Analysis and optimal control of a Huanglongbing mathematical model with resistant vector

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Abstract

Huanglongbing (HLB) is an incurable disease that affects citrus trees. To better understand the transmission of HLB, the mathematical model is developed to investigate the transmission dynamics of the disease between Asian citrus psyllid (ACP) and citrus trees. Through rigorous mathematical derivations, we derive the expression of the basic reproduction number ($R_0$) of HLB. The findings show that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$, and if $R_0 > 1$ the system is uniformly persistent. By applying the global sensitivity analysis of $R_0$, we can obtain some parameters that have the greatest influence on the HLB transmission dynamics. Additionally, the optimal control theory is used to explore the corresponding optimal control problem of the HLB model. Numerical simulations are conducted to reinforce the analytical results. These theoretical and numerical results provide useful insights for understanding the transmission dynamics of HLB and may help policy makers to develop intervention strategies for the disease.

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1. Introduction

Huanglongbing (HLB) is one of the most destructive diseases of citrus, which is caused by the bacterium Candidatus Liberibacter asiaticus (CLas) and vectored by the Asian citrus psyllid (ACP) (Bové, 2006; Gottwald, 2010; Li et al., 2015). It is regarded as the most devastating citrus disease worldwide, and was first found in southern China in 1919, and now it is transmitted in fifty different countries (Bové, 2006; Wang & Trivedi, 2013). It causes substantial economic burdens to individual growers, citrus industries and governments (Taylor et al., 2016). Recently, citrus production has endured serious economic losses due to the occurrence of citrus HLB disease. This disease reduces fruit quality and yield, infringes on citrus health and has become one of the biggest challenges for citrus growers all over the world (Arredondo Valdés et al., 2016). The United States, Brazil and China, the world’s largest citrus producers, are all threatened by HLB disease. Up to now, there is still no effective method to eradicate HLB, and once orchards are infected with the disease, they are often destroyed due to low yields (McCullum & Baldwin, 2017). In view of

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the serious impact of HLB on the agricultural economy, it is of great importance to understand HLB transmission dynamics between citrus trees and ACP and to implement some effective intervention strategies to curb its transmission (Chiyaka et al., 2012).

Mathematical models have been playing a vital role in understanding HLB disease transmission dynamics and also in decision making processes regarding intervention mechanisms for the disease control (Chowdhury et al., 2019; Jackson & Chen-Charpentier, 2019; Jeger et al., 2018; Li et al., 2020; Meng & Li, 2010; Zhang et al., 2021; Zhang & Georgescu, 2015; Zhao et al., 2017). However, despite their significant impact on the agricultural economy, very few mathematical models have investigated how HLB disease is transmitted between the insect vector and the host (Taylor et al., 2016; Chiyaka et al., 2012; Lee et al., 2015; Vilamiu et al., 1479; Jacobsen et al., 2013; Gao et al., 2018; Khan et al., 2021). For the goal of highlighting the importance of flush for ACP dynamics, Chiyaka and Halbert (Chiyaka et al., 2012) formulated a mathematical model to study the spread of HLB within a tree because the infection psyllid transmits among the different flush patches on the tree. To explore the impact of seasonal fluctuations on HLB disease, Gao and Yu (Gao et al., 2018) built an impulsive switching model for the disease with seasonal fluctuations. Jacobsen and Stupiansky (Jacobsen et al., 2013) investigated a deterministic model for HLB transmission within a single citrus garden, which included the intervention strategy of roguing. Other further studies include Tu and Gao (Tu et al., 2019) developed a vector-borne disease model with stage structure and analyzed the effect of the measure in controlling the spread of HLB. We review those models that have been applied to HLB here because they show the main insights models have provided for this disease system.

Although some of the studies mentioned above took into account different mathematical models of HLB and disease intervention strategies, they did not consider the optimality of these intervention strategies and the resistance of the vector ACP, which may sometimes be limited by resource availability. In particular, a comparative analysis to understand the costs of different intervention strategies is important for decision makers who are often faced with the resource allocation challenges. In view of this, the application of optimal control theory can serve as a useful tool for assessing the effectiveness of various policies and interventions relative to the cost of implementing them (Alzahrani et al., 2021; Khan & Fatmawatic, 2021; Okosun et al., 2013). Based on these considerations, in this paper, we formulate a compartmental model to investigate HLB transmission dynamics with resistant ACP in a single orchard of citrus trees. Through extensive calculations, we obtain the basic reproduction number, i.e., the HLB disease below this threshold disappears but outbreaks above this threshold. The persistence of the system is further explored. Subsequently, optimal control theory is used to study the effectiveness and optimality of all possible combinations of two interventions for HLB disease, i.e., removing trees with HLB symptoms and spraying insecticides. We derive the optimal control conditions by using the Pontryagin’s Maximum Principle (Pontryagin, 2018). Furthermore, we obtain through numerical experiments that the optimal control strategy outperforms the constant intervention strategy in reducing the prevalence of the diseased citrus trees, and the cost of implementing the optimal control is far lower than the constant intervention strategy.

The remaining of this paper is organized as follows: the deterministic mathematical model is proposed to study the HLB transmission dynamics which incorporates ACP and citrus trees in Section 2. The basic reproduction number and local stability of the eight-dimensional HLB system are obtained in Section 3. Furthermore, the uniformly persistent of the nonlinear system are explored. The optimal control problem of the compartment model is conducted in Section 4. The global sensitivity and uncertainty analysis are performed, and some suggestions for HLB intervention strategies from the proposed model are summarized in Section 5. Finally, this paper ends with a brief conclusion and the potential outlook for future work in Section 6.

2. Model formulation

According to the work of (Hu et al., 2018), it was reported that trunk injection of penicillin, streptomycin and oxytetra-cycline hydrochloride, were effective in reducing the concentration of CLas and slowing down progress of HLB disease. In this paper, the model analyses the transmission dynamics of HLB with fraction of susceptible vaccinated citrus trees. Therefore, the citrus tree population given as \( N_h(t) \) is divided into susceptible individuals \( (S_h(t)) \), vaccinated individuals \( (V_h(t)) \), exposed individuals \( (E_h(t)) \), and infected individuals \( (I_h(t)) \), at any time \( t \). Thus, \( N_h(t) = S_h(t) + V_h(t) + E_h(t) + I_h(t) \). The ACP population, denoted by \( N_v(t) \), is divided into susceptible sensitive ACP \( (S_v(t)) \), susceptible resistant ACP \( (S_{rv}(t)) \), infected sensitive ACP \( (I_v(t)) \), and infected resistant ACP \( (I_{rv}(t)) \), at any time \( t \). Thus, \( N_v(t) = S_v(t) + S_{rv}(t) + I_v(t) + I_{rv}(t) \).

We assume that the recruitment to the citrus population is by replanting at a rate proportional to the difference between the actual number of citrus trees present \( N_h \) and maximum population size \( K \). Resistant classes \( S_{rv} \) and \( I_{rv} \) are increased by sensitive vectors \( S_v \) and \( I_v \) who develop resistance at the rates \( \varphi_1 \) and \( \varphi_2 \), respectively. Moreover, let \( \eta_1 \) the force of infection from ACP to trees and \( \eta_2 \) the force of infection from trees to ACP. The HLB model incorporating resistance for ACP population is represented as a system of first order nonlinear ordinary differential equations as follows:
\[
\begin{align*}
\frac{dS_h}{dt} &= r(K - N_h) + \omega_h V_h - \eta_h(t) S_h - (\nu_h + \mu_h) S_h, \\
\frac{dV_h}{dt} &= \eta_h S_h - (1 - \theta) \eta_h(t) V_h - (\omega_h + \mu_h) V_h, \\
\frac{dE_h}{dt} &= \eta_h(t)(S_h + (1 - \theta)V_h) - (\delta_h + \mu_h) E_h, \\
\frac{dI_h}{dt} &= \delta_h E_h - (\gamma_h + \mu_h) I_h, \\
\frac{dS_v}{dt} &= \Lambda_1 - \eta_v(t) S_v - (\varphi_1 + \mu_v) S_v, \\
\frac{dS_v}{dt} &= \Lambda_2 - \eta_v(t) S_v + \varphi_1 S_v - \mu_v S_v, \\
\frac{dI_v}{dt} &= \eta_v(t) S_v - (\varphi_2 + \mu_v) I_v, \\
\frac{dS_v}{dt} &= \eta_v(t) S_v + \varphi_2 I_v - \mu_v I_v.
\end{align*}
\]

where

\[\eta_h(t) = \frac{b_v(a_h I_{v1} + \beta_h I_{v2}(t))}{N_h(t)}, \quad \text{and} \quad \eta_v(t) = \frac{b_v(a_v E_h(t) + \beta_h I_h(t))}{N_h(t)}\]

A schematic diagram of the model is depicted in Fig. 1, and the state variables and the parameters are described in Table 1. The basic qualitative properties of the model (2.1) will be explored in the subsequent section.

