epsilon_1}{3}$. Let $x(t)$ be a nonnegative nonzero solution of (16)-(19) with $||x(0)|| < \epsilon$. By the argument above if $x(0) \in \partial\mathbb{R}^4_+$ the solution eventually becomes positive so it suffices to consider positive solutions only. Then $\limsup_{t\to\infty} I_1(t)+I_2(t)+R(t) \geq \epsilon_1$ implies that there is a $T > 0$ such that $||x(T)|| > \epsilon$. By Theorem 1 of Fonda [5] there exists $\eta > 0$ such that $\liminf_{t\to\infty} d(x(t), 0) \geq \eta$ for all nonnegative nonzero solutions $x(t)$.

Now let $f = 1$. Suppose that $\limsup_{t\to\infty} V_+(t) \leq \epsilon_2$ for some positive solution $x(t)$ and $\epsilon_2 > 0$. Then there exists $\tau > 0$ such that for $t > \tau$ and $y = (I_1(t), I_2(t), R(t))^T$,

$$\dot{y} \leq \epsilon_2\omega + Ay$$

where

$$\omega = \left(\frac{\beta}{V^*}, 0, 0\right) \quad \text{and} \quad A = \begin{bmatrix} -\gamma & 0 & 0 \\ \gamma & -(\alpha+\rho) & 0 \\ 0 & \alpha & -\rho \end{bmatrix}.$$

Clearly the principal eigenvalue of A is negative; by the Perron-Frobenius Theorem there exists $\lambda > 0$ and a positive left eigenvector v such that $vA = -\lambda v$. Define $\Psi(y) = v \cdot y$. Then

$$\dot{\Psi}(y) = v \cdot \dot{y} \leq \epsilon_2(v \cdot \omega) + vAy = \epsilon_2(v \cdot \omega) - \lambda\Psi(x)$$

from which it follows that

$$\limsup_{t\to\infty} \Psi(y) \leq \frac{\epsilon_2(v \cdot \omega)}{\lambda}$$

and hence there exists $C > 0$ such that $\limsup_{t\to\infty} I_1(t) + I_2(t) + R(t) \leq C\epsilon_2$. If $\epsilon_2 < \frac{\epsilon_1}{C}$ we get a contradiction. Therefore $\limsup_{t\to\infty} V_+(t) \geq \frac{\epsilon_1}{C}$.

To show M is also a uniform repeller in this case, suppose $\epsilon < \frac{\epsilon_1}{2C}$ and $x(t)$ is a solution such that $x(0) \in \Sigma \setminus M$ and $d(x(0), M) < \epsilon$. If $x(0) \in \partial\mathbb{R}^4_+$ then there exists $i \in \{1, 2, 4\}$ such that $x_i(0) > 0$ and it is straightforward to show that $x(t) \in \mathring{\mathbb{R}}^4_+$ for some $t > 0$. So it suffices to consider positive solutions. Now $\limsup_{t\to\infty} V_+(t) \geq \frac{\epsilon_1}{C}$ implies that there exists $T > 0$ such that $d(x(T), M) \geq ||V_+(T)|| \geq \frac{\epsilon_1}{2C} > \epsilon$. By

Theorem 1 of Fonda [5] there exists $\eta > 0$ such that $\liminf_{t\to\infty} d(x(t), M) \geq \eta$ for all such solutions, in particular for positive solutions.

Therefore for $0 < f \leq 1$ the stable manifold of M does not intersect $\mathring{\mathbb{R}}^4_+$. Thus by Theorem 4.3 in [6] the flow is uniformly strongly persistent; that is, there exists $\epsilon_0 > 0$ such that $\liminf_{t\to\infty} d(x(t), \partial\mathbb{R}^4_+) > \epsilon_0$ for all positive solutions $x(t)$ of (16)-(19). □

2.5. **Extinction of the disease when $R_0 \leq 1$.**

Theorem 2.4. *If $R_0 \leq 1$, then all nonnegative solutions of (1)-(6) converge to the DFE $(0, 0, 0, 0, N_0, V^*)$.*

Proof. Note that $R_0 \leq 1$ implies that $f > 0$. Suppose $\limsup_{t\to\infty} I_1(t) = m > 0$. Then for every $\epsilon > 0$ there exists $\tau_1 > 0$ such that $I_1(t) \leq m + \epsilon$ for all $t \geq \tau_1$. Substituting, we have that

$$\dot{I}_2(t) \leq \gamma(m + \epsilon) - (\alpha + \rho)I_2(t)$$

for all $t \geq \tau_1$. Then there exists $\tau_2 > \tau_1$ such that

$$I_2(t) \leq \frac{\gamma(m + \epsilon)}{\alpha + \rho} + \epsilon$$

for all $t \geq \tau_2$. This means that

$$I_1(t) + I_2(t) \leq m + 2\epsilon + \frac{\gamma(m + \epsilon)}{\alpha + \rho}$$

for all $t \geq \tau_2$. Substituting again, we get that

$$\dot{R}(t) \leq \alpha\left(\frac{\gamma(m + \epsilon)}{\alpha + \rho} + \epsilon\right) - \rho R(t)$$

for all $t \geq \tau_2$. Thus there exists $\tau_3 > \tau_2$ such that

$$R(t) \leq \frac{\alpha}{\rho}\left(\frac{\gamma(m + \epsilon)}{\alpha + \rho} + \epsilon\right) + \epsilon$$

for all $t \geq \tau_3$. So

$$I_2(t) + R(t) \leq \frac{\gamma(m + \epsilon) + \alpha\epsilon}{\rho} + 2\epsilon$$

for all $t \geq \tau_3$.

