

Research article

Transmission dynamics and optimal control of stage-structured HLB model

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Abstract: Citrus Huanglongbing (HLB) is one of severe quarantine diseases affecting citrus production both in abroad and domestic. Based on the mechanism and characteristics of citrus HLB transmission, we establish a vector-borne model with stage structure and integrated strategy and investigate the effect of the strategy in controlling the spread of HLB. By calculating, we obtain the basic reproductive number R_0 , and prove that the disease can be eradicated if $R_0 < 1$, whereas the disease will persist if $R_0 > 1$. Meanwhile, we apply the optimal control theory to obtain an optimal integrated strategy. Finally, we use our model to simulate the data of the numbers of inspected and infected citrus trees in “Yuan Orchard”, located in Ganzhou City, Jiangxi Province in the southeast of P.R China. We also give some numerical simulations for our theoretical findings.

Keywords: Huanglongbing; stage-structured model; basic reproductive number; population dynamics; optimal control

1. Introduction

Huanglongbing (HLB), more commonly known as citrus greening disease, is one of the most dangerous and devastating diseases of citrus worldwide [1, 2]. Two decades ago, it invaded the Western Hemisphere, primarily Florida and Brazil, where it has spread rapidly and caused major damage to global citrus production. It is estimated that citrus acreage in Florida has decreased by 40% and production by 49% since their historical peaks, all of which occurred in the last 20 years [3]. Up to 2015, 20% of Brazil’s commercial citrus species has been infected with HLB, and the disease caused great damage to citrus industry by shortening tree lifespan and poor yield and quality [4]. In São Paulo, 64.1% of the commercial citrus blocks and 6.9% of the citrus trees were affected by HLB in 2012 [5]. In addition, Ganzhou is the top citrus producing area in Jiangxi Province, China, with an annual pro-

duction of approximately 1.2 million tons [6, 7]. However, the producing-area has also suffered from the widespread out of HLB and the output has significantly decreased in recent years [8]. The citrus acreage in Ganzhou had decreased from 2.48 million acres in 2013 to 1.00 million acres in 2018.

HLB is caused by phloem-restricted bacteria of the *Candidatus Liberibacter* group, which can be transmitted by two species of citrus psyllids, the Asian citrus psyllid (ACP), *Diaphorina citri* Kuwayama, and the African citrus psyllid, *Trioza erytreae* [3]. ACP is divided into eggs, 1st through 5th nymphal instars and adults [9]. The five nymphal instars of ACP can be differentiated by their distinct morphological characteristics [10]. Adults and nymphs are capable of acquiring the HLB pathogen after feeding on an infected plant for 30 min or longer [11]. Although the nymphs hardly move, they soon become Las-carrying (*Candidatus Liberibacter asiaticus*) adults with the ability to fly and transmit Las to other citrus plants. Thus the control period of vector psyllid should include the nymphal stages [10]. The results from [10] reported that psyllids can carry Las in either adult or nymphal stages, except in the 1st and 2nd instars, the 3rd through 5th instars have stronger transmission ability than adults.

Generally a healthy citrus tree is inoculated by infected nymphs and adults, there is an incubation period in which the tree exhibits no symptoms but may act as a source of the disease [2]. The survey results from [12, 13] indicated that the incubation period from grafting to development of HLB symptoms is 3 to 12 months under greenhouse conditions. Since incubation period is long and diagnosis is difficult, citrus trees can not be easily found in time after infection. This issue reduces the effectiveness of control strategy in which infected trees are removed to eliminate sources of HLB [14].

Recently, mathematical modeling has become an important and useful tool in understanding the epidemiology of vector-transmitted plant pathogens [15, 16, 17, 18, 19]. For citrus HLB, a few mathematical models currently exist which analyze how HLB spreads within individual trees [20], within a citrus grove [21, 22, 23, 24, 25], or from grove to grove [26]. In [21], the authors reviewed how mathematical models have yielded useful insights into controlling disease spread for vector-borne plant diseases, especially HLB. Note that, for citrus psyllid, different stages have different biological characters, such as reproductive potential, growth, temperature tolerance, transmission efficiency. Therefore, it is very necessary to consider the stage structure of psyllid in mathematical model, including eggs, the 1st and 2nd nymphal instars, the 3rd through 5th nymphal instars and adults. However, the stage structure of psyllid has not been discussed in the previous HLB models. Motivated by the preceding discussion, our first purpose of this paper is to formulate and investigate a HLB model, in which the incubation period of citrus tree and the stage structure of psyllid are taken into consideration.

There is no good source of genetic resistance to HLB in the genus citrus, and the disease cannot be cured once the trees are infected [2, 27]. Current programs for HLB have focused on nutrient solution injection to reduce the infection of the bacteria, removal of infected trees to reduce sources of the disease and insecticide spraying control of the psyllid vector and planting with HLB-free nursery stock [2]. The second aim of this paper is to achieve awareness about the most desirable technique for minimizing the transmission of HLB by using the optimal control theory.

In this paper, different from the previous simple classification of psyllid into susceptible and infected populations, we consider that citrus psyllid in different stages have different abilities and ways to transmit HLB. Based on the above facts, in the next section, we establish a stage-structured HLB model, and obtain the basic reproductive number R_0 of the model. In Section 3, we obtain the equivalent threshold condition T_0 of the basic reproductive number R_0 , and prove extinction of the disease when $R_0 \leq 1$ while persistence of the disease when $R_0 > 1$. In Section 4, we apply the optimal control

technique to minimize the population of infected citrus trees, dead trees and total number of vector population. Different control strategies should be used for the elimination of infection in the population of citrus trees. In Section 5, we use numerical simulation to demonstrate and support the theoretical results. In the last section, we give a brief conclusion.

2. A mathematical model and the basic reproductive number R_0

2.1. Model formulation

In this subsection, Citrus HLB model where transmission is via vector psyllids is formulated. The notation used in the mathematical model includes four states for citrus tree population. S denotes susceptible trees (individuals who can be infected by disease) and R represents dead trees. Due to the latency delay we split the infected trees into a latent stage, E (individuals who infected and asymptomatic but no infectious) and an infectious stage, I (individuals who have the ability to transmit the disease to others). Let $N(t)$ be the total numbers of citrus tree population at time t in a grove, that is, $N(t) = S(t) + E(t) + I(t) + R(t)$. Based on the fact that 3rd through 5th nymphal instars can transmit HLB, the citrus psyllid population is divided into six state variables. We let X_e represent eggs (produced by susceptible adults of psyllids), X_r denote 1st and 2nd nymphal instars (individuals who do not have the ability to transmit the disease to other susceptible trees), X_i and Y_i denote 3rd through 5th susceptible and infected nymphal instars, respectively. X_m and Y_m denote susceptible and infected adults of psyllid, respectively. We assume that the grove is subject to roguing and replanting management strategy. Moreover, we ignore the natural mortality rate of the citrus tree. The model flow diagram is depicted in Figure 1. Considering HLB transmission between citrus trees and psyllids, we establish the following HLB model.

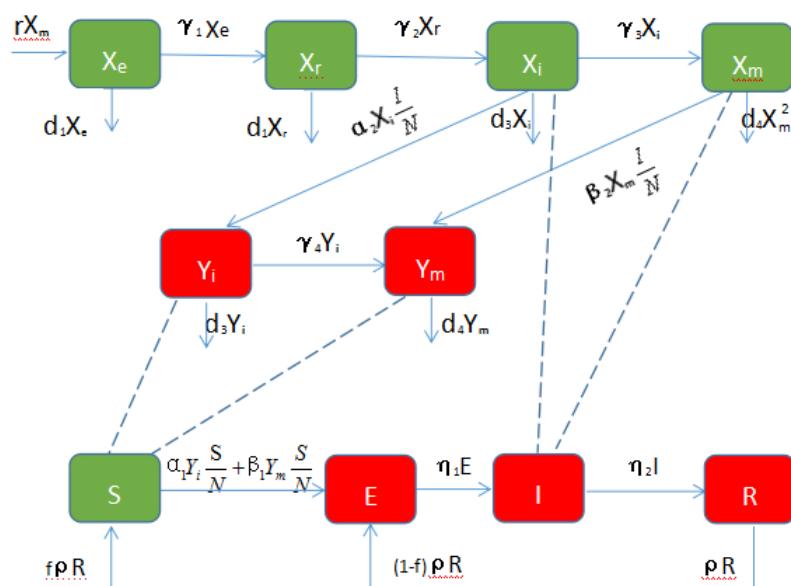


Figure 1. A schematic showing transitions to different categories for trees and psyllids.

$$\begin{aligned}
\frac{dS}{dt} &= f\rho R - \alpha_1 Y_i \frac{S}{N} - \beta_1 Y_m \frac{S}{N}, \\
\frac{dE}{dt} &= \alpha_1 Y_i \frac{S}{N} + \beta_1 Y_m \frac{S}{N} - \eta_1 E + (1-f)\rho R, \\
\frac{dI}{dt} &= \eta_1 E - \eta_2 I, \\
\frac{dR}{dt} &= \eta_2 I - \rho R, \\
\frac{dX_e}{dt} &= rX_m - \gamma_1 X_e - d_1 X_e, \\
\frac{dX_r}{dt} &= \gamma_1 X_e - \gamma_2 X_r - d_2 X_r, \\
\frac{dX_i}{dt} &= \gamma_2 X_r - \alpha_2 X_i \frac{I}{N} - \gamma_3 X_i - d_3 X_i, \\
\frac{dX_m}{dt} &= \gamma_3 X_i - \beta_2 X_m \frac{I}{N} - d_4 X_m^2, \\
\frac{dY_i}{dt} &= \alpha_2 X_i \frac{I}{N} - \gamma_4 Y_i - d_3 Y_i, \\
\frac{dY_m}{dt} &= \gamma_4 Y_i + \beta_2 X_m \frac{I}{N} - d_4 Y_m.
\end{aligned} \tag{1}$$

In model (1), 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 to a latent stage immediately. We also assume $\eta_1, \gamma_1, \gamma_3$ and γ_4 are the conversion rates, η_2 is the disease-induced mortality rate of tree, r denotes oviposition rates of susceptible adult psyllid, d_1 and d_2 represent the natural mortality rates of eggs and 1st through 2nd nymphal, respectively, d_3 is the natural mortality rate of 3rd through 5th nymphal, d_4 is the natural mortality rate of adult psyllid. Let α_1 be the infection rate from 3rd through 5th infected nymphals to susceptible trees, α_2 be the infection rate from infected trees to 3rd through 5th susceptible nymphals, β_1 be the infection rate from infected adult psyllid to susceptible trees, and β_2 be the infection rate from infected trees to susceptible adult psyllids.