3. Model analysis

To better organize the analysis, we simplify some terms in the model by setting \(d_1 = r + \mu_h, d_2 = \omega_h + \mu_h, d_3 = \nu_h + \mu_h, d_4 = \delta_h + \mu_h, d_5 = \gamma_h + \mu_h, d_6 = \varphi_1 + \mu_v, d_7 = \varphi_2 + \mu_v, d_8 = r + \nu_h + \mu_h\), and \(d_9 = \nu_h + \omega_h + \mu_h\). We denote

\[G_h = \left\{ (S_h, V_h, E_h, I_h) \in \mathbb{R}^4_+ : N_h \leq \frac{rK}{d_1} \leq N_h^0 \right\}\]
Lemma 3.1. Denote \( \Gamma(t) = (S_h(t), V_h(t), E_h(t), I_h(t), S_{v1}(t), S_{v2}(t), I_{v1}(t), I_{v2}(t))^T \). Let the initial data \( \Gamma(0) \gg 0 \). Then the solutions \( \Gamma(t) \) of the model (2.1) are non-negative for all \( t > 0 \). Furthermore

\[
\limsup_{t \to \infty} N_h(t) \leq N_h^0, \quad \lim_{t \to \infty} N_v(t) = N_v^0.
\]

The region \( G \subset \mathbb{R}_+^8 \) is positively-invariant for the model (2.1) with non-negative initial conditions.

Proof. Let the initial data of the model (2.1) \( \Gamma(0) \gg 0 \). It is obvious that \( \frac{dS_v}{dt} = a_{hv} V_v(t) - b_h \leq 0 \), so we have \( S_v(t) \geq 0 \), for all \( t > 0 \). Similarly, we can get \( V_h(t) \geq 0, E_h(t) \geq 0, I_h(t) \geq 0, S_{v1}(t) \geq 0, S_{v2}(t) > 0, I_{v1}(t) \geq 0 \) and \( I_{v2}(t) \geq 0 \), for all \( t > 0 \).

Adding the first four equations of model (2.1), the total number of citrus trees \( N_h \) satisfies

\[
\frac{dN_h}{dt} = r(K - N_h) - \mu_h N_h - \gamma_h I_h.
\]

thus,

\[
\frac{dN_h}{dt} \leq r(K - N_h) - \mu_h N_h.
\]

This implies that

\[
\limsup_{t \to \infty} N_h(t) \leq N_h^0.
\]

Adding the fifth to eighth equations of model (2.1), the total number of ACP \( N_v \) satisfies

\[
G_v = \left\{ (S_{v1}, S_{v2}, I_{v1}, I_{v2}) \in \mathbb{R}_+^4 : N_v \leq \frac{\Lambda_1 + \Lambda_2}{\mu_v} \leq N_v^0 \right\}, \quad G = G_h \times G_v \subset \mathbb{R}_+^8.
\]
\[
\frac{dN_r}{dt} = \Lambda_1 + \Lambda_2 - \mu_v N_r.
\]

thus,
\[
\lim_{t \to \infty} N_r(t) = N_r^0.
\]

Further, we know that \( \frac{dN_h}{dt} \leq 0 \) if \( N_h \geq N_h^0 \) and \( \frac{dN_v}{dt} \leq 0 \) if \( N_v \geq N_v^0 \). Therefore, the region \( G \) is positively-invariant. Clearly, the model (2.1) has a disease free equilibrium (DFE) point, \( E_0 = (S_h^0, Z_h^0, V_h^0, S_v^0, S_v^0, 0, 0) \), where
\[
S_h^0 = \frac{d_2 N_h^0}{d_5}, \quad V_h^0 = \frac{\nu_h N_h^0}{d_5 d_1}, \quad S_v^0 = \frac{\nu_v N_h^0}{d_5 d_1} \quad \text{and} \quad S_v^0 = \frac{\Lambda_1 v_1 + \Lambda_2}{\mu_v}.
\]

Denote
\[
k_1 = \frac{b_{r}a_{h}S_h^0}{N_h^0}, \quad k_2 = \frac{b_{r}a_{h}S_h^0}{N_h^0}, \quad k_3 = \frac{(1 - \theta)b_{r}a_{h}V_h^0}{N_h^0}, \quad k_4 = \frac{(1 - \theta)b_{r}a_{h}V_h^0}{N_h^0},
\]
\[
k_5 = \frac{b_{r}a_{h}S_v^0}{N_h^0}, \quad k_6 = \frac{b_{r}a_{h}S_v^0}{N_h^0}, \quad k_7 = \frac{b_{r}a_{h}S_v^0}{N_h^0}, \quad k_8 = \frac{b_{r}a_{h}S_v^0}{N_h^0}.
\]

Then the Jacobian matrix of system (2.1) with respect to \( E_0 \) is
\[
J(E_0) = 
\begin{pmatrix}
J_{11} & J_{12} & 0 & -J_{14} \\
0 & J_{22} & 0 & J_{24} \\
0 & -J_{32} & J_{33} & 0 \\
0 & J_{32} & 0 & J_{44}
\end{pmatrix},
\]

(3.1)

where
\[
J_{11} = \begin{pmatrix}
-d_8 & r + \omega_h \\
-r & -d_2
\end{pmatrix}, \quad J_{12} = \begin{pmatrix}
-r & -r \\
0 & 0
\end{pmatrix}, \quad J_{14} = \begin{pmatrix}
k_1 & k_2 \\
k_3 & k_4
\end{pmatrix}, \quad J_{22} = \begin{pmatrix}
d_4 & 0 \\
-d_4 & -d_9
\end{pmatrix},
\]
\[
J_{24} = \begin{pmatrix}
k_1 + k_3 & k_2 + k_4 \\
0 & 0
\end{pmatrix}, \quad J_{32} = \begin{pmatrix}
k_5 & k_6 \\
k_7 & k_8
\end{pmatrix}, \quad J_{33} = \begin{pmatrix}
-d_6 & 0 \\
\varphi_1 & -\mu_v
\end{pmatrix}, \quad J_{44} = \begin{pmatrix}
-d_7 & 0 \\
\varphi_2 & -\mu_v
\end{pmatrix}.
\]

Denote
\[
R_0 = \sqrt{\frac{(k_5 d_5 + k_6 \omega_h)(\mu_v (k_1 + k_3) + \varphi_2 (k_2 + k_4)) + (k_7 d_5 + k_8 \omega_h) (k_2 + k_4) d_7}{\mu_v d_4 d_5 d_7}}.
\]

(3.2)

To better organize the analysis, we denote
\[
p_0 = \mu_v d_4 d_5 d_7, \quad p_1 = k_5 d_5 \mu_v (k_1 + k_3), \quad p_2 = k_5 d_5 \varphi_2 (k_2 + k_4), \quad p_3 = k_6 \omega_h \mu_v (k_1 + k_3),
\]
\[
p_4 = k_6 \omega_h \varphi_2 (k_2 + k_4), \quad p_5 = k_7 d_5 (k_2 + k_4) d_7, \quad p_6 = k_8 \omega_h (k_2 + k_4) d_7, \quad \text{and}
\]

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Lemma 3.2. All eigenvalues of the matrix $J(E_0)$ (3.1) have negative real parts if and only if $R_0 < 1$.

Proof. The characteristic equation of $J_{11}$ is the following polynomial equation

$$\lambda^2 + (d_2 + d_8)\lambda + d_1d_9 = 0. \quad (3.3)$$

According to Vieta theorem, the two roots of equation (3.3) are negative. It is easy to see that two eigenvalue of $J_{33}$ are $-\mu_v$ and $-d_6$, which are negative. Denote

$$M = \begin{pmatrix} J_{22} & J_{24} \\ J_{52} & J_{44} \end{pmatrix} \quad (3.4)$$

We can see that all eigenvalues of the matrix $M$ are the roots of the following quartic polynomial equation

$$\lambda^4 + A\lambda^3 + B\lambda^2 + C\lambda + D = 0.$$ 

Obviously, if $R_0 < 1$, then $B > 0$, $C > 0$ and $D > 0$. The Hertz determinants of the first to fourth order polynomial are as follows
\[ \Delta_1 = A > 0, \]
\[ \Delta_2 = AB - C \]
\[ = Q_3 \left( 1 - R_0^2 \right) + Q_2 + Q_4, \]
\[ \Delta_3 = (AB - C)C - A^2D \]
\[ = Q_3 \left( \frac{p_0}{\mu} + \frac{p_0}{d_7} + \frac{p_0}{d_5} \right) \left( 1 - R_0^2 \right)^2 + Q_6 \left( 1 - R_0^2 \right) + (Q_2 + Q_4) \left( \frac{p_0}{d_4} + Q_1 \right). \]
\[ \Delta_4 = D\Delta_3. \]

It is obvious that \( \Delta_2 > 0, \Delta_3 > 0 \) and \( \Delta_4 > 0 \) if \( R_0 < 1 \). By the criterion of Routh–Hurwitz, we obtain that all eigenvalues of \( M \) have negative real parts if and only if \( R_0 < 1 \). Therefore all eigenvalues of \( f(E_0) \) have negative real parts if \( R_0 < 1 \).