We have that $\dot{N}(t) = b(1 - \frac{N}{N_0})$, so $N(t) \to N_0$ as $t \to \infty$. Thus there exists $\tau_4 > 0$ such that $N_0 - \epsilon \leq N(t)$ for all $t \geq \tau_4$. We also have that $\dot{V}(t) = rV(1 - \frac{V}{K}) - \mu V$, and thus $V(t) \to V^*$ as $t \to \infty$. So there exists $\tau_5 > 0$ such that $V^* - \epsilon \leq V(t)$ for all $t \geq \tau_5$. We now substitute this to get that

$$\dot{V_+}(t) \leq \beta\frac{m + 2\epsilon + \frac{\gamma(m+\epsilon)}{\alpha+\rho}}{N_0 - \epsilon} - \mu V_+(t)$$

for all $t \geq \tau_2, \tau_4$. Then there exists $\tau_6 > \max\{\tau_2, \tau_4\}$ such that

$$V_+(t) \leq \frac{\beta(m + 2\epsilon + \frac{\gamma(m+\epsilon)}{\alpha+\rho})}{\mu(N_0 - \epsilon)} + \epsilon$$

for all $t \geq \tau_6$. Now we can rewrite

$$\begin{aligned}
\dot{I}_1(t) &\leq \frac{S(t)}{N(t)}\beta\frac{\beta(m+2\epsilon+\frac{\gamma(m+\epsilon)}{\alpha+\rho})+\epsilon\mu(N_0-\epsilon)}{\mu(N_0-\epsilon)(V^*-\epsilon)} + (1-f)\rho(\frac{\gamma(m+\epsilon)+\alpha\epsilon}{\rho}+2\epsilon) - \gamma I_1(t) \\
&\leq \frac{N(t)-I_1(t)}{N(t)}\beta\frac{\beta(m+2\epsilon+\frac{\gamma(m+\epsilon)}{\alpha+\rho})+\epsilon\mu(N_0-\epsilon)}{\mu(N_0-\epsilon)(V^*-\epsilon)} + (1-f)\rho(\frac{\gamma(m+\epsilon)+\alpha\epsilon}{\rho}+2\epsilon) - \gamma I_1(t) \\
&\leq \beta\frac{\beta(m+2\epsilon+\frac{\gamma(m+\epsilon)}{\alpha+\rho})+\epsilon\mu(N_0-\epsilon)}{\mu(N_0-\epsilon)(V^*-\epsilon)} + (1-f)\rho(\frac{\gamma(m+\epsilon)+\alpha\epsilon}{\rho}+2\epsilon) \\
&\quad -\gamma I_1(t) - \beta\frac{\beta(m+2\epsilon+\frac{\gamma(m+\epsilon)}{\alpha+\rho})+\epsilon\mu(N_0-\epsilon)}{\mu(N_0-\epsilon)(V^*-\epsilon)(N_0+\epsilon)}I_1(t) \text{ for all } t \geq \tau_3, \tau_5, \tau_6.
\end{aligned}$$

Then there exists $\tau_7 > \max\{\tau_3, \tau_5, \tau_6\}$ such that

$$I_1(t) \leq \frac{\beta\frac{\beta(m+2\epsilon+\frac{\gamma(m+\epsilon)}{\alpha+\rho})+\epsilon\mu(N_0-\epsilon)}{\mu(N_0-\epsilon)(V^*-\epsilon)} + (1-f)\rho(\frac{\gamma(m+\epsilon)+\alpha\epsilon}{\rho}+2\epsilon)}{\gamma + \beta\frac{\beta(m+2\epsilon+\frac{\gamma(m+\epsilon)}{\alpha+\rho})+\epsilon\mu(N_0-\epsilon)}{\mu(N_0-\epsilon)(V^*-\epsilon)(N_0+\epsilon)}} + \epsilon$$

for all $t \geq \tau_7$. Recall from Section 2.3 the quantity T_0 whose threshold behavior is equivalent to that of R_0. Now as $\epsilon \to 0$, the inequality becomes

$$\begin{aligned}
I_1(t) &\leq \frac{\frac{\beta^2(m+\frac{\gamma m}{\alpha+\rho})}{\mu N_0 V^*}+(1-f)\gamma m}{\gamma+\frac{\beta^2(m+\frac{\gamma m}{\alpha+\rho})}{\mu N_0^2 V^*}} = \frac{m\gamma\left(\frac{\beta^2(\frac{1}{\gamma}+\frac{1}{\alpha+\rho})}{\mu N_0 V^*}+(1-f)\right)}{\gamma+\frac{m\gamma}{N_0}\left(\frac{\beta^2(\frac{1}{\gamma}+\frac{1}{\alpha+\rho})}{\mu N_0 V^*}\right)} \\
&= \frac{mT_0}{1+\frac{m}{N_0}(T_0-(1-f))} = \frac{mT_0}{1-\frac{m}{N_0}(1-f)+\frac{m}{N_0}T_0}.
\end{aligned}$$

Note that $m = \limsup_{t\to\infty} I_1(t) \leq \limsup_{t\to\infty} N(t) = N_0$, so the constant $1-\frac{m}{N_0}(1-f)$ is always positive. This means that $\frac{mT_0}{1-\frac{m}{N_0}(1-f)+\frac{m}{N_0}T_0}$ is an increasing function of T_0, therefore

$$I_1(t) \leq \frac{m}{1+f\frac{m}{N_0}} < m,$$

since $R_0 \leq 1$ implies $T_0 \leq 1$. Thus $\limsup_{t\to\infty} I_1(t) < m$, a contradiction. So $m = 0$. Then $\limsup_{t\to\infty} I_2(t) = \limsup_{t\to\infty} R(t) = \limsup_{t\to\infty} V_+(t) = 0$ follows as well from the inequalities obtained throughout the proof. This means that we must have $\lim_{t\to\infty} S(t) = N_0$ and $\lim_{t\to\infty} V_-(t) = V^*$. So all nonnegative solutions converge to the DFE. □