Subject to the restriction $\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt} \equiv 0$, without loss of generality, let $N(t) = 1$, and now $S + E + I + R = 1$. Here S, E, I and R are defined separately as susceptibility rate, latent rate, infection rate and removal rate, respectively. Thus, system (1) can be reduced to the form:

$$\begin{aligned}
\frac{dS}{dt} &= f\rho R - \alpha_1 Y_i S - \beta_1 Y_m S, \\
\frac{dE}{dt} &= \alpha_1 Y_i S + \beta_1 Y_m S - \eta_1 E + (1-f)\rho R, \\
\frac{dI}{dt} &= \eta_1 E - \eta_2 I, \\
\frac{dR}{dt} &= \eta_2 I - \rho R, \\
\frac{dX_e}{dt} &= rX_m - \gamma_1 X_e - d_1 X_e, \\
\frac{dX_r}{dt} &= \gamma_1 X_e - \gamma_2 X_r - d_2 X_r, \\
\frac{dX_i}{dt} &= \gamma_2 X_r - \alpha_2 X_i I - \gamma_3 X_i - d_3 X_i, \\
\frac{dX_m}{dt} &= \gamma_3 X_i - \beta_2 X_m I - d_4 X_m^2, \\
\frac{dY_i}{dt} &= \alpha_2 X_i I - \gamma_4 Y_i - d_3 Y_i, \\
\frac{dY_m}{dt} &= \gamma_4 Y_i + \beta_2 X_m I - d_4 Y_m.
\end{aligned} \tag{2}$$

From biological considerations, we study (2) in the closed set $\Gamma = \{(S, E, I, R, X_e, X_r, X_i, X_m, Y_i, Y_m) \in R^{10} \mid S, E, I, R \geq 0, S + E + I + R = 1, X_e, X_r, X_i, X_m, Y_i, Y_m \geq 0\}$, which is invariant set under nonnegative initial conditions. It is easy to proof the boundedness of the solutions of system (2). We omit it.

The system (2) always exists a disease-free equilibrium (DFE) $P_0 = (S^0, 0, 0, 0, X_e^0, X_r^0, X_i^0, X_m^0, 0, 0)$, where

$$S^0 = 1, \quad X_e^0 = \frac{r}{\gamma_1 + d_1} X_m^0, \quad X_r^0 = \frac{r\gamma_1}{(\gamma_1 + d_1)(\gamma_2 + d_2)} X_m^0,$$

$$X_i^0 = \frac{r\gamma_1\gamma_2}{(\gamma_1 + d_1)(\gamma_2 + d_2)(\gamma_3 + d_3)} X_m^0, \quad X_m^0 = \frac{r\gamma_1\gamma_2\gamma_3}{(\gamma_1 + d_1)(\gamma_2 + d_2)(\gamma_3 + d_3)d_4}.$$

2.2. The basic reproductive number R_0

The basic reproductive number R_0 of an infectious disease is a fundamental concept in the study of disease transmissions. It represents the average number of secondary cases produced, in a completely susceptible population, by a typical infective individual [28]. If $R_0 < 1$, then on average an infected individual produces less than one new infected individual over the course of its infectious period, and the infection cannot grow. Conversely, if $R_0 > 1$, then each infected individual produces, on average, more than one new infection, and the disease can invade the population [29]. Diekmann et al. [29] define R_0 as the spectral radius of the next generation matrix. That is, we rewrite the vector field of (2) as

$$\frac{dx_i}{dt} = \mathcal{F}_i(x) - \mathcal{V}_i(x), \quad i = 1, 2, \dots, 10.$$

and $\mathcal{V}_i(x) = \mathcal{V}^-_i(x) - \mathcal{V}^+_i(x)$, where $x = (E, I, R, Y_i, Y_m, S, X_e, X_r, X_i, X_m)$,

$$\mathcal{F}(x) = \begin{pmatrix} \alpha_1 Y_i S + \beta_1 Y_m S + (1-f)\rho R \\ 0 \\ 0 \\ \alpha_2 X_i I \\ \beta_2 X_m I \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V}(x) = \begin{pmatrix} \eta_1 E \\ \eta_2 I - \eta_1 E \\ \rho R - \eta_2 I \\ \gamma_4 Y_i + d_3 Y_i \\ d_4 Y_m - \gamma_4 Y_i \\ \alpha_1 Y_i S + \beta_1 Y_m S - f \rho R \\ \gamma_1 X_e + d_1 X_e - r X_m \\ \gamma_2 X_r + d_2 X_r - \gamma_1 X_e \\ \alpha_2 X_i I + \gamma_3 X_i + d_3 X_i - \gamma_2 X_r \\ \beta_2 X_m I + d_4 X_m^2 - \gamma_3 X_i \end{pmatrix}.$$

F, V are defined as

$$F = \left(\frac{\partial \mathcal{F}_i}{\partial x_j}(\hat{P}_0) \right)_{1 \leq i, j \leq 5}, \quad V = \left(\frac{\partial \mathcal{V}_i}{\partial x_j}(\hat{P}_0) \right)_{1 \leq i, j \leq 5},$$

where $\hat{P}_0 = (0, 0, 0, 0, 0, S^0, X_e^0, X_r^0, X_i^0, X_m^0)$. Thus

$$F = \begin{pmatrix} 0 & 0 & (1-f)\rho & \alpha_1 & \beta_1 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_2 X_i^0 & 0 & 0 & 0 \\ 0 & \beta_2 X_m^0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \eta_1 & 0 & 0 & 0 & 0 \\ -\eta_1 & \eta_2 & 0 & 0 & 0 \\ 0 & -\eta_2 & \rho & 0 & 0 \\ 0 & 0 & 0 & \gamma_4 + d_3 & 0 \\ 0 & 0 & 0 & -\gamma_4 & d_4 \end{pmatrix}.$$

Obviously, $-V$ is cooperative. By simple computation, we get

$$FV^{-1} = \begin{pmatrix} 1-f & 1-f & 1-f & \frac{\alpha_1}{\gamma_4 + d_3} + \frac{\beta_1 \gamma_4}{(\gamma_4 + d_3)d_4} & \frac{\beta_1}{d_4} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\alpha_2 X_i^0}{\eta_2} & \frac{\alpha_2 X_i^0}{\eta_2} & 0 & 0 & 0 \\ \frac{\beta_2 X_m^0}{\eta_2} & \frac{\beta_2 X_m^0}{\eta_2} & 0 & 0 & 0 \end{pmatrix}.$$

The eigenvalues of FV^{-1} are determined by

$$\lambda^3 \left[\lambda^2 - (1-f)\lambda - \left(\frac{\alpha_1}{\gamma_4 + d_3} + \frac{\beta_1 \gamma_4}{(\gamma_4 + d_3)d_4} \right) \frac{\alpha_2 X_i^0}{\eta_2} - \frac{\beta_2 X_m^0 \beta_1}{\eta_2 d_4} \right] = 0. \quad (3)$$

It is easy to obtain that the spectral radius of FV^{-1} is:

$$\rho(FV^{-1}) = R_0 = \frac{1-f}{2} + \sqrt{\frac{(1-f)^2}{4} + R_1 + R_2},$$

where

$$R_1 = \left(\frac{\alpha_1}{\gamma_4 + d_3} + \frac{\beta_1 \gamma_4}{(\gamma_4 + d_3)d_4} \right) \frac{\alpha_2}{\eta_2} X_i^0 \quad \text{and} \quad R_2 = \frac{\beta_1 \beta_2}{\eta_2 d_4} X_m^0. \quad (4)$$

3. Main results

3.1. An equivalent threshold quantity T_0

In order to give a more reasonable biological interpretation, we give an equivalent threshold quantity as

$$T_0 = R_1 + R_2 + 1 - f,$$

where R_1 and R_2 are defined in (4). The biological meaning of quantity T_0 can be explained as follows. Suppose that a single infected tree in stage E is introduced into a completely susceptible grove. The average number of secondary infections resulting from the psyllid in stage X_i (that is susceptible 3rd through 5th nymph) contact infected tree is,

$$R_1 = \left(\frac{\alpha_1}{\gamma_4 + d_3} + \frac{\beta_1 \gamma_4}{(\gamma_4 + d_3)d_4} \right) \frac{\alpha_2}{\eta_2} X_i^0.$$

Further, the average number of secondary infections resulting from the psyllid in stage X_m (that is susceptible adult psyllid) contact infected tree is,

$$R_2 = \frac{\beta_1 \beta_2}{\eta_2 d_4} X_m^0.$$

The citrus tree will necessarily be removed in the R stage and will on average produce $1 - f$ newly infected E trees. Thus the expected number of secondary infections is exactly T_0 . In the following, we will prove the equivalence of T_0 and R_0 for the local stability of the DFE P_0 .

Theorem 3.1. (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 (2) evaluated at DFE P_0 have negative real parts.

Proof. It follows from (3) that the basic reproductive number R_0 is the largest positive root of

$$\rho(\lambda) = \lambda^2 - (1-f)\lambda - \left(\frac{\alpha_1}{\gamma_4 + d_3} + \frac{\beta_1 \gamma_4}{(\gamma_4 + d_3)d_4} \right) \frac{\alpha_2 X_i^0}{\eta_2} - \frac{\beta_2 X_m^0 \beta_1}{\eta_2 d_4} = \lambda^2 - (1-f)\lambda - R_1 - R_2 = 0.$$

Clearly, the leading coefficient of $\rho(\lambda)$ is positive, and thus $R_0 < 1$ if and only if

$$\rho(1) = f - R_1 - R_2 > 0.$$

Therefore, $\rho(1) > 0$ is equivalent to $T_0 < 1$ and the first assertion holds.