**Lemma 3.2** implies the following result holds.

**Theorem 3.1.** For system (2.1), the disease-free equilibrium \( E_0 \) is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

In order to analyze the permanence of system (2.1), we first give the following lemma.

**Lemma 3.3.** (See [Zhao, 2003]) Assume that

1. \( f(X_0) \subset X_0 \) and \( f \) has a global attractor \( \Omega \).
2. The maximal compact invariant set \( \Omega_0 = \Omega \cap M_0 \) off in \( \partial X_0 \), possibly empty, has an acyclic covering \( \tilde{M} = \{M_1, \ldots, M_k\} \) with the following properties:
   1. \( M_i \) is isolated in \( X \).
   2. \( W^i(M_i) \cap X_0 = \emptyset \) for each \( 1 \leq i \leq k \).

Then \( f \) is uniformly persistent with respect to \( (X_0, \partial X_0) \), i.e., there is \( \eta > 0 \) such that for any compact internally chain transitive set \( L \) with \( L \subset M_i \) for all \( 1 \leq i \leq k \), \( \inf_{x \in L} d(x, \partial X_0) > \eta \).

**Theorem 3.2.** If \( R_0 > 1 \), then system (2.1) is uniformly persistent.

**Proof.** We define \( X = \{(S_h, V_h, E_h, I_h, S_{v1}, S_{v2}, I_{v1}, I_{v2}) \in \mathbb{R}^8 : S_h \geq 0, V_h \geq 0, E_h \geq 0, I_h \geq 0, S_{v1} \geq 0, S_{v2} \geq 0, I_{v1} \geq 0, I_{v2} \geq 0\}, \)
\[ X_0 = \{(S_h, V_h, E_h, I_h, S_{v1}, S_{v2}, I_{v1}, I_{v2}) \in X : E_h > 0, I_h > 0, I_{v1} > 0, I_{v2} > 0\}, \)
and \( \partial X_0 = X \times X_0 \).

In order to show that system (2.1) is uniformly persistent, we only need to prove that \( \partial X_0 \) repels uniformly the solutions of \( X_0 \). Obviously, both \( X \) and \( X_0 \) are positively invariant and \( \partial X_0 \) is relatively closed in \( X \) and (2.1) is point dissipative. Let
\[ M_3 = \{(S_h(t), V_h(t), E_h(t), I_h(t), S_{v1}(t), S_{v2}(t), I_{v1}(t), I_{v2}(t)) \in \partial X_0 : (S_h(t), V_h(t), E_h(t), I_h(t), S_{v1}(t), S_{v2}(t), I_{v1}(t), I_{v2}(t)) \in \partial X_0, \forall t \geq 0 \} \]

We now show that
\[ M_3 = \{(S_h(t), V_h(t), 0, 0, S_{v1}(t), S_{v2}(t), 0, 0) \in X : S_h(t) \geq 0, V_h(t) \geq 0, S_{v1}(t) \geq 0, S_{v2}(t) \geq 0\} \]
\[ (3.5) \]

Assume \( (S_h(t), V_h(t), E_h(t), I_h(t), S_{v1}(t), S_{v2}(t), I_{v1}(t), I_{v2}(t)) \in M_3 \). It suffices to show that \( E_h(t) = 0, I_h(t) = 0, I_{v1}(t) = 0 \) and \( I_{v2}(t) = 0 \) for all \( t \geq 0 \). Suppose not. Then there exists a \( t_0 > 0 \) such that \( E_h(t_0), I_h(t_0), I_{v1}(t_0), I_{v2}(t_0) \) at least one is greater than 0. Without loss of generality, we only discuss the case \( E_h(t_0) > 0, S_h(t_0) = 0, V_h(t_0) = 0, I_h(t_0) = 0, S_{v1}(t_0) = 0, S_{v2}(t_0) = 0, I_{v1}(t_0) = 0 \) and \( I_{v2}(t_0) = 0 \). Since
\[ \frac{dS_{h}}{dt}|_{t=t_0} = \delta h E_h(t_0) > 0, \quad \frac{dS_{v1}}{dt}|_{t=t_0} = \Lambda_1 > 0, \quad \frac{dS_{v2}}{dt}|_{t=t_0} = \Lambda_2 > 0, \quad \frac{dE_{h}}{dt}|_{t=t_0} > -d_4 E_h, \]
it follows that there is an \( \epsilon_0 > 0 \) small enough such that \( S_{y_1}(t) > 0, S_{y_2}(t) > 0, E_0(t) > 0 \) and \( I_0(t) > 0 \) for all \( t_0 < t < t_0 + \epsilon_0 \). Furthermore, let \( t_1 = t_0 + \frac{\epsilon_0}{2} \), then we have \( S_{y_1}(t_1) > 0, S_{y_2}(t_1) > 0, E_0(t_1) > 0 \) and \( I_0(t_1) > 0 \). If \( I_{y_1}(t_1) > 0 \), then

\[
\frac{dI_{y_1}}{dt}_{|t=t_1} > -(\varphi_2 + \mu_v)I_{y_1},
\]

this implies that \( I_{y_1}(t) > 0 \) for all \( t \geq t_1 \). If \( I_{y_1}(t_1) = 0 \), we get

\[
\frac{dI_{y_1}}{dt}_{|t=t_1} = -\eta_v(t_1)S_{y_1}(t_1) > 0,
\]

It then follows that there is an \( \epsilon_1 (0 < \epsilon_1 < \frac{\epsilon_0}{2}) \) such that \( I_{y_1}(t) > 0 \) for all \( t_1 < t < t_1 + \epsilon_1 \). Similarly, we can show that there exists an \( \epsilon_2 (0 < \epsilon_2 < \frac{\epsilon_0}{2}) \) such that \( I_{y_2}(t) > 0 \) for all \( t \in (t_1 + \frac{\epsilon_1}{2}, t_1 + \frac{\epsilon_1}{2} + \epsilon_2) \), respectively. Thus, for all \( t_1 + \frac{\epsilon_1}{2} < t < t_1 + \frac{\epsilon_1}{2} + \epsilon_2 \), we have \( E_0(t) > 0, I_0(t) > 0, I_{y_1}(t) > 0 \) and \( I_{y_2}(t) > 0 \). This contradicts the assumption that \( (S_0(0), V_0(0), E_0(0), I_0(t), S_{y_1}(0), S_{y_2}(0), I_{y_1}(0), I_{y_2}(0)) \notin M_0 \). Thus (3.5) holds.

It is obvious that \( E_0 = (S_0^0, V_0^0, 0, 0, S_{y_1}^0, S_{y_2}^0, 0, 0) \) is the unique equilibrium in \( M_0 \). We now demonstrate that \( E_0 \) repels the solutions in \( X_0 \).

By the proof of Lemma 3.2, we know that \( det(M) < 0 \) provided that \( R_0 > 1 \), here \( M \) is defined in (3.4). Therefore, if \( R_0 > 1 \), then there exist \( \zeta_1 > 0 \) and \( \zeta_2 > 0 \) small enough such that

\[
\begin{align*}
(k_3d_5 + k_6\delta h)(\mu_v(k_1 + k_3) + \varphi_2(k_2 + k_4)) + (k_5d_5 + k_8\delta h)(k_2 + k_4)d_7 &> \mu_v d_4 d_5 d_7, \quad (3.6) \\
\det(M) = \det\begin{bmatrix} J_{22} & J_{23} & J_{24} & J_{25} \\ J_{26} & J_{27} & J_{28} & J_{29} \\ J_{30} & J_{31} & J_{32} & J_{33} \\ J_{34} & J_{35} & J_{36} & J_{37} \end{bmatrix} &< 0, \quad (3.7)
\end{align*}
\]

and

\[
\begin{align*}
\dot{S}_0^0 &= \frac{d^2rK + (1 - \theta)\eta_hrK - 2d_2\tilde{\zeta}_2 - 2r(1 - \theta)\eta_h\tilde{\zeta}_2}{d_1d_9 + (1 - \theta)\eta_hd_8 + d_2\eta_h + (1 - \theta)\eta_h^2} > S_0^0 - \zeta_1, \\
\dot{V}_0^0 &= \frac{\nu h rK - 2r\nu h \tilde{\zeta}_2}{d_1d_9 + (1 - \theta)\eta_hd_8 + d_2\eta_h + (1 - \theta)\eta_h^2} > V_0^0 - \zeta_1, \\
\dot{S}_{y_1}^0 &= \frac{\Lambda_1}{\eta_v + \varphi_1 + \mu_v} > S_{y_1}^0 - \zeta_1, \\
\dot{S}_{y_2}^0 &= \frac{\Lambda_2 + \varphi_1 S_{y_1}^0}{\eta_v + \mu_v} > S_{y_2}^0 - \zeta_1,
\end{align*}
\]