2.6. **Transient behavior.** In the model we have been considering thus far, we have accounted for roguing of symptomatic as well as dead trees. However, it is worthwhile to examine the cases when only roguing of symptomatic trees occurs or when there is no roguing at all. We will show that in either of these situations the disease is transient; that is,

$$\lim_{t\to\infty} I_1(t) = \lim_{t\to\infty} I_2(t) = \lim_{t\to\infty} V_+(t) = 0.$$

The system that we analyze now is the same as our original system, except that it does not include a roguing term in the equation for $\dot{R}$, and thus also does not

account for the replanting of a rogued dead tree in either the $\dot{S}$ or $\dot{I}_1$ equations. We allow for roguing of I_2 trees at rate ρ, which can be zero. Note that since dead trees are no longer rogued and replanted possibly as infected trees, the R compartment is not considered to be infected.

$$\dot{I}_1 = \beta\frac{S}{N}\frac{V_+}{V} - \gamma I_1 + \rho(1-f)I_2, \tag{20}$$

$$\dot{I}_2 = \gamma I_1 - (\alpha+\rho)I_2, \tag{21}$$

$$\dot{R} = \alpha I_2, \tag{22}$$

$$\dot{V}_+ = \beta\frac{V_-}{V}\frac{I_1+I_2}{N} - \mu V_+. \tag{23}$$

$$\dot{S} = b\left(1-\frac{N}{N_0}\right) - \beta\frac{S}{N}\frac{V_+}{V} + \rho f I_2, \tag{24}$$

$$\dot{V}_- = r(V_-+V_+)\left(1-\frac{V_-+V_+}{K}\right) - \beta\frac{V_-}{V}\frac{I_1+I_2}{N} - \mu V_-. \tag{25}$$

Theorem 2.5. *For all nonnegative solutions of the system (20)-(25) and for all $\rho \geq 0$ the disease is transient.*

Proof. For this system $\lim_{t\to\infty} N(t) = N_0$ and $\lim_{t\to\infty} V(t) = V^*$ as shown for the original system in the proof of Theorem 2.4. Here we have $\dot{R} = \alpha I_2$. Since $I_2 \geq 0$ and $\dot{I}_2(t)$ is bounded, an application of Barbalat's lemma [7] implies that $\lim_{t\to\infty} I_2 = 0$ so that R does not increase without bound. This similarly forces $\lim_{t\to\infty} I_1 = 0$ as a result of equation (21). Thus $\lim_{t\to\infty} V_+ = 0$ as well because of equation (23). So all infected compartments eventually become extinct. □

Theorem 2.6. *In the system (20)-(25), $\lim\limits_{t\to\infty} S = S_\infty > 0$ for any $\rho \geq 0$.*

Proof. Integrating equation (22), we have that $R(t) - R(0) = \alpha\int_0^t I_2(\tau)\,d\tau$. Since $R(0)$ and $R(t)$ are both bounded, their difference is bounded, and thus $\int_0^t I_2(\tau)\,d\tau$ is bounded. Similarly, equation (21) gives that $I_2(t) - I_2(0) + (\alpha+\rho)\int_0^t I_2(\tau)\,d\tau = \gamma\int_0^t I_1(\tau)\,d\tau$, and thus $\int_0^t I_1(\tau)\,d\tau$ is bounded as well. Let $\epsilon > 0$. Since $N(t) \leq N_0$ for all t, $\lim_{t\to\infty} N(t) = N_0$, and $\lim_{t\to\infty} V(t) = V^*$, then there exists $T > 0$ such that $N_0 - \epsilon \leq N(t) \leq N_0$ and $V^* - \epsilon \leq V(t) \leq V^* + \epsilon$ for all $t \geq T$. Since $\rho \geq 0$ by equation (24) we have the inequality

$$\dot{S} \geq -\beta\frac{S}{N}\frac{V_+}{V} \geq -\beta\frac{S}{(N_0-\epsilon)}\frac{V_+}{(V^*-\epsilon)}.$$

Therefore $S(t) \geq S(0)\exp\left(-\frac{\beta}{(N_0-\epsilon)(V^*-\epsilon)}\int_0^t V_+(\tau)\,d\tau\right)$ for all $t \geq T$. By way of contradiction, assume that $S_\infty = 0$. Then by the above inequality, $\lim_{t\to\infty}\int_0^t V_+(\tau)\,d\tau = \infty$. Note that $\beta\frac{V_-}{V}\frac{I_1+I_2}{N} \leq \beta\frac{V}{V}\frac{I_1+I_2}{N_0-\epsilon}$ for all $t \geq T$. Then for all $t \geq T$

$$\begin{aligned}
&\int_0^t \beta\frac{V_-(\tau)}{V(\tau)}\frac{I_1(\tau)+I_2(\tau)}{N(\tau)}\,d\tau \\
&\leq \int_0^T \beta\frac{V_-(\tau)}{V(\tau)}\frac{(I_1(\tau)+I_2(\tau))}{N(\tau)}\,d\tau + \int_T^t \beta\frac{(I_1(\tau)+I_2(\tau))}{N_0-\epsilon}\,d\tau \\
&\leq \beta T + \frac{\beta}{N_0-\epsilon}\int_T^t (I_1(\tau)+I_2(\tau))\,d\tau
\end{aligned}$$

$$\leq \beta T + \frac{\beta}{N_0 - \epsilon} \int_0^t (I_1(\tau) + I_2(\tau))\, d\tau,$$

which is bounded. By equation (23), we have $V_+(t) - V_+(0) = \int_0^t \beta \frac{V_-(\tau)}{V} \frac{I_1(\tau)+I_2(\tau)}{N}\, d\tau - \mu \int_0^t V_+(\tau)\, d\tau$. Since the first integral is bounded by the previous inequality, it follows that $\lim_{t\to\infty} V_+(t) = -\infty$, a contradiction. Thus $S_\infty > 0$. □