Next, we want to prove the second assertion. Calculating the Jacobian matrix of system (2) at the DFE P_0 :

$$J(P_0) = \begin{pmatrix} \eta_1 & 0 & (1-f)\rho & \alpha_1 & \beta_1 & 0 & 0 & 0 & 0 & 0 \\ \eta_1 & \eta_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \eta_2 & \rho & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_2 X_i^0 & 0 & \gamma_4 + d_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_2 X_m^0 & 0 & \gamma_4 & d_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & f\rho & -\alpha_1 & -\beta_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma_1 + d_1 & 0 & 0 & r \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma_1 & \gamma_2 + d_2 & 0 & 0 \\ 0 & -\alpha_2 X_i^0 & 0 & 0 & 0 & 0 & 0 & \gamma_2 & \gamma_3 + d_3 & 0 \\ 0 & -\beta_2 X_m^0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_3 & 2d_4 X_m^0 \end{pmatrix}.$$

Clearly, $J(P_0)$ can be seen as a block matrix of 2×2 and each block is a 5×5 matrix. The eigenvalues are determined by the following characteristic equation of $J(P_0)$:

$$p(\lambda) = \lambda(\lambda + \gamma_1 + d_1)(\lambda + \gamma_2 + d_2)(\lambda + \gamma_3 + d_3)(\lambda + \gamma_4 + d_3)(\lambda + d_4)(\lambda + 2d_4 X_m^0) \\ \times [(\lambda + \eta_1)(\lambda + \eta_2)(\lambda + \rho) - \eta_1 \eta_2 (1-f)\rho] - 2\eta_1 r \gamma_1 \gamma_2 \gamma_3 \lambda (\lambda + \rho) \\ \times [\beta_1 \gamma_4 \alpha_2 X_i^0 - \alpha_1 \alpha_2 X_i^0 (\lambda + d_4) - \beta_1 \beta_2 X_m^0 (\lambda + d_4)(\lambda + \gamma_4 + d_3)] = 0.$$

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

$$q(\lambda) = \frac{p(\lambda)}{\lambda(\lambda + \eta_1)(\lambda + \eta_2)(\lambda + \rho)(\lambda + \gamma_1 + d_1)(\lambda + \gamma_2 + d_2)(\lambda + \gamma_3 + d_3)(\lambda + \gamma_4 + d_3)(\lambda + d_4)(\lambda + 2d_4 X_m^0)} \\ = 1 - \frac{2\eta_1 \beta_1 r \gamma_1 \gamma_2 \gamma_3 \gamma_4 \alpha_2 X_i^0}{(\lambda + \eta_1)(\lambda + \eta_2)(\lambda + \gamma_1 + d_1)(\lambda + \gamma_2 + d_2)(\lambda + \gamma_3 + d_3)(\lambda + \gamma_4 + d_3)(\lambda + d_4)(\lambda + 2d_4 X_m^0)} \\ - \frac{2\eta_1 \alpha_1 r \gamma_1 \gamma_2 \gamma_3 \alpha_2 X_i^0}{(\lambda + \eta_1)(\lambda + \eta_2)(\lambda + \gamma_1 + d_1)(\lambda + \gamma_2 + d_2)(\lambda + \gamma_3 + d_3)(\lambda + \gamma_4 + d_3)(\lambda + 2d_4 X_m^0)} \\ - \frac{2\eta_1 \beta_1 r \gamma_1 \gamma_2 \gamma_3 \beta_2 X_m^0}{(\lambda + \eta_1)(\lambda + \eta_2)(\lambda + \gamma_1 + d_1)(\lambda + \gamma_2 + d_2)(\lambda + \gamma_3 + d_3)(\lambda + 2d_4 X_m^0)} - \frac{\eta_1 \eta_2 (1-f)\rho}{(\lambda + \eta_1)(\lambda + \eta_2)(\lambda + \rho)}.$$

Obviously, $q(\lambda)$ is monotone increasing in λ when $\lambda > 0$. It follows from that, since the leading coefficient of $p(\lambda)$ is positive, we know that $p(\lambda)$ has no positive real roots if and only if $q(0) > 0$, which is equivalent to

$$\left(\frac{\alpha_1}{\gamma_4 + d_3} + \frac{\beta_1 \gamma_4}{(\gamma_4 + d_3) d_4} \right) \frac{\alpha_2}{\eta_2} X_i^0 + \frac{\beta_1 \beta_2}{\eta_2 d_4} X_m^0 + 1 - f < 1,$$

that is, $T_0 < 1$. Next, we need to prove all complex eigenvalues of $J(P_0)$ have negative real parts. Suppose the contrary. Then we define $G(\lambda) = 1 - q(\lambda)$ and suppose $p(\lambda) = 0$ with $Re(\lambda) \geq 0$ and $Im(\lambda) \neq 0$. Then $G(\lambda) = 1$ where

$$\begin{aligned} G(\lambda) = & \frac{2\eta_1\beta_1r\gamma_1\gamma_2\gamma_3\gamma_4\alpha_2X_i^0}{(\lambda+\eta_1)(\lambda+\eta_2)(\lambda+\gamma_1+d_1)(\lambda+\gamma_2+d_2)(\lambda+\gamma_3+d_3)(\lambda+\gamma_4+d_3)(\lambda+d_4)(\lambda+2d_4X_m^0)} \\ & + \frac{2\eta_1\alpha_1r\gamma_1\gamma_2\gamma_3\alpha_2X_i^0}{(\lambda+\eta_1)(\lambda+\eta_2)(\lambda+\gamma_1+d_1)(\lambda+\gamma_2+d_2)(\lambda+\gamma_3+d_3)(\lambda+\gamma_4+d_3)(\lambda+2d_4X_m^0)} \\ & + \frac{2\eta_1\beta_1r\gamma_1\gamma_2\gamma_3\beta_2X_m^0}{(\lambda+\eta_1)(\lambda+\eta_2)(\lambda+\gamma_1+d_1)(\lambda+\gamma_2+d_2)(\lambda+\gamma_3+d_3)(\lambda+2d_4X_m^0)} + \frac{\eta_1\eta_2(1-f)\rho}{(\lambda+\eta_1)(\lambda+\eta_2)(\lambda+\rho)}. \end{aligned} \quad (5)$$

We claim $|G(\lambda)| < G(Re(\lambda))$. From (5), we have

$$\begin{aligned} & |G(\lambda)| \\ \leq & \frac{2\eta_1\beta_1r\gamma_1\gamma_2\gamma_3\gamma_4\alpha_2X_i^0}{|\lambda+\eta_1||\lambda+\eta_2||\lambda+\gamma_1+d_1||\lambda+\gamma_2+d_2||\lambda+\gamma_3+d_3||\lambda+\gamma_4+d_3||\lambda+d_4||\lambda+2d_4X_m^0|} \\ & + \frac{2\eta_1\alpha_1r\gamma_1\gamma_2\gamma_3\alpha_2X_i^0}{|\lambda+\eta_1||\lambda+\eta_2||\lambda+\gamma_1+d_1||\lambda+\gamma_2+d_2||\lambda+\gamma_3+d_3||\lambda+\gamma_4+d_3||\lambda+2d_4X_m^0|} \\ & + \frac{2\eta_1\beta_1r\gamma_1\gamma_2\gamma_3\beta_2X_m^0}{|\lambda+\eta_1||\lambda+\eta_2||\lambda+\gamma_1+d_1||\lambda+\gamma_2+d_2||\lambda+\gamma_3+d_3||\lambda+2d_4X_m^0|} + \frac{\eta_1\eta_2(1-f)\rho}{|\lambda+\eta_1||\lambda+\eta_2||\lambda+\rho|} \\ < & \frac{2\eta_1\beta_1r\gamma_1\gamma_2\gamma_3\gamma_4\alpha_2X_i^0}{\Upsilon(Re(\lambda)+\gamma_1+d_1)(Re(\lambda)+\gamma_2+d_2)(Re(\lambda)+\gamma_3+d_3)(Re(\lambda)+\gamma_4+d_3)(Re(\lambda)+d_4)(Re(\lambda)+2d_4X_m^0)} \\ & + \frac{2\eta_1\alpha_1r\gamma_1\gamma_2\gamma_3\alpha_2X_i^0}{\Upsilon(Re(\lambda)+\gamma_1+d_1)(Re(\lambda)+\gamma_2+d_2)(Re(\lambda)+\gamma_3+d_3)(Re(\lambda)+\gamma_4+d_3)(Re(\lambda)+2d_4X_m^0)} \\ & + \frac{2\eta_1\beta_1r\gamma_1\gamma_2\gamma_3\beta_2X_m^0}{\Upsilon(Re(\lambda)+\gamma_1+d_1)(Re(\lambda)+\gamma_2+d_2)(Re(\lambda)+\gamma_3+d_3)(Re(\lambda)+2d_4X_m^0)} + \frac{\eta_1\eta_2(1-f)\rho}{\Upsilon(Re(\lambda)+\rho)} \\ = & G(Re(\lambda)), \end{aligned}$$

where $\Upsilon = (Re(\lambda) + \eta_1)(Re(\lambda) + \eta_2)$, and the second inequality is strict since $Im(\lambda) \neq 0$. Then we get $T_0 < 1 \Leftrightarrow q(0) > 0 \Leftrightarrow G(0) < 1$, which implies $G(Re(\lambda)) < 1$ since G is decreasing in λ . Thus $|G(\lambda)| < G(Re(\lambda)) < 1$. This contradicts $G(\lambda) = 1$. \square

3.2. Extinction of the disease when $R_0 < 1$

Theorem 3.2. If $R_0 < 1$, then the DFE P_0 of system (2) is globally attractive.

Proof. Note that $R_0 < 1$ implies that $f > 0$. Suppose $\limsup_{t \rightarrow \infty} E(t) = m > 0$. Then for every $\varepsilon > 0$ there exists $\tau_1 > 0$, such that

$$E(t) \leq m + \varepsilon, \quad \text{for all } t \geq \tau_1. \quad (6)$$

It follows from the third equation of system (2) and (6) that

$$\frac{dI(t)}{dt} \leq \eta_1(m + \varepsilon) - \eta_2I_2(t),$$

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

$$I(t) \leq \frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon, \quad (7)$$

for all $t \geq \tau_2$. It follows from the fourth equation of system (2) and (7) that

$$\frac{dR(t)}{dt} \leq \eta_2 \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right) - \rho R(t).$$

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

$$R(t) \leq \frac{\eta_2}{\rho} \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right) + \varepsilon, \quad \text{for all } t \geq \tau_3. \quad (8)$$

Further, from system (2), we have

$$\begin{aligned} \frac{dX_e}{dt} &= rX_m - \gamma_1 X_e - d_1 X_e, \\ \frac{dX_r}{dt} &= \gamma_1 X_e - \gamma_2 X_r - d_2 X_r, \\ \frac{dX_i}{dt} &\leq \gamma_2 X_r - \gamma_3 X_i - d_3 X_i, \\ \frac{dX_m}{dt} &\leq \gamma_3 X_i - d_4 X_m^2. \end{aligned} \quad (9)$$

Considering the auxiliary system of (9):

$$\begin{aligned} \frac{d\tilde{X}_e}{dt} &= r\tilde{X}_m - \gamma_1 \tilde{X}_e - d_1 \tilde{X}_e, \\ \frac{d\tilde{X}_r}{dt} &= \gamma_1 \tilde{X}_e - \gamma_2 \tilde{X}_r - d_2 \tilde{X}_r, \\ \frac{d\tilde{X}_i}{dt} &= \gamma_2 \tilde{X}_r - \gamma_3 \tilde{X}_i - d_3 \tilde{X}_i, \\ \frac{d\tilde{X}_m}{dt} &= \gamma_3 \tilde{X}_i - d_4 \tilde{X}_m^2. \end{aligned} \quad (10)$$