hold, where

\[
\begin{align*}
\dot{k_1} &= \frac{b_{m_0}}{N_v}(S_0^0 - \zeta_1), \\
\dot{k_2} &= \frac{b_0}{N_v}(S_0^0 - \zeta_1), \\
\dot{k_3} &= \frac{(1 - \theta)b_0}{N_v}(V_0^0 - \zeta_1), \\
\dot{k_4} &= \frac{(1 - \theta)b_0}{N_v}(V_0^0 - \zeta_1), \\
\dot{k_5} &= \frac{b_{m_0}}{N_v}(S_1^0 - \zeta_1), \\
\dot{k_6} &= \frac{b_{m_0}}{N_v}(S_2^0 - \zeta_1), \\
\dot{k_7} &= \frac{b_{m_0}}{N_v}(S_0^0 - \zeta_1), \\
\dot{k_8} &= \frac{b_0}{N_v}(S_0^0 - \zeta_1), \\
\dot{\eta}_h &= \frac{k^K - \gamma \tilde{\zeta}_2}{d_7}, \\
\dot{\eta}_v &= \frac{b_{m_0} \delta_v + b_0 \delta_v}{N_v},
\end{align*}
\]

and

\[
\begin{pmatrix} J_{24} \\ J_{26} \\ J_{30} \\ J_{34} \end{pmatrix} = \begin{pmatrix} k_1 & k_3 & k_4 & 0 \\ k_3 & k_6 & k_7 & 0 \\ k_5 & k_6 & k_8 & 0 \\ k_5 & k_6 & 0 & 0 \end{pmatrix}, \quad \begin{pmatrix} J_{32} \end{pmatrix} = \begin{pmatrix} k_3 & k_6 & k_7 & 0 \end{pmatrix}
\]

Suppose \( (S_0(t), V_0(t), E_0(t), I_0(t), S_{y_1}(t), S_{y_2}(t), I_{y_1}(t), I_{y_2}(t)) \) is a solution of system (2.1) with initial value \( (S_0(0), V_0(0), E_0(0), I_0(0), S_{y_1}(0), S_{y_2}(0), I_{y_1}(0), I_{y_2}(0)) \) \( \in X_0 \). We now claim that

\[
\limsup_{t \to \infty} \{E_0(t), I_0(t), I_{y_1}(t), I_{y_2}(t)\} > \zeta_2. \quad (3.9)
\]

Suppose that the claim is not valid. Then there is a \( T_0 > 0 \) such that
\[ E_h(t) \leq \zeta_2, \quad I_h(t) \leq \zeta_2, \quad I_{v1}(t) \leq \zeta_2, \quad I_{v2}(t) \leq \zeta_2, \quad \text{for all } t \geq T_0. \] (3.10)

It follows from system (2.1) and (3.10) that
\[
\frac{dN_h}{dt} \geq r(K - N_h) - \mu_h N_h - \gamma_h \zeta_2. 
\]
Thus, there exists a \( T_1 > T_0 \) such that
\[ N_h > \frac{rK - \gamma_h \zeta_2}{d_1} = N^*_h \quad \text{for all } t \geq T_1. \] (3.11)

From the first and second equations of the model (2.1) and (3.11), we have
\[
\begin{align*}
\frac{dS_h}{dt} & \geq r(K - S_h - V_h - 2\zeta_2) + \omega_h V_h - \bar{\eta}_h S_h - d_3 S_h, \\
\frac{dV_h}{dt} & \geq \nu_h S_h - (1 - \theta) \bar{\eta}_h V_h - d_2 V_h.
\end{align*}
\]
for \( t \geq T_1 \). Consider the following comparison system
\[
\begin{align*}
\frac{d\tilde{S}_h}{dt} & = r(K - \tilde{S}_h - \tilde{V}_h - 2\zeta_2) + \omega_h \tilde{V}_h - \bar{\eta}_h \tilde{S}_h - d_3 \tilde{S}_h, \\
\frac{d\tilde{V}_h}{dt} & = \nu_h \tilde{S}_h - (1 - \theta) \bar{\eta}_h \tilde{V}_h - d_2 \tilde{V}_h.
\end{align*}
\] (3.12)

We can restrict \( \zeta_2 \) to be small enough such that system (3.12) admits a unique positive equilibrium \((\tilde{S}_h^0, \tilde{V}_h^0)\), which is globally asymptotically stable (the proof is stated in Appendix A). By the comparison principle in differential equations and (3.8), there is a \( T_2 > 0 \) such that
\[ S_h(t) \geq S_h^0 - \zeta_1, \quad V_h(t) \geq V_h^0 - \zeta_1, \quad \text{for } t > T_2. \] (3.13)

From the fifth equation of system (2.1) and (3.11), we get
\[
\frac{dS_{v1}}{dt} \geq \Lambda_1 - \bar{\eta}_v S_{v1} - (\varphi_1 + \mu_v) S_{v1},
\]
for \( t \geq T_1 \). According to the comparison principle and (3.8), there exists a \( T_3 > T_2 \), such that
\[ S_{v1}(t) > S_{v1}^0 - \zeta_1, \quad \text{for } t \geq T_3. \] (3.14)

It follows from the sixth equation of system (2.1), (3.11) and (3.14) that
\[
\frac{dS_{v2}}{dt} \geq \Lambda_2 - \bar{\eta}_v S_{v2} + \varphi_1 S_{v1}^0 - \mu_v S_{v2},
\]
for \( t \geq T_3 \). Then, there exists a \( T_4 > T_3 \), such that
\[ S_{v2}(t) > S_{v2}^0 - \zeta_1, \quad \text{for } t \geq T_4. \] (3.15)

Consequently, from system (2.1) and (3.13)–(3.15), we obtain that for \( t > T_4 \),
\[
\begin{aligned}
\frac{dE_h}{dt} &= \frac{b_h(\alpha_h I_0 + \beta_h I_2)}{N_h^0} \left((S_h^0 - \zeta_1) + (1 - \theta)(V_h^0 - \zeta_1)\right) - d_4 E_h, \\
\frac{dh}{dt} &\geq \delta_h E_h - d_5 h, \\
\frac{dI_1}{dt} &\geq \frac{b_h(\alpha_h E_h + \beta_h I_1)}{N_h^0} (S_{I_1}^0 - \zeta_1) - (\varphi_2 + \mu_r) I_1, \\
\frac{dI_2}{dt} &\geq \frac{b_h(\alpha_h E_h + \beta_h I_2)}{N_h^0} (S_{I_2}^0 - \zeta_1) + \varphi_2 I_1 - \mu_r I_2.
\end{aligned}
\]

Consider the following auxiliary system:

\[
\begin{aligned}
\frac{dE_h}{dt} &= \frac{b_h(\alpha_h I_0 + \beta_h I_2)}{N_h^0} \left((S_h^0 - \zeta_1) + (1 - \theta)(V_h^0 - \zeta_1)\right) - d_4 E_h, \\
\frac{dh}{dt} &= \delta_h E_h - d_5 h, \\
\frac{dI_0}{dt} &= \frac{b_h(\alpha_h E_h + \beta_h I_1)}{N_h^0} (S_{I_1}^0 - \zeta_1) - (\varphi_2 + \mu_r) I_1, \\
\frac{dI_2}{dt} &= \frac{b_h(\alpha_h E_h + \beta_h I_2)}{N_h^0} (S_{I_2}^0 - \zeta_1) + \varphi_2 I_1 - \mu_r I_2.
\end{aligned}
\] (3.16)

Obviously, system (3.16) has zero equilibrium point. By simple calculation, we obtain that the Jacobian matrix of system (3.16) at zero equilibrium point is \( \bar{M} \) (defined in (3.7)). Since \( \bar{M} \) admits positive off-diagonal elements, by Perron-Frobenius Theorem, we can see that there exists a positive eigenvector \( \nu_M^\dagger \) for the maximin eigenvalue \( \lambda_M \) of \( \bar{M} \). Extensive calculations yield that \( \lambda_M > 0 \) since (3.6) and (3.7) hold. Consequently, we can get \( \lim_{t \to \infty} E_h(t) = \infty, \lim_{t \to \infty} I_h(t) = \infty, \lim_{t \to \infty} I_{21}(t) = \infty, \lim_{t \to \infty} I_{22}(t) = \infty \). Furthermore, by the comparison principle, we obtain \( \lim_{t \to \infty} E_h(t) = \infty, \lim_{t \to \infty} I_h(t) = \infty, \lim_{t \to \infty} I_{21}(t) = \infty, \lim_{t \to \infty} I_{22}(t) = \infty \). This contradicts \( E_h(t) \leq \zeta_2, I_h(t) \leq \zeta_2, I_{21}(t) \leq \zeta_2, I_{22}(t) \leq \zeta_2 \), for all \( t \geq T_0 \). Hence, (3.9) holds and the claim is proved. This indicates \( W^\dagger(E_0) \cap X_0 = \emptyset \). Obviously, every forward orbit in \( M_0 \) converges to \( E_0 \). It follows from Lemma 3.3 that system (2.1) is uniformly persistent with respect to \((X_0, \partial X_0)\). This completes the proof.