Theorems 2.5 and 2.6 tell us that when roguing of dead trees does not occur, the disease will eventually die out leaving only healthy and dead trees. The presence of remaining healthy trees is a feature similar to that of the standard SIR epidemic model. This leads to a question about the benefits of roguing and replanting. Is it worthwhile to rogue infected, symptomatic trees, or should no trees be rogued? In either case the disease is transient; the difference is the number of trees that remain healthy. We use simulations to compare the values of S_∞, the number of unaffected trees, in each situation. Consider the hypothesized parameter values $N_0 = b = \beta = \alpha = \gamma = \mu = 1$ and $r = 1.5$ in both cases, with $\rho = 1$ and $f = 0.5$ for the system including roguing and $\rho = 0$ for the system excluding roguing. We begin with only susceptible trees and a small number of infected psyllids; the initial conditions are $(I_1(0), I_2(0), R(0), V_+(0), S(0), V_-(0)) = (0, 0, 0, 0.1, 1, 0.9)$. This results in $S_\infty = 0.2750$ with roguing and $S_\infty = 0.2192$ without roguing, so more trees are unaffected when roguing occurs. With all combinations of parameter values that we explored, the simulations support the notion that more healthy trees will remain when symptomatic trees are rogued than when no trees are rogued.

3. **Modifications of the model.** In formulating the original model, we assumed that psyllids are equally attracted to all types of trees, including the dead ones, which may be biologically unrealistic. We now consider a modified version of our model in which we assume that the psyllids are not attracted to dead trees. That is, the probability that a tree bitten by a vector is a susceptible tree is defined to be

$$\phi = \frac{S}{S + I_1 + I_2}.$$

The new model is then given by:

$$\dot{I}_1 = \beta\phi\frac{V_+}{V} - \gamma I_1 + \rho(1-f)(I_2 + R), \tag{26}$$

$$\dot{I}_2 = \gamma I_1 - (\alpha + \rho) I_2, \tag{27}$$

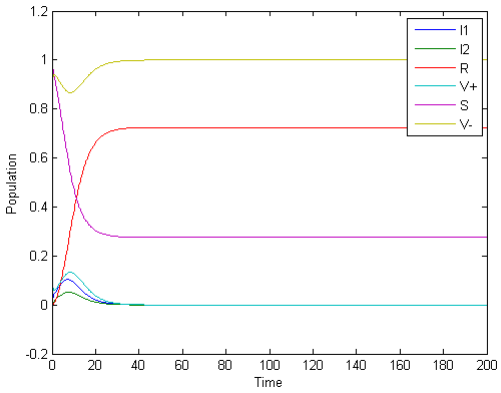
$$\dot{R} = \alpha I_2 - \rho R, \tag{28}$$

$$\dot{V}_+ = \beta\frac{V_-}{V}(1-\phi) - \mu V_+, \tag{29}$$

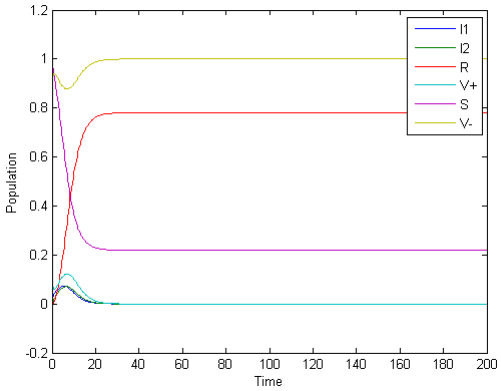
$$\dot{S} = b\left(1 - \frac{N}{N_0}\right) - \beta\phi\frac{V_+}{V} + \rho f(I_2 + R), \tag{30}$$

$$\dot{V}_- = r(V_- + V_+)\left(1 - \frac{V_- + V_+}{K}\right) - \beta\frac{V_-}{V}(1-\phi) - \mu V_-, \tag{31}$$

which we study away from the singularity $S = I_1 = I_2 = 0$. The system (26)-(31) has the same DFE as our original system (1)-(6). It also holds that $N = N_0$ and $V = V^*$ at the equilibrium of (26)-(31). However in the new system we have the possibility for two positive endemic equilibria. We define the following quadratic



(a) $\rho = 1, f = 0.5$



(b) $\rho = 0$

FIGURE 3. Simulation of system (20)-(25) using MATLAB ode45 solver with $r = 1.5$, $N_0 = b = \beta = \alpha = \gamma = \mu = 1$, initial condition $(I_1(0), I_2(0), R(0), V_+(0), S(0), V_-(0)) = (0, 0, 0, 0.1, 1, 0.9)$, and ρ as specified in (a) and (b).

equation:

$$g(\phi) = \frac{\gamma\alpha}{\rho(\alpha+\rho)}\phi^2 - \left(\frac{\rho+\gamma}{\rho} + \frac{\gamma f N_0}{\beta}\right)\phi + \frac{\gamma N_0 f(\beta + V^*\mu)}{\beta^2}$$

where easy calculation shows that $g(\phi^*) = 0$ for any endemic equilibrium value ϕ^*. In Theorem 3.1, we will prove the existence of the two positive equilibria given certain parameter values. Additionally, computation reveals that the next generation matrix of (26)-(31) is identical to the next generation matrix of (1)-(6) calculated in Part 1 of the Appendix. Therefore for the modified system we also have

$$R_0 = \frac{1-f}{2} + \sqrt{\frac{(1-f)^2}{4} + \frac{\beta^2}{\mu V^* N_0}\left(\frac{1}{\gamma} + \frac{1}{\alpha+\rho}\right)}.$$