Clearly, (10) is a quasi-monotone system, and the unique positive equilibrium $(X_e^0, X_r^0, X_i^0, X_m^0)$ of system (10) is globally asymptotically stable if $R_0 < 1$. By comparison theorem in differential equations, we obtain that there exists $\tau_4 > \tau_3$ such that

$$X_e(t) \leq X_e^0 + \varepsilon, \quad X_r(t) \leq X_r^0 + \varepsilon, \quad X_i(t) \leq X_i^0 + \varepsilon, \quad X_m(t) \leq X_m^0 + \varepsilon, \quad (11)$$

for all $t \geq \tau_4$. Substituting (7) and (11) into the ninth equation of system (2), we have that

$$\frac{dY_i(t)}{dt} \leq \alpha_2(X_i^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right) - (\gamma_4 + d_3)Y_i(t),$$

for all $t \geq \tau_4$. Then there exists $\tau_5 > \tau_4$ such that

$$Y_i(t) \leq \frac{\alpha_2(X_i^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{\gamma_4 + d_3} + \varepsilon. \quad (12)$$

for all $t \geq \tau_5$. Substituting (7), (11) and (12) into the last equation of system (2), we get that

$$\frac{dY_m(t)}{dt} \leq \gamma_4 \left(\frac{\alpha_2(X_i^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{\gamma_4 + d_3} + \varepsilon \right) + \beta_2(X_m^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right) - d_4 Y_m(t)$$

for all $t \geq \tau_5$. Thus there exists $\tau_6 > \tau_5$ such that

$$Y_m(t) \leq \frac{\gamma_4 \left(\frac{\alpha_2(X_i^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{\gamma_4 + d_3} + \varepsilon \right) + \beta_2(X_m^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{d_4} + \varepsilon. \quad (13)$$

for all $t \geq \tau_6$. Now, substituting (8), (12) and (13) into the second equation of system (2), we get that for $t \geq \tau_6$

$$\begin{aligned} \frac{dE(t)}{dt} &\leq \alpha_1 \left(\frac{\alpha_2(X_i^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{\gamma_4 + d_3} + \varepsilon \right) \\ &+ \beta_1 \left(\frac{\gamma_4 \left(\frac{\alpha_2(X_i^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{\gamma_4 + d_3} + \varepsilon \right) + \beta_2(X_m^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{d_4} + \varepsilon \right) - \eta_1 E(t) \\ &+ (1 - f)\rho \left(\frac{\eta_2}{\rho} \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right) + \varepsilon \right). \end{aligned}$$

Then there exists $\tau_7 > \tau_6$ such that

$$\begin{aligned}
 E(t) &\leq \frac{\alpha_1}{\eta_1} \left(\frac{\alpha_2(X_i^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{\gamma_4 + d_3} + \varepsilon \right) \\
 &+ \frac{\beta_1}{\eta_1} \left(\frac{\gamma_4 \left(\frac{\alpha_2(X_i^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{\gamma_4 + d_3} + \varepsilon \right) + \beta_2(X_m^0 + \varepsilon) \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right)}{d_4} + \varepsilon \right) \\
 &+ (1-f) \frac{\rho}{\eta_1} \left(\frac{\eta_2}{\rho} \left(\frac{\eta_1(m + \varepsilon)}{\eta_2} + \varepsilon \right) + \varepsilon \right). \tag{14}
 \end{aligned}$$

for all $t \geq \tau_7$. Letting $\varepsilon \rightarrow 0$, the inequality (14) becomes

$$\begin{aligned}
 E(t) &\leq \frac{\alpha_1}{\eta_1} \frac{\alpha_2 X_i^0 \eta_1 m}{\eta_2(\gamma_4 + d_3)} + \frac{\beta_1}{\eta_1} \left(\frac{\gamma_4 \alpha_2 X_i^0 \eta_1 m}{\eta_2(\gamma_4 + d_3) d_4} + \frac{\beta_2 X_m^0 \eta_1 m}{\eta_2 d_4} \right) + (1-f) \frac{\eta_2 \eta_1 m}{\eta_1 \eta_2} \\
 &= \left(\frac{\alpha_1 \alpha_2 X_i^0}{\eta_2(\gamma_4 + d_3)} + \frac{\beta_1 \alpha_2 \gamma_4 X_i^0}{\eta_2 d_4(\gamma_4 + d_3)} + \frac{\beta_1 \beta_2 X_m^0}{\eta_2 d_4} + 1-f \right) m \\
 &= T_0 m
 \end{aligned}$$

It follows from Theorem 3.1 that, $R_0 < 1$ implies $T_0 < 1$. Thus $\limsup_{t \rightarrow \infty} E(t) < m$, a contradiction. So $m = 0$. Following (7), (8), (12)-(14) and the nonnegativity of the solutions, we have $\lim_{t \rightarrow \infty} E(t) = \lim_{t \rightarrow \infty} I(t) = \lim_{t \rightarrow \infty} R(t) = \lim_{t \rightarrow \infty} Y_i(t) = \lim_{t \rightarrow \infty} Y_m(t) = 0$. By the theory of asymptotically autonomous semiflows (see [30]), we have

$$\lim_{t \rightarrow \infty} S(t) = S^0, \quad \lim_{t \rightarrow \infty} X_e(t) = X_e^0, \quad \lim_{t \rightarrow \infty} X_r(t) = X_r^0, \quad \lim_{t \rightarrow \infty} X_i(t) = X_i^0, \quad \lim_{t \rightarrow \infty} X_m(t) = X_m^0.$$

Therefore all nonnegative solutions converge to the DFE P_0 . \square

3.3. Persistence of the disease when $R_0 > 1$

It follows from Theorem 3.1 that DFE is locally asymptotically stable as $R_0 < 1$, while DFE is unstable as $R_0 > 1$. Theorem 3.2 in subsection 3.2 illustrates the global stable result of DEF for the case $R_0 < 1$.

Theorem 3.3. If $R_0 > 1$, then the disease is uniformly persistent for system (2). That is, there is a positive constant $\varepsilon_0 > 0$, such that

$$\liminf_{t \rightarrow \infty} E(t) > \varepsilon_0, \quad \liminf_{t \rightarrow \infty} I(t) > \varepsilon_0, \quad \liminf_{t \rightarrow \infty} R(t) > \varepsilon_0, \quad \liminf_{t \rightarrow \infty} Y_i(t) > \varepsilon_0, \quad \liminf_{t \rightarrow \infty} Y_m(t) > \varepsilon_0. \tag{15}$$

Proof. Denote $\widetilde{K} = \{(S, E, I, R, X_e, X_r, X_i, X_m, Y_i, Y_m) \in R_+^{10}\}$, $K_0 = \{(S, E, I, R, X_e, X_r, X_i, X_m, Y_i, Y_m) \in \widetilde{K} : S \geq 0, E > 0, I > 0, R > 0, X_e \geq 0, X_r \geq 0, X_i \geq 0, X_m \geq 0, Y_i > 0, Y_m > 0\}$, and $\partial K_0 = \widetilde{K} \setminus K_0$. Let $u(t, t_0, x_0)$ be the unique solution of system (2) with the initial value $x_0 = (S_0, E_0, I_0, R_0, X_{e0}, X_{r0}, X_{i0}, X_{m0}, Y_{i0}, Y_{m0})$ at time t_0 .

Define poincaré map $P : \widetilde{K} \rightarrow \widetilde{K}$ associated with system (2) as follows:

$$P(x_0) = u(t_0 + 1, x_0), \quad \forall x_0 \in \widetilde{K}.$$

Set

$$M_\partial = \{x_0 \in \partial K_0 \mid P^m(x_0) \in \partial K_0, \forall m \in \mathbb{Z}_+\}.$$

We claim that

$$M_\partial = \{(S, 0, 0, 0, X_e, X_r, X_i, X_m, 0, 0) \mid S \geq 0, X_e \geq 0, X_r \geq 0, X_i \geq 0, X_m \geq 0\}.$$

Obviously, $\{(S, 0, 0, 0, X_e, X_r, X_i, X_m, 0, 0) \mid S \geq 0, X_e \geq 0, X_r \geq 0, X_i \geq 0, X_m \geq 0\} \subseteq M_\partial$. Next, We want to show

$$M_\partial \setminus \{(S, 0, 0, 0, X_e, X_r, X_i, X_m, 0, 0) \mid S \geq 0, X_e \geq 0, X_r \geq 0, X_i \geq 0, X_m \geq 0\} = \emptyset. \quad (16)$$

If (16) does not hold, then there exists a point $(S_0, E_0, I_0, R_0, X_{e0}, X_{r0}, X_{i0}, X_{m0}, Y_{i0}, Y_{m0}) \in M_\partial \setminus \{(S, 0, 0, 0, X_e, X_r, X_i, X_m, 0, 0) \mid S \geq 0, X_e \geq 0, X_r \geq 0, X_i \geq 0, X_m \geq 0\}$. Next, for the five initial values E_0, I_0, R_0, Y_{i0} , and Y_{m0} , we divided into four cases to discuss.

Case (i) One initial value is equal to zero, and the others are lager than zero. Without loss of generality, we choose $E_0 = 0, I_0 > 0, R_0 > 0, Y_{i0} > 0$ and $Y_{m0} > 0$. It is obvious that $S(t) > 0, Y_i(t) > 0, Y_m(t) > 0$ and $R(t) > 0$ for any $t > t_0$. Then from the second equation of system (2), we get $\frac{dE(t)}{dt}|_{t=t_0} = \alpha_1 Y_i(t_0)S(t_0) + \beta_1 Y_m(t_0)S(t_0) + (1-f)\rho R(t_0) > 0$. Thus, $(S, E, I, R, X_e, X_r, X_i, X_m, Y_i, Y_m) \notin \partial K_0$ for $0 < t - t_0 \ll 1$. This is a contradiction. The other subcases can be similarly proved.

Case (ii) Two initial values are equal to zero, and the others are larger than zero. Let $E_0 = I_0 = 0, R_0 > 0, Y_{i0} > 0$ and $Y_{m0} > 0$. It is obvious that $S(t) > 0, Y_i(t) > 0, Y_m(t) > 0$, and $R(t) > 0$ for any $t > t_0$. From the second equation of system (2), we get $\frac{dE(t)}{dt}|_{t=t_0} = \alpha_1 Y_i(t_0)S(t_0) + \beta_1 Y_m(t_0)S(t_0) + (1-f)\rho R(t_0) > 0$. So $E(t) > 0$ for $0 < t - t_0 \ll 1$. This implies that $I(t) > 0$ for $0 < t - t_0 \ll 1$. Therefore, we have $(S, E, I, R, X_e, X_r, X_i, X_m, Y_i, Y_m) \notin \partial K_0$ for $0 < t - t_0 \ll 1$. This is a contradiction. Similarly, we can prove the other subcases.