4. Optimal control

Insecticide applications and removing infected trees are two tactics being employed for the control of HLB (Bovè, 2006; Gottwald, 2010). There is an urgent need to explore an optimal control strategy in terms of possible combination of strategies to prevent the spread of citrus HLB while minimizing the implementation cost. In this paper, we introduce into the transmission model (2.1) a time dependent control variable \( u_0(t) \), representing removing effort of infected citrus trees, and also consider a time dependent control variable \( u_1(t) \), representing killing effort of sensitive ACP and resistant ACP. Thus, model (2.1) with the time dependent control variables \( u_0(t) \) and \( u_1(t) \) becomes:
\[
\begin{aligned}
&\frac{dS_h}{dt} = r(K - N_h) + \omega_h V_h - \eta_h(t) S_h - d_3 S_h, \\
&\frac{dV_h}{dt} = \nu_h S_h - (1 - \theta) \eta_h(t) V_h - d_2 V_h, \\
&\frac{dE_h}{dt} = \eta_h(t) S_h + (1 - \theta)V_h - d_4 E_h, \\
&\frac{dl_h}{dt} = \delta_h E_h - (\rho_1 u_h(t) + d_5) l_h, \\
&\frac{ds_{1,1}}{dt} = \lambda_1 - \eta_v(t) s_{1,1} - (\mu_v + \rho_2 u_v(t)) s_{1,1} - \varphi_1 s_{1,1}, \\
&\frac{ds_{1,2}}{dt} = \lambda_2 - \eta_v(t) s_{1,2} - (\mu_v + \rho_3 u_v(t)) s_{1,2} + \varphi_1 s_{1,2}, \\
&\frac{dl_{1,1}}{dt} = \eta_v(t) s_{1,1} - (\mu_v + \rho_2 u_v(t)) l_{1,1} - \varphi_2 l_{1,1}, \\
&\frac{dl_{1,2}}{dt} = \eta_v(t) s_{1,2} - (\mu_v + \rho_3 u_v(t)) l_{1,2} + \varphi_2 l_{1,2},
\end{aligned}
\]

where \(\rho_1, \rho_2, \rho_3\) is the control intensity coefficient. Our goal is to minimize the cost function defined as

\[
J(u) = \int_0^{t_f} (A_1 E_h(t) + A_2 l_h(t) + A_3 N_v(t) + A_4 u_h^2(t) + A_5 u_v^2(t)) dt
\]

subject to system (4.1), where \(t_f\) is the control period. This performance specification involves minimizing the numbers of exposed and infected citrus tree, along with the cost of applying the controls \(u(t) = (u_h(t), u_v(t))\). The quadratic costs have been frequently used (Agusto, 2013; Pei et al., 2016; Zhang et al., 2020). The coefficients, \(A_i, i = 1, \ldots, 5\) represent the desired weights on the benefit and cost the controls \(u(t) = (u_h(t), u_v(t))\) is a bounded Lebesgue integrable functions (Agusto & Lenhart, 2013; Pei et al., 2016). And we need to find an optimal control \(u^* = (u_h^*, u_v^*)\), such that

\[
J(u^*) = \min_{u} J(u),
\]

where the control set,

\[
U = \{u(t) : [0, t_f] \rightarrow [0, 1] \times [0, 1]\}.
\]

where \(u(t)\) is Lebesgue measurable. The necessary conditions that an optimal control quintuple must satisfy derive from the Pontryagin’s Minimum Principle (Pontryagin, 2018). This principle converts (4.1) and (4.2) into a problem of minimizing pointwise a Hamiltonian \(H\) with respect to the controls \(u_h(t)\) and \(u_v(t)\). In order to obtain the optimality conditions, we first formulate the following Hamiltonian functional from the objective functional (4.2) and the governing dynamics (4.1):

\[
H = A_1 E_h(t) + A_2 l_h(t) + A_3 N_v(t) + A_4 u_h^2(t) + A_5 u_v^2(t) \\
+ \lambda_{sh}[r(K - N_h) + \omega_h V_h - \eta_h(t) S_h - d_3 S_h] \\
+ \lambda_{vh}[\nu_h S_h - (1 - \theta) \eta_h(t) V_h - d_2 V_h] \\
+ \lambda_{eh}[\eta_h(t) S_h + (1 - \theta)V_h - d_4 E_h] \\
+ \lambda_{lh}[\delta_h E_h - (\rho_1 u_h(t) + d_5) l_h] \\
+ \lambda_{ls_1,1}[\lambda_1 - \eta_v(t) s_{1,1} - (\mu_v + \rho_2 u_v(t)) s_{1,1} - \varphi_1 s_{1,1}] \\
+ \lambda_{ls_1,2}[\lambda_2 - \eta_v(t) s_{1,2} - (\mu_v + \rho_3 u_v(t)) s_{1,2} + \varphi_1 s_{1,2}] \\
+ \lambda_{ll_1,1}[\eta_v(t) s_{1,1} - (\mu_v + \rho_2 u_v(t)) l_{1,1} - \varphi_2 l_{1,1}] \\
+ \lambda_{ll_2,2}[\eta_v(t) s_{1,2} - (\mu_v + \rho_3 u_v(t)) l_{1,2} + \varphi_2 l_{1,2}].
\]

where \(\lambda_{sh}, \lambda_{vh}, \lambda_{eh}, \lambda_{lh}, \lambda_{ls_1,1}, \lambda_{ls_2,2}, \lambda_{ll_1,1}, \lambda_{ll_2,2}\) are the associated adjoints for the states \(S_h, V_h, E_h, l_h, s_{1,1}, s_{1,2}, l_{1,1}, l_{1,2}\). The system of adjoint equations is found by taking the appropriate partial derivatives of the Hamiltonian (4.4) with respect to the associated state and control variables.
Theorem 4.1. Given an optimal control variables \((u_h^*, u_v^*)\), and solutions \(S_h^*, V_h^*, E_h^*, I_h^*, S_{v1}^*, I_{v1}^*, I_{v2}^*\) of the corresponding state system (4.1) that minimizes \(J(u_h^*, u_v^*)\) over \(U\). Then there exists adjoint variables \(\lambda_{S_h}, \lambda_{V_h}, \lambda_{E_h}, \lambda_{I_h}, \lambda_{S_{v1}}, \lambda_{I_{v1}}, \lambda_{I_{v2}}\) satisfying

\[
\frac{d\lambda_i}{dt} = \frac{\partial H}{\partial y_i}.
\]  

(4.5)

and with transversality conditions

\[
\lambda_i(t_f) = 0, \text{ where } i = S_h, V_h, E_h, I_h, S_{v1}, I_{v1}, I_{v2}.
\]  

(4.6)

The optimality conditions is given as

\[
\frac{\partial H}{\partial u_j} = 0, j = h, v.
\]

Furthermore, the time dependent control variables \((u_h^*, u_v^*)\) are given as

\[
\begin{align*}
  u_h^* &= \max \left\{ 0, \min \left\{ \frac{\lambda_{I_h} \rho_1^2 I_h^*}{2 \lambda_4}, 1 \right\} \right\}, \\
  u_v^* &= \max \left\{ 0, \min \left\{ \frac{\lambda_{I_v} \rho_2 S_{v1}^* + \lambda_{I_v} \rho_3 S_{v2}^* + \lambda_{I_v} \rho_2 I_{v1}^* + \lambda_{I_v} \rho_3 I_{v2}^*}{2 \lambda_5}, 1 \right\} \right\}
\end{align*}
\]  

(4.7)

Proof. According to the result in (Fleming & Rishel, 2012), we can easily obtain the existence of an optimal control. Consequently, the differential equations governing the adjoint variables are obtained by the differentiation of the Hamiltonian function, evaluated at the optimal controls. Then the adjoint system yields:

\[
\begin{align*}
  \frac{d\lambda_{S_h}}{dt} &= \frac{\partial H}{\partial S_h}, \quad \lambda_{S_h}(t_f) = 0, \\
  \frac{d\lambda_{V_h}}{dt} &= \frac{\partial H}{\partial V_h}, \quad \lambda_{V_h}(t_f) = 0, \\
  \frac{d\lambda_{E_h}}{dt} &= \frac{\partial H}{\partial E_h}, \quad \lambda_{E_h}(t_f) = 0, \\
  \frac{d\lambda_{I_h}}{dt} &= \frac{\partial H}{\partial I_h}, \quad \lambda_{I_h}(t_f) = 0, \\
  \frac{d\lambda_{S_{v1}}}{dt} &= \frac{\partial H}{\partial S_{v1}}, \quad \lambda_{S_{v1}}(t_f) = 0, \\
  \frac{d\lambda_{S_{v2}}}{dt} &= \frac{\partial H}{\partial S_{v2}}, \quad \lambda_{S_{v2}}(t_f) = 0, \\
  \frac{d\lambda_{I_{v1}}}{dt} &= \frac{\partial H}{\partial I_{v1}}, \quad \lambda_{I_{v1}}(t_f) = 0, \\
  \frac{d\lambda_{I_{v2}}}{dt} &= \frac{\partial H}{\partial I_{v2}}, \quad \lambda_{I_{v2}}(t_f) = 0.
\end{align*}
\]