It is easy to show that $R_0 > 1$ when $f = 0$ and there exists a unique, globally attractive endemic equilibrium; the proof is similar to that of Theorem 2.2. However, in the case $f > 0$, we have the following result. For use in Theorem 3.1 we define

$$\phi_{\text{crit}} = \frac{\alpha+\rho}{2\alpha}\left(\frac{\rho+\gamma}{\gamma} + \frac{f\rho N_0}{\beta}\right).$$

Theorem 3.1. *Two distinct positive endemic equilibria of (26)-(31) exist if and only if*
(i) $\phi_{crit} < 1$ *and*
(ii)

$$\frac{\beta^2}{f\mu N_0}\left(\frac{1}{\gamma}+\frac{1}{\alpha+\rho}\right) < V^* < \frac{\beta}{\mu}\left[\phi_{crit}\left(1-\frac{\mu}{2\rho}+\frac{\beta(\rho+\gamma)}{2\rho\gamma f N_0}\right)-1\right].$$

Furthermore, (ii) implies that $R_0 < 1$.

Proof. For $\phi \in (0,1)$ we have that ϕ is an equilibrium value if and only if $g(\phi) = 0$. So it suffices to show that g has two roots in $(0,1)$ if and only if (i) and (ii) hold. Observe that $g(0) > 0$ and that the critical value of $g(\phi)$ is given by ϕ_{crit}. Then g will have two roots in $(0,1)$ if and only if $g(1) > 0$, $0 < \phi_{\text{crit}} < 1$ and $g(\phi_{\text{crit}}) < 0$. It is trivial that $0 < \phi_{\text{crit}}$. We have

$$g(1) = \frac{\gamma N_0 V^* \mu f}{\beta} - \left(1+\frac{\gamma}{\alpha+\rho}\right).$$

Then $g(1) > 0$ if and only if

$$\frac{\beta^2}{f\mu N_0}\left(\frac{1}{\gamma}+\frac{1}{\alpha+\rho}\right) < V^*$$

as in condition (ii). Note that it is trivial from this inequality that (ii) implies $R_0 < 1$. We prove now that $g(\phi_{\text{crit}}) < 0$ if and only if the right-hand inequality of (ii) holds. Indeed,

$$\begin{aligned}
g(\phi_{\text{crit}}) < 0 \quad &\Leftrightarrow \quad \frac{\gamma f N_0(V^*\mu+\beta)}{\beta^2} < \left(\frac{\gamma f N_0}{\beta}+\frac{\rho+\gamma}{\rho}\right)\phi_{\text{crit}} - \frac{\gamma\alpha}{\rho(\alpha+\rho)}\phi_{\text{crit}}^2 \\
&\Leftrightarrow \quad V^* < \frac{\beta^2}{\gamma f N_0 \mu}\left[\left(\frac{\gamma f N_0}{\beta}+\frac{\rho+\gamma}{\rho}\right)\phi_{\text{crit}} - \frac{\gamma\alpha}{\rho(\alpha+\rho)}\phi_{\text{crit}}^2\right] - \frac{\beta}{\mu} \\
&\Leftrightarrow \quad V^* < \frac{\beta^2}{\gamma f N_0 \mu}\phi_{\text{crit}}\left[\frac{\gamma f N_0}{\beta}\left(1-\frac{\mu}{2\rho}\right)+\frac{\rho+\gamma}{2\rho}\right] - \frac{\beta}{\mu} \\
&\Leftrightarrow \quad V^* < \frac{\beta}{\mu}\left[\phi_{\text{crit}}\left(1-\frac{\mu}{2\rho}+\frac{\beta(\rho+\gamma)}{2\rho\gamma f N_0}\right)-1\right].
\end{aligned}$$

□

We next examine the stability of the equilibria in the case that multiple positive endemic equilibria exist, which is when $R_0 < 1$. Consider the parameter values $f = \beta = 0.1$, $\rho = 0.01$, $r = 1.5$, and $b = N_0 = \alpha = \gamma = \mu = V^* = 1$, which result in the value $R_0 = 0.9199$. We find that the DFE as well as one of the endemic equilibria will be stable, as all eigenvalues of the Jacobian evaluated at each of these equilbria have negative real parts. However, the other endemic equilibrium is unstable; for this particular equilibrium, the Jacobian has a positive real eigenvalue.

We next examine the stability of the equilibria in the case that multiple positive endemic equilibria exist, which is when $R_0 < 1$. Consider the hypothesized parameter values $f = \beta = 0.1$, $\rho = 0.01$, $r = 1.5$, and $b = N_0 = \alpha = \gamma = \mu = V^* = 1$, which result in the value $R_0 = 0.9199$. We find that the DFE as well as one of the endemic equilibria will be stable, as all eigenvalues of the Jacobian evaluated at each of these equilbria have negative real parts. However, the other endemic equilibrium is unstable; for this particular equilibrium, the Jacobian has a positive real eigenvalue.

Simulations in Figure 4 show that, depending on the initial conditions used in combination with the above parameter values, the solutions $(I_1(t), I_2(t), R(t), V_+(t), S(t), V_-(t))$ will converge to one of the two stable equilibria. First, with the initial condition $(0, 0, 0.5, 0, 0.5, 1)$, convergence is to the DFE $(0, 0, 0, 0, 1, 1)$. If instead we use the initial condition $(0, 0, 0.9, 0, 0.1, 1)$ the solution converges to the endemic equilibria at $(0.01, 0.01, 0.977, 0.081, 0.003, 0.919)$.