Case (iii) Three initial values are equal to zero, and the others are larger than zero. Set $E_0 = I_0 = R_0 = 0, Y_{i0} > 0$ and $Y_{m0} > 0$. Clearly, $S(t) > 0, Y_i(t) > 0$ and $Y_m(t) > 0$, for any $t > t_0$. It follows from the second equation of system (2) that $\frac{dE(t)}{dt}|_{t=t_0} = \alpha_1 Y_i(t_0)S(t_0) + \beta_1 Y_m(t_0)S(t_0) > 0$. So $E(t) > 0$ for $0 < t - t_0 \ll 1$. It follows from the third and fourth equations of system (2) that $I(t) > 0$ and $R(t) > 0$ for $0 < t - t_0 \ll 1$. Therefore, $(S, E, I, R, X_e, X_r, X_i, X_m, Y_i, Y_m) \notin \partial K_0$ for $0 < t - t_0 \ll 1$. This is a contradiction. Similarly, we can prove the other subcases.

Case (iv) Four initial values are equal to zero, and the other is larger than zero. Set $E_0 = I_0 = R_0 = Y_{i0} = 0$ and $Y_{m0} > 0$. It is easy to see that $S(t) > 0$ and $Y_m(t) > 0$ for all $t > t_0$. From the second equation of system (2), we can get $\frac{dE(t)}{dt}|_{t=t_0} = \alpha_1 Y_i(t_0)S(t_0) > 0$, for $0 < t - t_0 \ll 1$. So $E(t) > 0$ for $0 < t - t_0 \ll 1$. It follows from the third, fourth and ninth equations of system (2) that $I(t) > 0, R(t) > 0$ and $Y_i(t) > 0$ for $0 < t - t_0 \ll 1$. Thus, $(S, E, I, R, X_e, X_r, X_i, X_m, Y_i, Y_m) \notin \partial K_0$ for $0 < t - t_0 \ll 1$. This is a contradiction. Similarly, we can prove the other subcases.

Thus

$$M_\partial = \{(S, 0, 0, 0, X_e, X_r, X_i, X_m, 0, 0) \mid S \geq 0, X_e \geq 0, X_r \geq 0, X_i \geq 0, X_m \geq 0\}.$$

In the following, we proceed by contradiction to prove that there exists $\xi > 0$ such that

$$\limsup_{m \rightarrow \infty} d(P^m(x_0), P_0) \geq \xi, \quad \forall x_0 \in K_0, m \in \mathbb{Z}_+. \quad (17)$$

where $P_0 = (S^0, 0, 0, 0, X_e^0, X_r^0, X_i^0, X_m^0, 0, 0)$.

It follows from Theorem 2 in [31] that $R_0 > 1 \iff \rho(FV^{-1}) > 1 \iff \rho(\exp(F - V)) > 1$. Therefore, if $R_0 > 1$, we can choose $\varepsilon_1 > 0$ sufficiently small such that

$$\rho(\exp(F - V - M_{\varepsilon_1})) > 1, \quad (18)$$

where

$$M_{\varepsilon_1} = \begin{pmatrix} 0 & 0 & 0 & \alpha_1 \varepsilon_1 & \beta_1 \varepsilon_1 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_2 \varepsilon_1 & 0 & 0 & 0 \\ 0 & \beta_2 \varepsilon_1 & 0 & 0 & 0 \end{pmatrix}.$$

If (17) does not hold, then for any $\zeta > 0$, we have

$$\limsup_{m \rightarrow \infty} d(P^m(x_0), P_0) < \zeta, \quad \text{for some } x_0 \in K_0.$$

Without loss of generality, suppose that

$$d(P^m(x_0), P_0) < \zeta, \quad \forall \zeta > 0, \forall m \in \mathbb{Z}_+.$$

By the continuity of the solution with respect to initial values, we have that there exists sufficiently small $\varepsilon_1 > 0$ such that

$$\|u(t, P^m(x_0)) - u(t, P_0)\| \leq \varepsilon_1, \quad \forall t \in [t_0, t_0 + 1], \forall m \in \mathbb{Z}_+. \quad (19)$$

For any $t \geq t_0$, there exists an integer $l \in \mathbb{Z}_+$ such that $t - t_0 = l + \hat{t}$, where $\hat{t} \in [0, 1)$. It follows from (19) that

$$\|u(t, P^m(x_0)) - u(t, P_0)\| = \|u(t_0 + \hat{t}, P^{m+l}(x_0)) - u(t_0 + \hat{t}, P_0)\| \leq \varepsilon_1.$$

Therefore, we have

$$S(t) \geq S^0 - \varepsilon_1, \quad X_i(t) \geq X_i^0 - \varepsilon_1, \quad X_m(t) \geq X_m^0 - \varepsilon_1, \quad \text{for all } t \geq t_0. \quad (20)$$

From system (2) and inequality (20), we get

$$\begin{aligned}\frac{dE(t)}{dt} &\geq \alpha_1 Y_i(S^0 - \varepsilon_1) + \beta_1 Y_m(S^0 - \varepsilon_1) - \eta_1 E + (1-f)\rho R, \\ \frac{dI(t)}{dt} &= \eta_1 E - \eta_2 I, \\ \frac{dR(t)}{dt} &= \eta_2 I - \rho R, \\ \frac{dY_i(t)}{dt} &\geq \alpha_2(X_i^0 - \varepsilon_1)I - \gamma_4 Y_i - d_3 Y_i, \\ \frac{dY_m(t)}{dt} &\geq \gamma_4 Y_i + \beta_2(X_m^0 - \varepsilon_1)I - d_4 Y_m.\end{aligned}\tag{21}$$

Obviously, system (21) is a quasi-monotonic system. Consider the following comparison system:

$$\frac{d\hat{Z}(t)}{dt} = (F - V - M_{\varepsilon_1})\hat{Z}(t),\tag{22}$$

where $\hat{Z}(t) = (\hat{E}(t), \hat{I}(t), \hat{R}(t), \hat{Y}_i(t), \hat{Y}_m(t))^T$ and

$$F - V - M_{\varepsilon_1} = \begin{pmatrix} -\eta_1 & 0 & (1-f)\rho & \alpha_1(S^0 - \varepsilon_1) & \beta_1(S^0 - \varepsilon_1) \\ \eta_1 & -\eta_2 & 0 & 0 & 0 \\ 0 & \eta_2 & -\rho & 0 & 0 \\ 0 & \alpha_2(X_i^0 - \varepsilon_1) & 0 & -(\gamma_4 + d_3) & 0 \\ 0 & \beta_2(X_m^0 - \varepsilon_1) & 0 & \gamma_4 & -d_4 \end{pmatrix}$$

By [32], we know that there exists a positive vector v such that $\hat{Z}(t) = v \exp(\eta t)$ is a solution of system (22), where $\eta = \ln \rho(\exp(F - V - M_{\varepsilon_1}))$. From (18), we can get $\eta > 0$ and thus $\hat{Z}(t) \rightarrow \infty$ as $t \rightarrow \infty$, that is, $\hat{E}(t) \rightarrow \infty$, $\hat{I}(t) \rightarrow \infty$, $\hat{R}(t) \rightarrow \infty$, $\hat{Y}_i(t) \rightarrow \infty$ and $\hat{Y}_m(t) \rightarrow \infty$ as $t \rightarrow \infty$. According to the comparison theorem in differential equations, we can easily obtain that

$$E(t) \rightarrow \infty, \quad I(t) \rightarrow \infty, \quad R(t) \rightarrow \infty, \quad Y_i(t) \rightarrow \infty, \quad Y_m(t) \rightarrow \infty, \quad \text{as } t \rightarrow \infty$$

This contradicts with the boundedness of the solutions. Thus, we have proved that (17) holds and P is weakly uniformly persistent with respect to $(K_0, \partial K_0)$.

Obviously, the poincaré map P has a global attractor P_0 . P_0 is an isolated invariant set in \widetilde{K} and $W^s(P_0) \cap K_0 = \emptyset$, and it is acyclic in M_∂ . Every solution in M_∂ converges to P_0 . According to Zhao [33], we derive that P is uniformly persistent with respect to $(K_0, \partial K_0)$. This implies that the solution of system (2) is uniformly persistent with respect to $(K_0, \partial K_0)$, that is, (15) holds. \square

4. Optimal control problem

Optimal control theory has been used to explore optimal control strategies for various infectious diseases [34, 35, 36]. The purpose of this section is to seek an optimal integrated strategy to prevent

the spread of citrus HLB. In the following, we begin with the presentation of the optimal control problem for the transmission dynamics of HLB in order to derive nutrient solution injection, removal of infected trees and insecticide spraying strategies with minimal implementation cost. We will show that it is possible to implement control techniques while minimizing the cost of implementation of such measures.

In the host citrus trees population, the associated force of infections are reduced by factors of $(1 - u_1)$, where u_1 measures the precaution effort of nutrient solution injection. The control variable u_2 represents the removing of infected trees. The control variable u_3 shows the eradication effort of insecticide spraying. It follows that the reproduction rate of psyllid population (including egg, nymph, adult stages) is reduced by a factor of $(1 - u_3)$. Based on the assumptions and extensions mentioned above, system (2) with control strategy can be improved as following forms:

$$\begin{aligned}
 \frac{dS}{dt} &= f(\rho R + u_2 I) - \alpha_1(1 - u_1)Y_i S - \beta_1(1 - u_1)Y_m S, \\
 \frac{dE}{dt} &= \alpha_1(1 - u_1)Y_i S + \beta_1(1 - u_1)Y_m S - \eta_1(1 - u_1)E + (1 - f)(\rho R + u_2 I), \\
 \frac{dI}{dt} &= \eta_1(1 - u_1)E - \eta_2 I - u_2 I, \\
 \frac{dR}{dt} &= \eta_2 I - \rho R, \\
 \frac{dX_e}{dt} &= r(1 - u_3)X_m - \gamma_1 X_e - d_1 X_e - r_0 u_3 X_e, \\
 \frac{dX_r}{dt} &= \gamma_1 X_e - \gamma_2 X_r - d_2 X_r - r_0 u_3 X_r, \\
 \frac{dX_i}{dt} &= \gamma_2 X_r - \alpha_2 X_i I - \gamma_3 X_i - d_3 X_i - r_0 u_3 X_i, \\
 \frac{dX_m}{dt} &= \gamma_3 X_i - \beta_2 X_m I - d_4 X_m^2 - r_0 u_3 X_m, \\
 \frac{dY_i}{dt} &= \alpha_2 X_i I - \gamma_4 Y_i - d_3 Y_i - r_0 u_3 Y_i, \\
 \frac{dY_m}{dt} &= \gamma_4 Y_i + \beta_2 X_m I - d_4 Y_m - r_0 u_3 Y_m,
 \end{aligned} \tag{23}$$

subject to nonnegative initial conditions, here r_0 is a conversion rate.