Evaluated at the optimal controls and corresponding state variables, results in the stated adjoint system (4.4) and (4.5) given by
\[
\frac{d\lambda_{S_1}}{dt} = \lambda_{S_1}(r + d_3) - \lambda_{V_1}v_h + \eta_h(t)\frac{N^*_u - S^*_h}{N^*_h}(\lambda_{S_1} - \lambda_{E_1}) - \eta_h(t)^*\frac{(1 - \theta)V^*_h}{N^*_h}(\lambda_{V_1} - \lambda_{E_1})
\]
\[
- \eta_v(t)^*\frac{\lambda_{S_1}}{N^*_h}(\lambda_{S_1} - 1) - \eta_v(t)^*\frac{\lambda_{S_2}}{N^*_h}(\lambda_{S_2} - 1),
\]
\[
\frac{d\lambda_{V_1}}{dt} = \lambda_{V_1}(r - w_1) + \lambda_{V_1}d_2 - \eta_h(t)\frac{S^*_h}{N^*_h}(\lambda_{S_1} - 1) + (1 - \theta)\eta_h(t)^*\frac{V^*_h}{N^*_h}(\lambda_{V_1} - \lambda_{E_1})
\]
\[
- \eta_v(t)^*\frac{\lambda_{S_1}}{N^*_h}(\lambda_{S_1} - 1) - \eta_v(t)^*\frac{\lambda_{S_2}}{N^*_h}(\lambda_{S_2} - 1),
\]
\[
\frac{d\lambda_{E_1}}{dt} = -A_1 + \lambda_{S_1}r + \lambda_{E_1}d_4 - \lambda_{h_1}d_h - \eta_h(t)\frac{S^*_h}{N^*_h}(\lambda_{S_1} - 1) + (1 - \theta)\eta_h(t)^*\frac{V^*_h}{N^*_h}(\lambda_{V_1} - \lambda_{E_1})
\]
\[
- (\eta_v(t)^* - b_v\alpha_v)\frac{S^*_h}{N^*_h}(\lambda_{S_1} - 1) - (\eta_v(t)^* - b_v\alpha_v)\frac{S^*_h}{N^*_h}(\lambda_{S_2} - 1),
\]
\[
\frac{d\lambda_{S_2}}{dt} = -A_2 + \lambda_{S_2}(\rho_2 u_1 + m_v) - \eta_v(t)^*\frac{S^*_h}{N^*_h}(\lambda_{S_1} - 1)
\]
\[
- (\eta_v(t)^* - b_v\alpha_v)\frac{S^*_h}{N^*_h}(\lambda_{S_1} - 1),
\]
\[
\frac{d\lambda_{I_1}}{dt} = -A_3 + \lambda_{I_1}(\rho_1 u_1 + m_v) + \varphi_1(\lambda_{S_1} - 1)
\]
\[
- \eta_v(t)^*\frac{S^*_h}{N^*_h}(\lambda_{S_1} - 1),
\]
\[
\frac{d\lambda_{I_2}}{dt} = -A_3 + \lambda_{I_2}(\rho_2 u_1 + m_v) + \varphi_2(\lambda_{S_1} - 1)
\]
\[
+ b_v\alpha_v\frac{S^*_h}{N^*_h}(\lambda_{S_1} - 1) + (1 - \theta)b_v\beta_v\frac{V^*_h}{N^*_h}(\lambda_{V_1} - \lambda_{E_1}),
\]
\[
\frac{d\lambda_{I_3}}{dt} = -A_3 + \lambda_{I_3}(\rho_3 u_1 + m_v)
\]
\[
+ \frac{b_v\beta_v\frac{S^*_h}{N^*_h}(\lambda_{S_1} - 1) + (1 - \theta)b_v\beta_v\frac{V^*_h}{N^*_h}(\lambda_{V_1} - \lambda_{E_1})}{N^*_h},
\]

In addition, differentiating the Hamiltonian functional with respect to the control variables in the interior of the control set \(U\), and then solving for controls \((u^*_h, u^*_v)\) result in the optimality conditions given as follows:

\[
\frac{\partial H}{\partial u_h} = 2A_4 u_h - \lambda_{h_1}p_1 l^*_h
\]
\[
\frac{\partial H}{\partial u_v} = 2A_3 u_v - \lambda_{I_1}p_2 S^*_v + \lambda_{S_2}p_3 S^*_v - \lambda_{I_2}p_2 I^*_v - \lambda_{I_3}p_3 I^*_v.
\]

By simple calculation, we have

\[
\begin{align*}
    u^*_h &= \frac{\lambda_{h_1}p_1 l^*_h}{2A_4}, \\
    u^*_v &= \frac{\lambda_{S_1}p_2 S^*_v + \lambda_{S_2}p_3 S^*_v - \lambda_{I_1}p_2 I^*_v + \lambda_{I_3}p_3 I^*_v}{2A_3}.
\end{align*}
\]

Taking into account the property of control set in (4.3), the characterization (4.6) can be obtained. This completes the proof.

In the next part, we will simulate numerically the solutions of the optimality system and the corresponding optimal control, and then give the interpretations from various cases.
5. Numerical simulation

In this section, we mainly present some numerical simulation results to verify or extend our results, which explore the impact of insecticides resistance on HLB transmission between citrus tree population and ACP population, and assess the effects of different control strategies against HLB. By using the Latin Hypercube Sampling (LHS) method (Blower & Dowlatabadi, 1994) and the forward-backward sweep method (Lenhart & Workman, 2007). Table 2 contains the value of the parameters that will be used in numerical simulations.

To examine the sensitivity of model results to the uncertainty of parameters, we do sensitivity and uncertainty analysis. Based on the parameters of model (2.1), we perform a global sensitivity analysis on the basic reproduction number R₀ by using the Latin Hypercube Sampling (LHS) method (Blower & Dowlatabadi, 1994), we compute the Partial Rank Correlation Coefficients (PRCCs) of R₀. The sensitivity and uncertainty analysis are presented in Fig. 2. It shows how uncertainty in model parameters may influence R₀. We can observe from Fig. 2(a) that R₀ is very sensitive to environmental carrying capacity of citrus trees K, ACP biting rate bᵥ, Vaccination rate vh, transmission probability from ACP to citrus trees αh and βh, vaccine efficacy θ, disease related death rate γh, ACP recruitment rate Δ₁ and Δ₂, transmission probability from Iₜ to ACP βᵥ, natural mortality rate of ACP μᵥ, but not sensitive to replanting rate of citrus trees r, natural mortality rate in citrus trees mₜ, disease progression rate of infectious of exposed citrus trees δ₀, transmission probability from Eₜ to ACP αᵥ, mutation rate from sensitive ACP to infected resistant ACP 4₁ and 4₂. For both transmission probabilities, βᵥ has a greater impact on R₀. Thus, decreasing βᵥ is very effective in reducing R₀, that is to say, decreasing the transmission probability from Iₜ to ACP plays a key role in the control of HLB. Fig. 2(b) (uncertainty analysis), we can see that about 88.7% of the distribution of R₀ is greater than 1.

Table 2
Numerical values of the parameters for HLB model (2.1).

| Parameter | Baseline value | Unit       | Reference              |
|-----------|----------------|------------|------------------------|
| K         | 1000           |            | Zhang et al. (2020)    |
| r         | 0.6            | year⁻¹     | Zhang et al. (2020)    |
| αₜₜ       | 0.05           | year⁻¹     | Assumed                |
| bᵥ        | 600            | year⁻¹     | Assumed                |
| αₜ       | 4.8830 × 10⁻⁴  |            | Taylor et al. (2016)   |
| βₜ       | 0.8 × 4.8830 × 10⁻⁴ | year⁻¹ | Assumed                |
| vₜ       | 0.7            |            | Assumed                |
| μₜ       | 0.04           |            | Assumed                |
| μᵥ       | 0.8            |            | Assumed                |
| δ₀       | 12.97          |            | Assumed                |
| γₜ       | 0.1            |            | Assumed                |
| Δ₁       | 3.3253 × 10⁵   |            | Taylor et al. (2016)   |
| Δ₂       | 0.8 × 3.3253 × 10⁵ | year⁻¹ | Assumed                |
| αᵥ       | 0.9 × 3.9064 × 10⁻⁴ | year⁻¹ | Assumed                |
| βᵥ       | 3.9064 × 10⁻⁴  |            | Taylor et al. (2016)   |
| μᵥ       | 5.9441         |            | Taylor et al. (2016)   |
| φ₁       | 0.3            |            | Assumed                |
| φ₂       | 0.24           |            | Assumed                |

Fig. 2. Sensitivity analysis and uncertainty analysis of the basic reproduction number R₀. (a) shows the sensitivity indices of R₀, and (b) shows histogram obtained from LHS using a sample size of 1000 for R₀.
This indicates that persistent HLB bacterial infection is likely to occur. Furthermore, by numerical calculation, we obtain the mean and standard deviation of $R_0$ are 1.5611 and 0.4973, respectively.

We consider the influence of two model parameter $\gamma_h$ and $\mu_v$ on the basic reproduction number $R_0$ in Fig. 3. We can observe from Fig. 3 that $R_0$ is very sensitive to $\gamma_h$ and $\mu_v$, it is seen that as $\gamma_h$ and $\mu_v$ increase, the basic reproduction number $R_0$ decreases quickly.