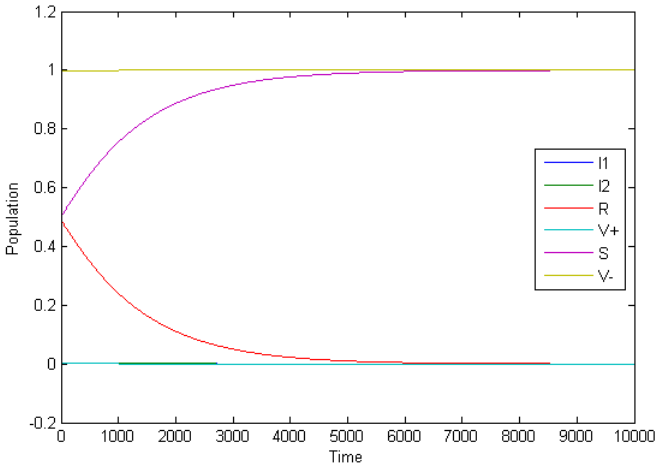
4. **Discussion.** The model developed in this paper describes the population dynamics for a grove in the presence of citrus greening disease. The model incorporates a control strategy of roguing and replanting infected trees. Models for other plant virus diseases have included roguing, such as those for banana bunchy top [1] and citrus tristeza virus [4]. Chan and Jeger [3] also developed a model including healthy, latently infected, infectious and post-infectious plants and investigated the population dynamics with and without roguing. Analysis of their model with roguing showed that the basic reproductive number and the equilibrium healthy population did not depend on whether roguing was done in the post-infectious category. How do different roguing methods affect our model? To answer this we considered a control method where roguing of the I_2 and R trees is done at rates ρ_1 and ρ_2, respectively. The case where $\rho_2 = 0$ was analyzed in Section 2.6 where we concluded that the disease is transient and a positive population of healthy trees will remain indefinitely. While we don't have a complete analytical understanding of the model when $\rho_1 > 0$ and $\rho_2 = 0$, simulations suggest that the level of remaining healthy trees is larger when roguing is performed in I_2 than with no roguing at all.

If both $\rho_1 > 0$ and $\rho_2 > 0$ then the disease is no longer transient and an endemic equilibrium will exist when $R_0 > 1$. Substituting ρ_1 and ρ_2 appropriately in system (1)-(6), the reproductive number is recalculated to be:

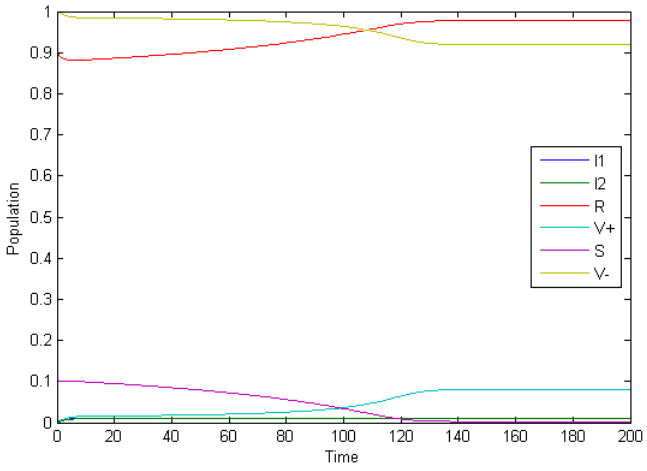
$$R_0 = \frac{1-f}{2} + \sqrt{\frac{(1-f)^2}{4} + \frac{\beta^2}{\mu V^* N_0}\left(\frac{1}{\gamma} + \frac{1}{\alpha+\rho_1}\right)}.$$

For our model, as for Chan and Jeger's model, R_0 does not depend on the rate of roguing of the dead trees. Although the disease will be maintained in the case when $R_0 > 1$, it may be that S^*, the endemic equilibrium healthy tree population, is higher even in the presence of disease as compared to the case with no roguing where the disease dies out. That is, there may be a tradeoff between allowing the disease to persist and maintaining a profitable level of healthy trees. Therefore it is worth considering the dependence of S^* on ρ_1 and ρ_2. Indeed, S^* is determined to be:

$$S^* = \frac{fN_0^2}{\beta}\left[\frac{1 + \frac{\mu V^*}{\beta}\left(1 + \frac{\alpha\gamma}{\rho_2(\alpha+\rho_1+\gamma)}\right)}{\frac{fN_0}{\beta} + \frac{1}{\gamma} + \frac{1}{\alpha+\rho_1}\left(1 + \frac{\alpha}{\rho_2}\right)}\right].$$



(a) $(0, 0, 0.5, 0, 0.5, 1)$



(b) $(0, 0, 0.9, 0, 0.1, 1)$

FIGURE 4. Simulation of system (26)-(31) using MATLAB ode45 solver with $f = \beta = 0.1$, $\rho = 0.01$, $r = 1.5$, $b = N_0 = \alpha = \gamma = \mu = V^* = 1$, and initial condition $(I_1(0), I_2(0), R(0), V_+(0), S(0), V_-(0))$ as specified in (a) and (b).

Unlike the result of Chan and Jeger we see that the equilibrium healthy population depends on whether roguing is done in both the infectious, symptomatic stage and in the post-infectious stage. From this expression we can determine that S^* increases as ρ_1 and ρ_2 increase (see Part 2 of the Appendix).