For the optimal control problem of (23), we consider the control variables $u(t) = (u_1, u_2, u_3) \in U$ relative to the state variables $S, E, I, R, X_e, X_r, X_i, X_m, Y_i, Y_m$ where control variables are bounded and measured with

$$U = \{(u_1, u_2, u_3) \mid u_i \text{ is Lebsegue measurable, } 0 \leq u_i(t) \leq 1, t \in [0, T], i = 1, 2, 3\}, \tag{24}$$

where T represents the control period. Let V be the total number of psyllid population, that is, $V = X_e + X_r + X_i + X_m + Y_i + Y_m$. For the control problem, we now define the objective functional as

$$J(u_1, u_2, u_3) = \int_0^T \left(A_1 I + A_2 R + A_3 V + \frac{B_1}{2} u_1^2 + \frac{B_2}{2} u_2^2 + \frac{B_3}{2} u_3^2 \right) dt. \tag{25}$$

subject to the control system (23). The objective is to minimize the cost functional (25). That is, the goal is minimizing the number of infected trees, dead trees and psyllid populations and the cost of implementing the control, by using possible minimal control variables $u_i(t)$ ($i = 1, 2, 3$). We choose to model the control efforts via a linear combination of quadratic terms, $u_i^2(t)$ ($i = 1, 2, 3$). Further, the constants A_1, A_2, A_3 and B_1, B_2, B_3 represent a measure of the relative cost of the interventions over the interval $[0, T]$. In order to find an optimal control, u_1^*, u_2^*, u_3^* such that

$$J(u_1^*, u_2^*, u_3^*) = \min_U J(u_1, u_2, u_3). \quad (26)$$

where U is defined in (24) and subject to control system (23) with nonnegative initial conditions. Next, we use Pontryagin's Maximum Principle to solve this optimal control problem.

4.1. Existence of the control problem

Following the idea of [37], we prove firstly the existence of the optimal control problem.

Theorem 4.1. For the objective functional $J(u_1, u_2, u_3) = \int_0^T (A_1 I + A_2 R + A_3 V + \frac{B_1}{2} u_1^2 + \frac{B_2}{2} u_2^2 + \frac{B_3}{2} u_3^2) dt$, associated with model (23) defined in U , then there exists an optimal control $u^* = (u_1^*, u_2^*, u_3^*)$, such that $J(u_1^*, u_2^*, u_3^*) = \min_U J(u_1, u_2, u_3)$.

Proof. By Theorem III.4.1 from [37], we only need to check the following assumptions:

- (H_1) The set of controls and corresponding state variables is nonempty.
- (H_2) The control set U is convex and closed.
- (H_3) Right hand side of each equation in control problem (23) is continuous, bound above by a sum of the bounded control and state, and can be written as a linear function of U with coefficients depending on time and the state.
- (H_4) There exist constants $C_1, C_2 > 0$ and $\beta > 1$ such that the integrand of the objective functional $L(y, u, t)$ is concave and satisfies

$$L(y, u, t) \geq C_1(|U_1|^2 + |U_2|^2 + |U_3|^2)^{\frac{\beta}{2}} - C_2.$$

Obviously, the state variables and the set of control are bounded and nonempty which confirm (H_1). Note that the solutions are bounded, so the admissible control set is bounded and convex, which confirms (H_2). The system is bilinear in control variables, so it confirms (H_3) (since the solutions are bounded). The hypothesis (H_4) can be verified as

$$A_1 I + A_2 R + A_3 V + \frac{1}{2}(B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2) \geq C_1(|U_1|^2 + |U_2|^2 + |U_3|^2)^{\frac{\beta}{2}} - C_2,$$

where $C_1, C_2 > 0, A_1, A_2, A_3, B_1, B_2, B_3 > 0, B_4 > 0$ and $\beta > 0$. In view of the result given by Lukes [38], we have that there exists an optimal control strategy (u_1^*, u_2^*, u_3^*) minimizing $J(u_1, u_2, u_3)$. \square

Next we explore the minimal value of $J(u_1, u_2, u_3)$. To accomplish this, we define the Lagrangian L and Hamiltonian H for the optimal control problem (23) as

$$L(I, R, V, u_1, u_2, u_3) = A_1 I + A_2 R + A_3 V + \frac{1}{2}(B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2),$$

and

$$\begin{aligned}
H(X, U, \lambda) = & L(I, R, V, u_1, u_2, u_3) + \lambda_1[f(\rho R + u_2 I) - \alpha_1(1 - u_1)Y_i S - \beta_1(1 - u_1)Y_m S] \\
& + \lambda_2[\alpha_1(1 - u_1)Y_i S + \beta_1(1 - u_1)Y_m S - \eta_1(1 - u_1)E + (1 - f)(\rho R + u_2 I)] \\
& + \lambda_3[\eta_1(1 - u_1)E - \eta_2 I - u_2 I] + \lambda_4[\eta_2 I - \rho R] + \lambda_5[r(1 - u_3)X_m - \gamma_1 X_e - d_1 X_e - r_0 u_3 X_e] \\
& + \lambda_6[\gamma_1 X_e - \gamma_2 X_r - d_2 X_r - r_0 u_3 X_r] + \lambda_7[\gamma_2 X_r - \alpha_2 X_i I - \gamma_3 X_i - d_3 X_i - r_0 u_3 X_i] \\
& + \lambda_8[\gamma_3 X_i - \beta_2 X_m I - d_4 X_m^2 - r_0 u_3 X_m] + \lambda_9[\alpha_2 X_i I - \gamma_4 Y_i - d_3 Y_i - r_0 u_3 Y_i] \\
& + \lambda_{10}[\gamma_4 Y_i + \beta_2 X_m I - d_4 Y_m - r_0 u_3 Y_m],
\end{aligned} \tag{27}$$

where $X = (S, E, I, R, X_e, X_r, X_i, X_m, Y_i, Y_m)$, $U = (u_1, u_2, u_3)$ and $\lambda = (\lambda_1, \lambda_2, \lambda_3, \dots, \lambda_{10})$.

4.2. Optimal control solution

In this subsection, by using Pontryagin's Maximum Principle [39], we will obtain the optimal solution of the control system (23).

Let u_1^*, u_2^* and u_3^* represent the optimal solution of the control problem (26), then there exists a nontrivial vector function $\lambda(t) = (\lambda_1(t), \lambda_2(t), \lambda_3(t), \dots, \lambda_{10}(t))$ satisfying three equalities:

(i) the state equation

$$\frac{dx}{dt} = \frac{\partial H(t, u_1^*, u_2^*, u_3^*, \lambda(t))}{\partial \lambda},$$

(ii) the optimality condition

$$0 = \frac{\partial H(t, u_1^*, u_2^*, u_3^*, \lambda(t))}{\partial u},$$

(iii) the adjoint equation

$$\frac{d\lambda}{dt} = -\frac{\partial H(t, u_1^*, u_2^*, u_3^*, \lambda(t))}{\partial X}.$$

Now, we apply the necessary conditions to the Hamiltonian H given by (27). Following the results in [39], we can obtain the following conclusions.

Theorem 4.2. Let $\hat{y}^* = (\hat{S}^*, \hat{E}^*, \hat{I}^*, \hat{R}^*, \hat{X}_e^*, \hat{X}_r^*, \hat{X}_i^*, \hat{X}_m^*, \hat{Y}_i^*, \hat{Y}_m^*)$ be an optimal solution associated with the optimal control strategy $u^*(t) = (u_1^*(t), u_2^*(t), u_3^*(t))$ for the optimal control problem (26), then there exists adjoint variables λ_i , ($i = 1, 2, \dots, 10$) satisfying

$$\begin{aligned}
\frac{d\lambda_1(t)}{dt} &= (\lambda_1 - \lambda_2)[\alpha_1(1 - u_1)Y_i + \beta_1(1 - u_1)Y_m] \\
\frac{d\lambda_2(t)}{dt} &= (\lambda_2 - \lambda_3)\eta_1(1 - u_1) \\
\frac{d\lambda_3(t)}{dt} &= -A_1 - fu_2\lambda_1 - (1 - f)u_2\lambda_2 + (\eta_2 + u_2)\lambda_3 - \eta_2\lambda_4 + \alpha_2X_i\lambda_7 \\
&\quad + \beta_2X_m\lambda_8 - \alpha_2X_i\lambda_9 - \beta_2X_m\lambda_{10} \\
\frac{d\lambda_4(t)}{dt} &= -A_2 - f\rho\lambda_1 - (1 - f)\rho\lambda_2 + \rho\lambda_4 \\
\frac{d\lambda_5(t)}{dt} &= -A_3 + (\gamma_1 + d_1 + \gamma_0u_3)\lambda_5 - \gamma_1\lambda_6 \\
\frac{d\lambda_6(t)}{dt} &= -A_3 + (\gamma_2 + d_2 + \gamma_0u_3)\lambda_6 - \gamma_2\lambda_7 \\
\frac{d\lambda_7(t)}{dt} &= -A_3 + (\alpha_2I + \gamma_3 + d_3 + \gamma_0u_3)\lambda_7 - \gamma_3\lambda_8 - \alpha_2I\lambda_9 \\
\frac{d\lambda_8(t)}{dt} &= -A_3 - r(1 - u_3)\lambda_5 + (\beta_2I + 2d_4X_m + \gamma_0u_3)\lambda_8 - \beta_2I\lambda_{10} \\
\frac{d\lambda_9(t)}{dt} &= -A_3 + (\lambda_1 - \lambda_2)\alpha_1(1 - u_1)S + (\gamma_4 + d_3 + \gamma_0u_3)\lambda_9 - \gamma_4\lambda_{10} \\
\frac{d\lambda_{10}(t)}{dt} &= -A_3 + (\lambda_1 - \lambda_2)\beta_1(1 - u_1)S + (d_4 + \gamma_0u_3)\lambda_{10}
\end{aligned}$$

with transversality conditions

$$\lambda_i(T) = 0, \quad i = 1, 2, \dots, 10.$$

Further, the control u_1^*, u_2^*, u_3^* are given by

$$\begin{aligned}
u_1^* &= \max \left\{ \min \left\{ 1, \frac{(\lambda_2 - \lambda_1)(\alpha_1 \hat{Y}_i^* \hat{S}^* + \beta_1 \hat{Y}_m^* \hat{S}^*) + (\lambda_3 - \lambda_2)\eta_1 \hat{E}^*}{B_1} \right\}, 0 \right\}, \\
u_2^* &= \max \left\{ \min \left\{ 1, \frac{(\lambda_2 - \lambda_1)f \hat{I}^* + (\lambda_3 - \lambda_2)\hat{I}^*}{B_2} \right\}, 0 \right\}, \\
u_3^* &= \max \left\{ \min \left\{ 1, \frac{r \hat{X}_m^* \lambda_5 + \gamma_0 \hat{X}_e^* \lambda_5 + \gamma_0 \hat{X}_r^* \lambda_6 + \gamma_0 \hat{X}_i^* \lambda_7 + \gamma_0 \hat{X}_m^* \lambda_8 + \gamma_0 \hat{Y}_i^* \lambda_9 + \gamma_0 \hat{Y}_m^* \lambda_{10}}{B_3} \right\}, 0 \right\}.
\end{aligned} \tag{28}$$

Proof. To determine the adjoint equations and the transversality conditions we use the Hamiltonian (27). The adjoint system results from Pontryagin's Maximum Principle [39].

$$\frac{d\lambda_1(t)}{dt} = -\frac{\partial H}{\partial S}, \quad \frac{d\lambda_2(t)}{dt} = -\frac{\partial H}{\partial E}, \dots, \frac{d\lambda_{10}(t)}{dt} = -\frac{\partial H}{\partial Y_m},$$

with $\lambda_i(T) = 0$ ($i = 1, 2, \dots, 10$).