The dynamics of the basic reproduction number of our model as a function of insecticide resistance intensities $\phi_1$ and $\phi_2$ is depicted in Fig. 4. It is seen that as the resistance coefficients $\phi_1$ and $\phi_2$ increase, the basic reproduction number $R_0$ decreases slowly. This indicates that the intensity of insecticide resistance present in ACP population may slightly reduce the effectiveness of control measures, which is in agreement with results of sensitivity analysis (see Fig. 2(a)).

In order to reduce the number of trees infected by HLB disease, two possible control measures have been considered in this paper, including removal of diseased trees and application of insecticides. The objective of this paper is to analyze the effect of three controls on the transmission of HLB, and explore the optimal solution of the optimality system (4.1) and the corresponding optimal control by using the forward-backward sweep method. The initial numbers of susceptible citrus trees and ACP are assumed that $S_h(0)=500$, $V_h(0)=492$, $S_{v1}(0)=30000$, $S_{v2}(0)=20000$. Then we assume that the initial numbers of

![Fig. 3](image)

**Fig. 3.** The basic reproduction number of model (2.1) as a function of disease related death rate $\gamma_h$ and natural mortality rate of ACP $\mu_v$. The red curve indicates that the basic regeneration number $R_0$ is equal to one.

![Fig. 4](image)

**Fig. 4.** The basic reproduction number of model (2.1) as a function of resistance intensities $\phi_1$ and $\phi_2$. 
infectious citrus trees and ACP are $E(h)(0) = 3$, $I_h(0) = 5$, $I_v(1)(0) = 20$, $I_v(2)(0) = 10$, respectively. In addition, the control intensity coefficients $\rho_1 = 10\mu_v$, $\rho_2 = 20\mu_v$ and $\rho_1 = 5\mu_v$. In the simulations, the values of other parameters are taken from Table 2.

In (Yan & Zou, 2008), the authors point out fixing the right weights in practical problems is a very difficult task which requires an amount of work on data analysis and fitting. For the weight factors in the objective function, we choose $A_1 = 1000$, $A_2 = 1000$, $A_3 = 0.01$, $A_4 = 100$ and $A_5 = 100$. Then the weights in the simulations here are only of theoretical sense to illustrate the control strategies proposed.

The following algorithm is used to compute the optimal controls and state values using a Runge-Kutta method of the fourth order. As illustrated in (Agusto & Khan, 2018), we should first give an initial estimate for the control quintuple, then solve the state variables forward in time by using the dynamics (4.1). The results obtained for the state variables are substituted into the adjoint equation (4.6). These adjoint equations with given final conditions (4.8) are then solved backward in time. Both the state and adjoint values are then used to update the control, and the process is repeated until the current state, adjoint, and controls values converge sufficiently.

The numbers of infectious citrus trees and infectious ACP under different control levels are depicted in Fig. 5. In view of reducing the total number of the diseased citrus trees (or the diseased ACP), we can observe that the optimal control strategy is consistent with the upper bound control strategy and is superior to the constant control. The enhancement of the level of

| Table 3 The costs of the objective function under different weights and control measures. |
|---------------------------------|----------------|----------------|----------------|----------------|----------------|
| (1000,1000,0.01,100,100)        | 10524          | 8064           | 7502           | 6716           | 6703           |
| (800,1000,0.01,1000,1000)       | 10424          | 8077           | 7739           | 8484           | 7517           |
| (1000,10000,0.01,10000,1000)    | 9191           | 7521           | 7203           | 7297           | 6955           |
| (500,1000,0.01,500,600)         | 10248          | 7951           | 7512           | 7498           | 7076           |
| (1000,2000,0.01,1000,2000)      | 19567          | 15389          | 14930          | 15694          | 14052          |

Fig. 5. Number of infectious citrus trees and infectious ACP under different control levels, (a) exposed citrus trees, (b) infected citrus trees, (c) infected sensitive ACP, (d) infected resistant ACP.
control can achieve significant effects on both the number of hosts and vectors. Moreover, it follows from Table 3 that the cost of the optimal control is less than that of the upper bound control. We may conclude that the optimal control strategy appears to offer a promising measure for HLB control.

![Fig. 6](image1.png) Optimal control law of $u_h$ and $u_v$.

![Fig. 7](image2.png) Number of infectious citrus trees and infectious ACP under different optimal control measures, i.e. (I) $u_h$ and $u_v$; (II) $u_h$ ($u_v = 0$); (III) $u_v$ ($u_h = 0$).
Fig. 6 gives the optimal control profile for $u_h$ and $u_v$. It shows that, the control variable $u_h$ starts at the upper bound 1 for about 90% time and then gradually decreases to the lower bound 0 at the end of the simulation period. While the control variables $u_v$ start at the upper bound for a shorter time and slowly decrease to the lower bound at the end of the simulation period.

Fig. 8. Optimal control law of: (I) $u_h$ ($u_v = 0$); (II) $u_v$ ($u_h = 0$).

Fig. 9. Comparison of the number of exposed citrus trees $E_i(t)$ with different weights.
Fig. 10. Comparison of the number of infectious citrus trees $I_i(t)$ with different weights.

Fig. 11. Comparison of the number of infected sensitive ACP $I_v(t)$ with different weights.
Fig. 7 presents the number of infectious citrus trees and infectious ACP under different control strategies. These control strategies are: (I) $u_{h}$ and $u_{v}$; (II) $u_{h}$, ($u_{v} = 0$); (III) $u_{v}$ ($u_{h} = 0$). From Fig. 7, we can observe that: (1) the cost of implementing strategy (II) is lower than that of implementing strategy (III); (2) the number of infectious citrus trees is the smallest when implementing strategy (I), while the number of infectious citrus trees is the largest when implementing strategy (II); (3) in addition to strategy (I), the final number of diseased ACP is the least when applying strategy (III), and the final number of diseased ACP is the most when applying strategy (II). These results imply that multiple control strategies should be adopted simultaneously to control the spread of HLB.

The optimal control trajectories under different control measures are presented in Fig. 8. It can be seen that in the early phase of HLB outbreak, the upper bound control strategies should be adopted in most cases to suppress the transmission of the disease.

Figs. 9–13 show the trajectories of the infectious citrus trees, the infectious ACP, and the optimal controls with different weights in objective function, respectively. Based on the simulation results, we can see that the weights have a great impact on the control cost (see Table 3), but have little or no effect on the spread of HLB. Consequently, the weights in the objective function have little impact on the optimal control strategy (Fig. 13).

6. Conclusion

In this paper, a deterministic mathematical model was first used to formulate the transmission dynamics of HLB between the ACP and the citrus trees, which incorporated the insecticide resistance of ACP. The stability of equilibria of the model was analyzed using the basic reproduction number $R_0$, derived by the next generation matrix. Theoretical results have shown that the disease free equilibrium is locally asymptotically stable if $R_0 < 1$, whereas the HLB system (2.1) is persistent if $R_0 > 1$.

Furthermore, we used the optimization theory and the three time-dependent control variables to establish an optimal control strategy for exclusion of HLB infection by minimizing the number of exposed and infected trees. The necessary conditions for the existence of optimal solution of control problem are obtained by using Pontryagin’s Maximum Principle.
Finally, we verified the analytical results by numerical simulations. Based on analytical and the numerical simulation results, the main conclusions of this paper can be summarized as follows:

1. The intensity of insecticide resistance present in ACP population may slightly reduce the effectiveness of control measures.
2. The optimal control strategy is superior to the constant control strategy in decreasing the prevalence of the infected citrus trees, and the cost of implementing optimal control is much lower than that of the constant control strategy.
3. In the early phase of the transmission of HLB, spraying insecticides to kill ACP is more effective than other control strategies in reducing the number of the infected ACP.
4. The weights in the objective function have little impact on the optimal control strategy.

Data availability

All data generated or analyzed during this study are included in this article.