These results for varying roguing methods depend on the novel feature of our model which is the inclusion of the positive probability $1 - f$ that a replanted tree will immediately become infected. That is, the trees in the R stage are a potential

source of infection due to the soil and remaining root system being a reservoir for the disease. As expected, we find that R_0 decreases as f increases. Dependence of the basic reproductive number on other parameters also agrees with what we predict biologically; R_0 increases as the biting rate increases or the mean lifetime of a psyllid or an infected tree increases. Less expected is that R_0 is unbounded as V^* or N_0 approaches zero. However, modifying the system slightly to include a saturating contact rate gives a bounded R_0 of a very similar form. With conditions on R_0 we were able to establish extinction and uniform persistence results, and perform numerical simulations which suggest additional stability conclusions.

Another modification to consider is incorporating psyllid migration. This certainly has a practical application, as psyllid movement is critical to the transport of disease between groves. Emigration of the insects out of the grove could be incorporated into the death rate μ. To account for migration into the grove, we could include a constant immigration rate. The case where a positive fraction of the immigrating psyllids are infected would no longer yield a DFE. However, when only healthy psyllids migrate to the grove, the V^* value would change while all other results would remain the same.

Recent field research has suggested that vertical transmission of the disease among psyllids is not only present, but perhaps very influential in the spread of the disease [10]. To allow for this possibility, we could incorporate a parameter π that represents the proportion of offspring of infected psyllids that are born infected, while the proportion $1-\pi$ are born healthy. Preliminary analysis shows that this modification effectively changes the parameter μ to $(1-\pi)\mu$, but all subsequent results remain the same.

All parameter values used in the simulations are hypothesized. Some parameter values can be estimated easier than others; for instance, knowing an approximate length of an infected tree's asymptomatic stage allows a rough estimate of the parameter γ. However, recent observations indicate this stage length may have as wide a range as six months to six years [10]. There is even less known about the biting rate of psyllids or the potential of soil to act as a reservoir for the disease, and thus it is much more difficult to estimate parameters such as β and f. Forthcoming data might allow estimation of parameters to provide more realistic simulations in future work.

Appendix.

Part 1. The basic reproductive number R_0 represents the average number of secondary infections that result from the introduction of one infected individual into a susceptible population. Mathematically, R_0 has been defined to be the spectral radius of the next generation matrix [17]. That is, we rewrite the vector field of (1)-(6) as

$$\dot{x_i} = F_i(x) - V_i(x), \qquad i = 1, ..., 6,$$

where $F_i(x)$ represents the rate of new infections appearing in state i and $V_i = V_i^- - V_i^+$, where V_i^+ is the rate of individuals entering state i by all other means and V_i^- is the rate of individuals leaving state i. We let

$$F = \left[\frac{\partial F_i}{\partial x_j}(x_0)\right], V = \left[\frac{\partial V_i}{\partial x_j}(x_0)\right] \qquad 1 \le i, j \le 4$$

where x_0 is the DFE. Thus F is the matrix of derivatives corresponding to new infections in the infected compartments while V is the matrix of derivatives corresponding to all other modes of entering or exiting an infected compartment. Van den Driessche and Watmough [17] then define

$$R_0 = \rho(FV^{-1}).$$

We compute the next generation matrix FV^{-1} for system (1)-(6). Calculation gives

$$F = \begin{bmatrix} 0 & \rho(1-f) & \rho(1-f) & \frac{\beta}{V^*} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\beta}{N_0} & \frac{\beta}{N_0} & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} \gamma & 0 & 0 & 0 \\ -\gamma & \alpha+\rho & 0 & 0 \\ 0 & -\alpha & \rho & 0 \\ 0 & 0 & 0 & \mu \end{bmatrix},$$

and

$$V^{-1} = \begin{bmatrix} \frac{1}{\gamma} & 0 & 0 & 0 \\ \frac{1}{\alpha+\rho} & \frac{1}{\alpha+\rho} & 0 & 0 \\ \frac{\alpha}{\rho(\alpha+\rho)} & \frac{\alpha}{\rho(\alpha+\rho)} & \frac{1}{\rho} & 0 \\ 0 & 0 & 0 & \frac{1}{\mu} \end{bmatrix}.$$

Therefore

$$FV^{-1} = \begin{bmatrix} 1-f & 1-f & 1-f & \frac{\beta}{\mu V^*} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\beta}{N_0}\left(\frac{1}{\gamma}+\frac{1}{\alpha+\rho}\right) & \frac{\beta}{N_0(\alpha+\rho)} & 0 & 0 \end{bmatrix}.$$

Clearly, FV^{-1} has two zero eigenvalues. The remaining eigenvalues are determined by

$$\lambda^2 - (1-f)\lambda - \frac{\beta^2}{\mu V^* N_0}\left(\frac{1}{\gamma}+\frac{1}{\alpha+\rho}\right) = 0.$$

Thus, the spectral radius of FV^{-1} is:

$$\rho(FV^{-1}) = R_0 = \frac{1-f}{2} + \sqrt{\frac{(1-f)^2}{4} + \frac{\beta^2}{\mu V^* N_0}\left(\frac{1}{\gamma}+\frac{1}{\alpha+\rho}\right)}.$$

Part 2.

Proposition 1. *Let $R_0 > 1$. Then the endemic equilibrium level of healthy trees*

$$S^* = \frac{fN_0^2}{\beta}\left[\frac{1+\frac{\mu V^*}{\beta}\left(1+\frac{\alpha\gamma}{\rho_2(\alpha+\rho_1+\gamma)}\right)}{\frac{fN_0}{\beta}+\frac{1}{\gamma}+\frac{1}{\alpha+\rho_1}\left(1+\frac{\alpha}{\rho_2}\right)}\right]$$

is increasing with respect to ρ_1 and ρ_2.