To obtain the characterization of the optimal control given by (28), solving the equations

$$\frac{\partial H}{\partial u_1} = 0, \quad \frac{\partial H}{\partial u_2} = 0, \quad \frac{\partial H}{\partial u_3} = 0,$$

on the interior of the control set and applying the property of the control space U , we can derive (28) holds. \square

5. Numerical simulations

In this section, we use firstly the model (2) to simulate the data on the number of infected citrus trees of Yuan Orchard from June, 2015 to December, 2015. Yuan Orchard is located in Ganzhou, China, which is one of our monitoring sites for citrus HLB. The infected rates of trees $I(t)$ are given in Table 1. Numerical simulation of $I(t)$ is shown in Figure 2. In order to carry out the numerical simulations, we need to estimate the model parameters. We get these parameter values in three ways: some parameter values ($\eta_1, \eta_2, r, \gamma_1, \gamma_2, \gamma_3, d_1, d_2, d_3, d_4, r_0$) are obtained from the literature; some parameter values (ρ, f) are estimated; and other parameter values ($\alpha_1, \alpha_2, \beta_1, \beta_2$) are fitted by the MATLAB tool fminsearch, which is fitted by calculating the minimum sum of square (MSS) (see [40]):

$$MSS = \sum_{i=1}^{11} (I(\text{data}_i) - I(i))^2.$$

Table 1. The values of $I(t)$.

Date	06/30	07/30	08/30	09/15	09/30	10/15
$I(t)$	0.0318	0.053	0.1327	0.1946	0.215	0.2778
Date	10/30	11/15	11/30	12/15	12/30	
$I(t)$	0.354	0.354	0.3717	0.3716	0.407	

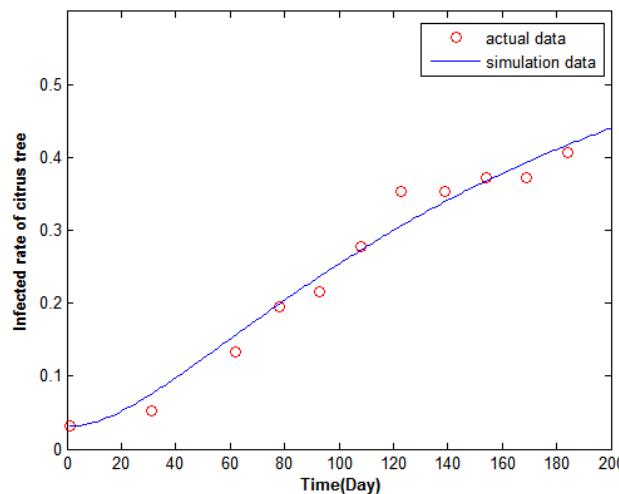


Figure 2. The little circle curves represent the values of the actual infected rates of trees $I(t)$. The solid curves are simulated by using the model (2). The values of parameters are given in Table 2.

By using the parameter values in Table 1, we can obtain $\alpha_1 = 0.00494 \text{ month}^{-1}$, $\alpha_2 = 0.00043 \text{ month}^{-1}$, $\beta_1 = 0.0097 \text{ month}^{-1}$ and $\beta_2 = 0.002258 \text{ month}^{-1}$ by fitting in simulations. All parameter values of the model are given in Table 2.

Table 2. Parameter values for the model (2).

Parameter	Value	Unit	Reference	Parameter	Value	Unit	Reference
η_1	0.1667	month ⁻¹	[41]	d_3	1.612	month ⁻¹	[9]
η_2	0.002258	month ⁻¹	[2]	d_4	0.788	month ⁻¹	[9, 42]
ρ	0.0791	-	Estimation	f	0.067	month ⁻¹	Estimation
r	62	month	[9, 42]	α_1	0.00494	month ⁻¹	Fitting
γ_1	5.49625	month ⁻¹	[9]	α_2	0.00043	month ⁻¹	Fitting
γ_2	5.376	month ⁻¹	[9]	β_1	0.0097	month ⁻¹	Fitting
γ_3, γ_4	2.188	month ⁻¹	[9]	β_2	0.002258	month ⁻¹	Fitting
d_1	2.494	month ⁻¹	[9]	r_0	6	-	[43]
d_2	4.867	month ⁻¹	[9]				

Next, we numerically examine the effect of the optimal control strategy on the spread of citrus HLB in a population of trees and psyllids. In this simulation without control population is labeled with bold line and the control by a dashed line. The weight constant values in the objective functional are $A_1 = 800$; $A_2 = 2000$; $A_3 = 2$; $B_1 = 200$; $B_2 = 1$; $B_3 = 50$. The control u_1 , u_2 and u_3 are all used to optimize the objective function J . Figures 3 and 4 showed that the control strategy resulted in a decrease in the number of infected citrus trees I , dead citrus trees R , psyllids at each stage, X_e , X_r , X_i , X_m , Y_i and Y_m while an increase is observed in the number of susceptible citrus trees S . In Fig. 5, we can observe that the optimal control profile for u_1 , u_2 and u_3 . Note that parameter values used in the numerical simulations are given in Table 1, and the initial conditions are taken as $S(0) = 0.6$, $E(0) = 0.1$, $I(0) = 0.2$, $R(0) = 0.1$, $X_e(0) = 100$, $X_r(0) = 60$, $X_i(0) = 60$, $X_m(0) = 50$, $Y_i(0) = 30$, $Y_m(0) = 20$.

6. Conclusion

In this paper, based on the mechanism and characteristics of citrus HLB transmission, we proposed a vector-borne plant disease model with stage structure in psyllids and studied the effect of intervention strategy in controlling the spread of HLB. We calculated the basic reproduction ratio R_0 for the epidemic model, and showed that the disease would die out when $R_0 < 1$, and the disease would be endemic when $R_0 > 1$.

Moreover, by using the optimal control theory, we analyzed the intervention strategy, nutrient solution injection, removal of infected trees and insecticide spraying, to determine the optimal integrated strategy. Using the Pontryagin's Maximum Principle, we investigated the existence of the optimal control problem. In addition, we minimized the number of infected citrus trees, dead citrus trees and the total number of psyllid population, by using three control variables. Numerical simulations illustrated the effectiveness of the proposed control problem.

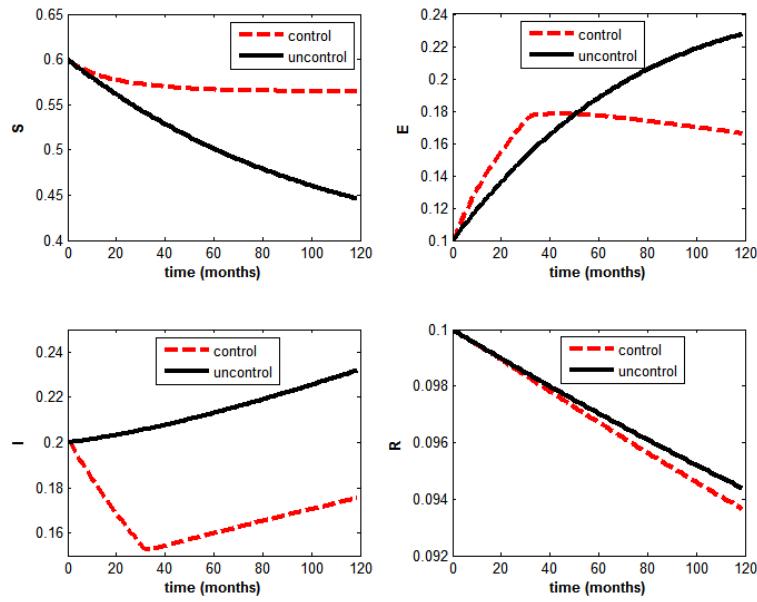


Figure 3. The plot represents population of susceptible, exposed, infected and dead citrus trees both with control and without control.

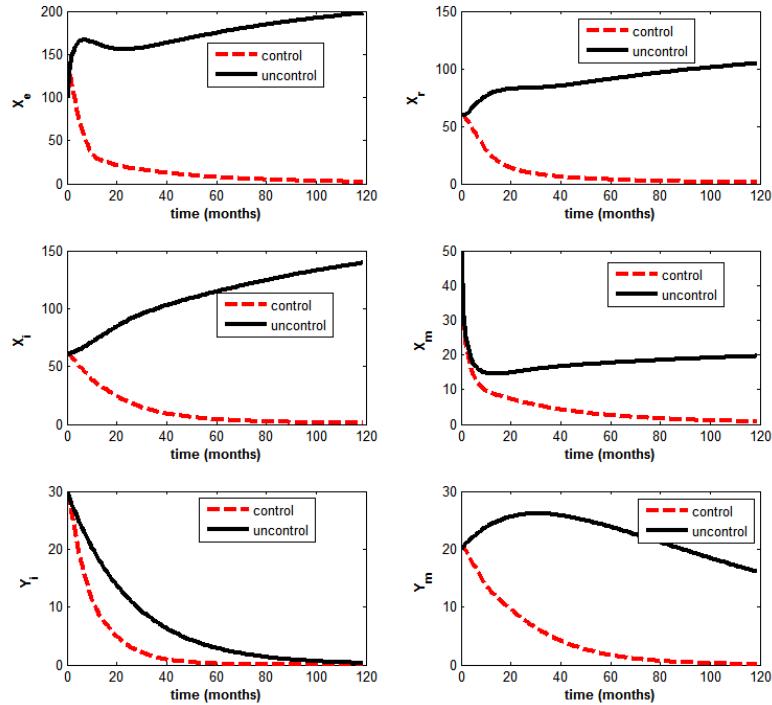


Figure 4. The plot represents different stages population of psyllid both with control and without control.