Declaration of competing interest

The authors declare that they have no conflicts of interest regarding the publication of this paper.
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Appendix A. The proof of global asymptotical stability of positive equilibrium \((S_0^h, V_0^h)\)

Denote \( \bar{N}_h = \bar{S}_h + \bar{V}_h \), then system (3.7) is equivalent to the following system

\[
\begin{align*}
\frac{d\bar{N}_h}{dt} &= r(K - 2x_2) - (r + \mu_h + \eta_h)\bar{N}_h + \theta \eta_h \bar{V}_h, \\
\frac{d\bar{V}_h}{dt} &= \eta_h \bar{N}_h - \eta_h \bar{V}_h - (1 - \theta) \eta_h \bar{V}_h - d_2 \bar{V}_h.
\end{align*}
\]

(6.1)

Denote \( x = \bar{N}_h, y = \bar{V}_h \), \( c_0 = r(K - 2x_2), c_1 = r + \mu_h + \eta_h, c_2 = \theta \eta_h, c_3 = \eta_h \) and \( c_4 = \eta_h(1 - \theta) \eta_h + d_2 \). By calculating, \( c_1c_4 - c_2c_3 = (r + \mu_h)(\eta_h + d_2) + \eta_h(1 - \theta) \eta_h + d_2 + (1 - \theta) \eta_h \eta_h > 0 \). So system (6.1) is given by

\[
\begin{align*}
\frac{dx}{dt} &= c_0 - c_1x + c_2y, \\
\frac{dy}{dt} &= c_3x - c_4y.
\end{align*}
\]

(6.2)

It is easy to obtain, the model (6.2) has a unique positive equilibrium point \((x^*, y^*)\), where

\[
x^* = \frac{c_0c_4}{c_1c_4 - c_2c_3}, \quad y^* = \frac{c_0c_3}{c_1c_4 - c_2c_3}.
\]

Thus system (6.2) can be rewritten as

\[
\begin{align*}
\frac{dx}{dt} &= -c_1(x - x^*) + c_2(y - y^*), \\
\frac{dy}{dt} &= c_3(x - x^*) - c_4(y - y^*).
\end{align*}
\]

(6.3)

We define a Lyapunov function

\[
L(x, y) = \frac{(x - x^*)^2}{2} + \frac{c_2}{c_3} \frac{(y - y^*)^2}{2}.
\]

Then the derivative of function \( L(x, y) \) along solutions of system (6.3) is given by

\[
\frac{dL}{dt} = (x - x^*)[-c_1(x - x^*) + c_2(y - y^*)] + \frac{c_2}{c_3} (y - y^*)[c_3(x - x^*) - c_4(y - y^*)]
\]

\[
= -c_1(x - x^*)^2 + 2c_2(x - x^*)(y - y^*) - \frac{c_2c_4}{c_3}(y - y^*)^2
\]

\[
= -c_1\left[(x - x^*)^2 - \frac{c_2}{c_1c_3}(y - y^*)^2\right] - \frac{c_2}{c_1c_3}(c_1c_4 - c_2c_3)(y - y^*)^2.
\]

Thus, we have \( \frac{dL}{dt} \leq 0 \) and the equality holds if and only if \( x = x^* \) and \( y = y^* \), that is, \( \bar{S}_h = \bar{S}_0^h \) and \( \bar{V}_h = \bar{V}_0^h \). It follows that the unique positive equilibrium point \((\bar{S}_0^h, \bar{V}_0^h)\) of (3.12) is globally asymptotically stable.

References

Agusto, F. (2013). Optimal isolation control strategies and cost-effectiveness analysis of a two-strain avian influenza model. Biosystems, 113, 155–164.
Agusto, F., & Khan, M. (2018). Optimal control strategies for dengue transmission in Pakistan. Mathematical Biosciences, 305, 102–121.
Agusto, F., & Lenhart, S. (2013). Optimal control of the spread of malaria superinfectivity. *Journal of Biological Systems, 21*, 1–26.

Alzahrani, E. O., Ahmad, W., Khan, M. A., & Malebary, S. J. (2021). Optimal control strategies of zika virus model with mutant. *Communications in Nonlinear Science and Numerical Simulation, 93*, 105532.

Arredondo Valdés, R., Delgado Ortiz, J. C., Beltrán Beache, M., Anguiano Cabello, J., Cerna Chávez, E., Rodríguez Pagaña, Y., & Ochoa Fuentes, Y. M. (2016). A review of techniques for detecting Huanglongbing (greening) in citrus. *Canadian Journal of Microbiology, 62*, 803–811.

Blower, S. M., & Dowlatabadi, H. (1994). Sensitivity and uncertainty analysis of complex models of disease transmission: An hiv model, as an example. *International Statistical Review/Revue Internationale de Statistique, 62*, 229–243.

Bove, J. M. (2006). Huanglongbing: A destructive, newly-emerging, century-old disease of citrus. *Journal of Plant Pathology, 88*, 7–37.

Chiyaka, C., Singer, B. H., Halbert, S. E., Morris, J. G., & van Bruggen, A. H. (2012). Modeling Huanglongbing transmission within a citrus tree. *Proceedings of the National Academy of Sciences, 109*, 12213–12218.

Chowdhury, J., Al Basir, F., Takeuchi, Y., Ghosh, M., & Roy, P. K. (2019). A mathematical model for pest management in jatropha curcas with integrated pesticides—an optimal control approach. *Ecological Complexity, 37*, 24–31.

Fleming, W. H., & Rishel, R. W. (2012). *Deterministic and stochastic optimal control (Vol. 1).* Springer Science & Business Media.

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

Gottwald, T. R. (2010). Current epidemiological understanding of citrus Huanglongbing. *Annual Review of Phytopathology, 48*, 119–139.

Hu, J., Jiang, J., & Wang, N. (2018). Control of citrus huanglongbing via trunk injection of plant defense activators and antibiotics. *Phytopathology, 108*, 186–195.

Jackson, M., & Chen-Charpentier, B. M. (2019). Modeling plant virus propagation with seasonality. *Journal of Computational and Applied Mathematics, 345*, 310–319.

Japson, K., Stupiansky, J., & Phlyugin, S. S. (2013). Mathematical modeling of citrus groves infected by Huanglongbing. *Mathematical Biosciences and Engineering, 10*, 705–728.

Jeger, M., Madden, L., & Van Den Bosch, F. (2018). Plant virus epidemiology: Applications and prospects for mathematical modeling and analysis to improve understanding and disease control. *Plant Disease, 102*, 837–894.

Khan, M. A., Fatmawati, et al. (2021). Dengue infection modeling and its optimal control analysis in east java, Indonesia. *Helijon, 7*, Article e06023.

Khan, M. F., Alrabahah, H., Ullah, S., Khan, M. A., Farooq, M., bin Mamat, M., & Asjad, M. I. (2021). A new fractional model for vector-host disease with saturated treatment function via singular and non-singular operators. *Alexandria Eng. J., 60*, 629–645.

Lee, J. A., Halbert, S. E., Dawson, W. O., Robertson, C. J., Keesling, J. E., & Singer, B. H. (2015). Asymptomatic spread of Huanglongbing and implications for disease control. *Proceedings of the National Academy of Sciences, 112*, 7603–7610.

Lenhart, S., & Workman, J. T. (2007). *Optimal control applied to biological models.* CRC press.

Meng, X., & Li, Z. (2010). The dynamics of plant disease models with continuous and impulsive cultural control strategies. *Horticultural Reviews, 44*, 315–361.

Mingxue, D. (2009). Forming process and basis and technological points of the theory emphasis on control citrus psylla for integrated control Huanglongbing. *Chin. Agric. Sci. Bull., 25*, 358–363.

Okon, K. O., Rachid, O., & Marcus, N. (2013). Optimal control strategies and cost-effectiveness analysis of a malaria psylla for integrated control Huanglongbing. *Chin. Agric. Sci. Bull., 25*, 1804.

Ponytryagin, L. S. (2018). *Mathematical theory of optimal processes.* Routledge.

Taylor, R. A., Mordecai, E. A., Gilligan, C. A., Rohr, J. R., & Johnson, L. R. (2016). Mathematical models are a powerful method to understand and control the spread of Huanglongbing. *PeerJ, 4*, 2642–2660.

Tu, Y., Gao, S., Liu, Y., Chen, D., & Xu, Y. (2019). Transmission dynamics and optimal control of stage structured HLB model. *Mathematical Biosciences and Engineering, 16*, 5180–5205.

R. G. d. Vilamiu, S. Ternes, G. A. Braga, F. F. Laranjeira, A model for Huanglongbing spread between citrus plants including delay times and human intervention, in: AIP conference proceedings, volume Vol. 1479, American Institute of Physics, pp. 2315-2319.

Wang, N., & Trivedi, P. (2013). Citrus huanglongbing: A newly relevant disease presents unprecedented challenges. *Phytopathology, 103*, 652–665.

Yan, X., & Zhou, Y. (2008). Optimal and sub-optimal quarantine and isolation control in sars epidemics. *Mathematical and Computer Modelling, 47*, 235–245.

Zhang, H., & Georgescu, P. (2015). The influence of the multiplicity of infection upon the dynamics of a crop-pest-pathogen model with defence mechanisms. *Applied Mathematical Modelling, 39*, 2416–2435.

Zhang, F., Qiu, Z., Huang, A., & Zhao, X. (2021). Optimal control and cost-effectiveness analysis of a Huanglongbing model with comprehensive interventions. *Applied Mathematical Modelling, 90*, 719–741.

Zhang, F., Qiu, Z., Zhong, B., Feng, T., & Huang, A. (2020). Modeling citrus Huanglongbing transmission within an orchard and its optimal control. *Mathematical Biosciences and Engineering, 17*, 2048–2069.

Zhao, X.-Q. (2003). *Dynamical systems in population biology* (Vol. 16). Springer.

Zhao, W., Li, J., Zhang, T., Meng, X., & Zhang, T. (2017). Persistence and ergodicity of plant disease model with markov conversion and impulsive toxicant input. *Communications in Nonlinear Science and Numerical Simulation, 48*, 70–84.