Proof. We first prove that S^* increases as ρ_1 increases. We have

$$S^* = \frac{fN_0^2}{\beta}\left[\frac{1+\frac{\mu V^*}{\beta}+\frac{\mu V^*\alpha\gamma}{\beta\rho_2(\alpha+\rho_1+\gamma)}}{\frac{fN_0}{\beta}+\frac{1}{\gamma}+\frac{1}{\alpha+\rho_1}\left(1+\frac{\alpha}{\rho_2}\right)}\right]$$
$$= \frac{fN_0^2}{\beta}\left[\frac{a+\frac{b}{x+\gamma}}{c+\frac{d}{x}}\right] = \frac{fN_0^2}{\beta}\left[\frac{ax^2+(a\gamma+b)x}{cx^2+(c\gamma+d)x+d\gamma}\right],$$

where $x=\alpha+\rho_1$, $a=1+\frac{\mu V^*}{\beta}$, $b=\frac{\mu V^*\alpha\gamma}{\beta\rho_2}$, $c=\frac{fN_0}{\beta}+\frac{1}{\gamma}$, and $d=1+\frac{\alpha}{\rho_2}$. Note that x, a, b, c, and d are positive for $\rho_1 \geq 0$. After differentiating and some simplification we determine

$$\frac{\partial S^*}{\partial \rho_1} = \frac{fN_0^2}{\beta}\left[\frac{(ad-bc)x^2+2ad\gamma x+d\gamma(a\gamma+b)}{(cx^2+(c\gamma+d)x+d\gamma)^2}\right]$$

$$\geq \frac{fN_0^2}{\beta}\left[\frac{x[(ad-bc)x+2ad\gamma]}{(cx^2+(c\gamma+d)x+d\gamma)^2}\right].$$

Therefore it suffices to show that $(ad-bc)x+2ad\gamma>0$. Recalling the expression for R_0 from the Discussion we observe that, similar to the proof of Theorem 2.1, $R_0>1$ if and only if

$$\frac{\beta^2\left(1+\frac{\gamma}{\alpha+\rho_1}\right)}{\mu V^* N_0 f\gamma} > 1.$$

Using this inequality we calculate

$$\begin{aligned}(ad-bc)x+2ad\gamma &= \left[1+\frac{\mu V^*}{\beta}+\frac{\alpha}{\rho_2}-\frac{\mu V^* N_0 f\gamma\alpha}{\beta^2\rho_2}\right](\alpha+\rho_1)\\ &\quad+2\gamma\left(1+\frac{\mu V^*}{\beta}\right)\left(1+\frac{\alpha}{\rho_2}\right)\\ &> \left[1+\frac{\mu V^*}{\beta}+\frac{\alpha}{\rho_2}-\frac{\alpha}{\rho_2}\left(1+\frac{\gamma}{\alpha+\rho_1}\right)\right](\alpha+\rho_1)\\ &\quad+2\gamma\left(1+\frac{\mu V^*}{\beta}+\frac{\alpha}{\rho_2}+\frac{\mu V^*\alpha}{\beta\rho_2}\right)\\ &> \left[1+\frac{\mu V^*}{\beta}-\frac{\alpha\gamma}{\rho_2(\alpha+\rho_1)}\right](\alpha+\rho_1)+\frac{2\alpha\gamma}{\rho_2}\\ &= \left(1+\frac{\mu V^*}{\beta}\right)(\alpha+\rho_1)+\frac{\alpha\gamma}{\rho_2}>0.\end{aligned}$$

It is proved similarly that S^* is increasing with respect to ρ_2. Indeed, we have

$$S^* = \frac{fN_0^2}{\beta}\left[\frac{1+\frac{\mu V^*}{\beta}+\frac{\mu V^*\alpha\gamma}{\beta(\alpha+\rho_1+\gamma)\rho_2}}{\frac{fN_0}{\beta}+\frac{1}{\gamma}+\frac{1}{\alpha+\rho_1}+\frac{\alpha}{(\alpha+\rho_1)\rho_2}}\right] = \frac{fN_0^2}{\beta}\left[\frac{g+\frac{h}{\rho_2}}{j+\frac{k}{\rho_2}}\right] = \frac{fN_0^2}{\beta}\left[\frac{g\rho_2+h}{j\rho_2+k}\right],$$

where $g=1+\frac{\mu V^*}{\beta}$, $h=\frac{\mu V^*\alpha\gamma}{\beta(\alpha+\rho_1+\gamma)}$, $j=\frac{fN_0}{\beta}+\frac{1}{\gamma}+\frac{1}{\alpha+\rho_1}$, and $k=\frac{\alpha}{\alpha+\rho_1}$. Note that g, h, j, and k are positive. Hence

$$\frac{\partial S^*}{\partial \rho_2} = \frac{fN_0^2}{\beta}\left(\frac{gk-hj}{(j\rho_2+k)^2}\right)$$

and it suffices to show that $gk - hj > 0$. Since $R_0 > 1$ we use the inequality above again to obtain

$$\begin{aligned} gk - hj &= \left(1 + \frac{\mu V^*}{\beta}\right)\frac{\alpha}{\alpha+\rho_1} - \frac{\mu V^* \alpha\gamma}{\beta(\alpha+\rho_1+\gamma)}\left(\frac{fN_0}{\beta} + \frac{\alpha+\rho_1+\gamma}{\gamma(\alpha+\rho_1)}\right) \\ &= \frac{\alpha}{\alpha+\rho_1} - \frac{\mu V^* \alpha N_0 f\gamma}{\beta^2(\alpha+\rho_1+\gamma)} \\ &= \frac{\alpha}{\alpha+\rho_1}\left(1 - \frac{\mu V^* N_0 f\gamma}{\beta^2\left(1+\frac{\gamma}{\alpha+\rho_1}\right)}\right) \\ &> 0. \end{aligned}$$

□

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