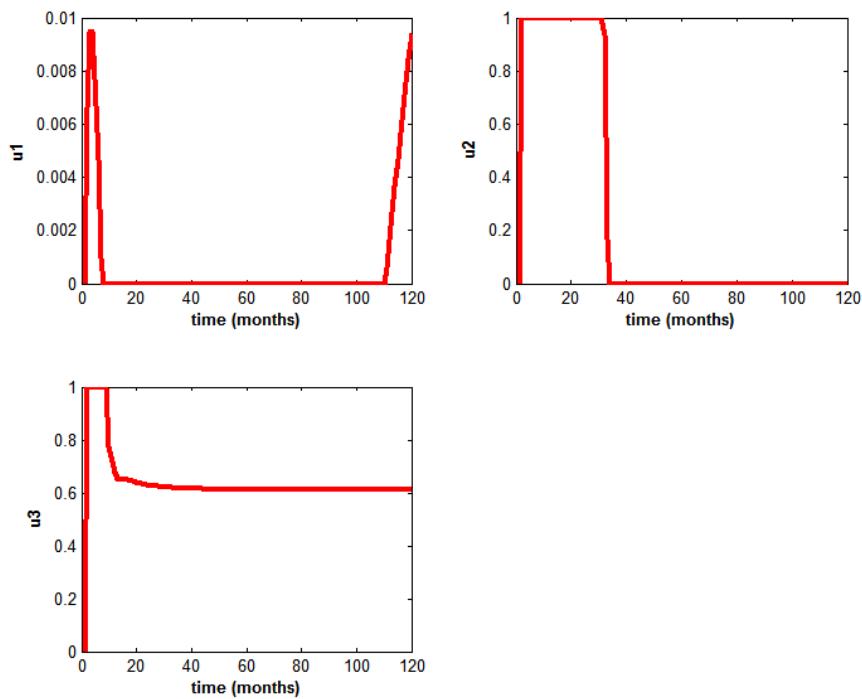


Figure 5. The plot represents the controls u_1 , u_2 and u_3 .

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Conflict of interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

References

1. D. Farnsworth, K. A. Grogan, A. H. C. V. Bruggen, et al., The potential economic cost and response to greening in Florida citrus, *Agric. Appl. Econ. Assoc.*, **29** (2014): 1–6.
2. J. M. Bove, Huanglongbing: A destructive, newly-emerging, century-old disease of citrus, *J. Plant. Pathol.*, **88** (2006): 7–37.
3. S. Alvarez, D. Solis and M. Thomas, Can Florida's citrus industry be saved while preserving the environment? An economic analysis for the bio-control of the Asian Citrus Psyllid, *S. Agric. Econ. Assoc.*, 2015.
4. H. Su, Research and health management of citrus Huanglongbing in Taiwan, *In: Proc Intern Research Conference on Huanglongbing*, Orlando, Florida, USA, (2008): 57–92.

5. A. J. Ayres, J. B. Jr and J. M. Bové, The experience with Huanglongbing management in Brazil, *Acta. Hortic.*, **1065** (2015): 55–62.
6. W. Li, Y. N. Yao, L. Wu, et al., Detection and seasonal variations of Huanglongbing disease in navel orange trees using direct ionization mass spectrometry, *J. Agric. Food. Chem.*, **67** (2019): 2265–2271.
7. C. Yang, H. Chen, H. Chen, et al., Antioxidant and anticancer activities of essential oil from Gannan navel orange peel, *Molecules*, **22** (2017): 1931–1400.
8. G. Rao, L. Huang, M. H. Liu, et al., Identification of Huanglongbing-infected navel oranges based on laser-induced breakdown spectroscopy combined with different Chemometric methods, *Appl. Optics*, **57** (2018): 8738–8742.
9. Y. H. Liu and J. H. Tsai, Effects of temperature on biology and life table parameters of the Asian citrus psyllid, *Diaphorina Citri* Kuwayama (Homoptera: Psyllidae), *Ann. Appl. Biol.*, **137** (2000): 201–206.
10. T. H. Hung, S. C. Hung, C. N. Chen, et al., Detection by PCR of *Candidatus Liberibacter asiaticus*, the bacterium causing citrus huanglongbing in vector psyllids: application to the study of vector pathogen relationships, *Plant Pathol.*, **53** (2004): 96–102.
11. F. Wu, Y. J. Cen, X. L. Deng, et al., Movement of *Diaphorina citri* (Hemiptera: Liviidae) adults between huanglongbing infected and healthy citrus, *Flor. Entomol.*, **98** (2015): 410–416.
12. W. Shen, S. E. Halbert, E. Dickstein, et al., Occurrence and ingrove distribution of citrus huanglongbing in north central Florida, *J. Plant. Pathol.*, **95** (2003): 361–371.
13. K. L. Manjunath, S. E. Halbert, C. Ramadugu, et al., Detection of *Candidatus Liberibacter asiaticus* in *Diaphorina citri* and its importance in the management of citrus huanglongbing in Florida, *Phytopathology*, **98** (2008): 387–396.
14. E. E. G. Cardwell, L. L. Stelinski and P. A. Stansly, Biology and management of Asian Citrus Psyllid, vector of the Huanglongbing pathogens, *Annu. Rev. Entomol.*, **58** (2013): 413–432.
15. X. Z. Meng and Z. Q. Li, The dynamics of plant disease models with continuous and impulsive cultural control strategies, *J. Theor. Biol.*, **266** (2010): 29–40.
16. X. S. Zhang and J. Holt, Mathematical models of cross protection in the epidemiology of plant-virus disease, *Anal. Theo. Plant. Pathol.*, **91** (2001): 924–934.
17. S. J. Gao, L. J. Xia, Y. Liu, et al., A plant virus disease model with periodic environment and pulse roguing, *Stud. Appl. Math.*, **136** (2016): 357–381.
18. S. Y. Tang, Y. N. Xiao and R. A. Cheke, Dynamical analysis of plant disease models with cultural control strategies and economic thresholds, *Math. Comput. Simul.*, **80** (2010): 894–921.
19. M. S. Chan and M. J. Jeger, An analytical model of plant virus disease dynamics with roguing and replanting, *J. Appl. Ecol.*, **31** (1994): 413–427.
20. C. Chiyaka, B. H. Singer, S. E. Halbert, et al., Modeling huanglongbing transmission within a citrus tree, *PNAS*, **109** (2012): 12213–12218.
21. R. A. Taylor, E. A. Mordecai, C. A. Gilligan, et al., Mathematical models are a powerful method to understand and control the spread of Huanglongbing, *Peer J*, **4** (2016): DOI 10.7717/peerj.2642.

22. K. Jacobsen, J. Stupiansky and S. S. Pilyugin, Mathematical modeling of citrus groves infected by Huanglongbing, *Math. Biosci. Eng.*, **10** (2013): 705–728.

23. R. G. dA. Vilamiu, S. Ternes, G. A. Braga, et al., A model for Huanglongbing spread between citrus plants including delay times and human intervention, *AIP. Conf. Proc.*, **1479** (2012): 2315–2319.

24. S. J. Gao, L. Luo, S. X. Yan, et al., Dynamical behavior of a novel impulsive switching model for HLB with seasonal fluctuations, *Complexity*, **2018** (2018): 1–11.

25. J.A. Lee, S. E. Halbert, W. O. Dawson, et al., Asymptomatic spread of Huanglongbing and implications for disease control, *Proc. Natl. Acad. Sci. USA.*, **112** (2015): 7605–7610.

26. M. Parry, G. J. Gibson, S. Parnell, et al., Bayesian inference for an emerging arboreal epidemic in the presence of control, *Proc. Natl. Acad. Sci. USA.*, **111** (2014): 6258–6262.

27. S. E. Halbert and K. L. Manjunath, Asian citrus psyllids (Sternorrhyncha: Psyllidae) and greening disease of citrus: A literature review and assessment of risk in Florida, *Flor. Entomol.*, **87** (2004): 330–353.

28. W. D. Wang and X. Q. Zhao, Threshold dynamics for compartmental epidemic models in periodic environments, *J. Dyn. Differ. Equ.*, **20** (2008): 699–717.

29. P. V. D. Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.*, **180** (2002): 29–48.

30. H. R. Thieme, Convergence results and poincare-bendixson trichotomy for asymptotically autonomous differential equations, *J. Math. Biol.*, **30** (1992): 755–763.

31. P. V. D. Driessche and J. Watmough, Reproductive numbers and subthreshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.*, **180** (2002): 29–48.

32. D. Xu and X. Q. Zhao, Dynamics in a periodic competitive model with stage structure, *J. Math. Anal. Appl.*, **311** (2005): 417–438.

33. X. Zhao, *Dynamical Systems in Population Biology*, Springer, New York, NY, USA, 2003.

34. T. K. Kar and A. Batabyal, Stability analysis and optimal control of an SIR epidemic model with vaccination, *Biosystems*, **104** (2011): 127–135.

35. L. Y. Pang, S. G. Ruan, S. H. Liu, et al., Transmission dynamics and optimal control of measles epidemics, *Appl. Math. Comput.*, **256** (2015): 131–147.

36. M. A. Khan, R. Khan, Y. Khan, et al., A mathematical analysis of Pine Wilt disease with variable population size and optimal control strategies, *Chaos, Solitons and Fract.*, **108** (2018): 205–217.

37. W. H. Fleming and R. W. Rishel, *Deterministic and Stochastic Optimal Control*, Springer-Verlag, New York, Berlin, 1975.

38. D. L. Lukes, *Differential equations: Classical to Controlled*, *Mathematics in Science and Engineering*, Academic Press, New York, 1982.

39. S. Lenhart and J. T. Workman, *Optimal control applied to biological models*, *Mathematical and Computational Biology Series*, Chapman & Hall/CRC Press, London/Boca Raton, 2007.

40. X. Zhang, Y. Zhao and A. Neumann, Partial immunity and vaccination for influenza, *J. Comput. Biol.*, **17** (2009): 1689–1696.

41. T. Gottwald, Current epidemiological understanding of citrus Huanglongbing, *Annu. Rev. Phytopathol.*, **48** (2010): 119–139.
42. K. S. P. Stelinski, R. H. Brlansky, T. A. Ebert, et al., Transmission parameters for *Candidatus Liberibacter asiaticus* by Asian citrus psyllid (Hemiptera: Psyllidae), *J. Econ. Entomol.*, **103** (2010): 1531–1541.
43. M. E. Rogers, General pest management considerations, *Citrus. Ind.*, **89** (2008): 12–15.



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