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Research Article

Threshold Dynamics of a Huanglongbing Model with Logistic Growth in Periodic Environments

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We analyze the impact of seasonal activity of psyllid on the dynamics of Huanglongbing (HLB) infection. A new model about HLB transmission with Logistic growth in psyllid insect vectors and periodic coefficients has been investigated. It is shown that the global dynamics are determined by the basic reproduction number R_0 which is defined through the spectral radius of a linear integral operator. If $R_0 < 1$, then the disease-free periodic solution is globally asymptotically stable and if $R_0 > 1$, then the disease persists. Numerical values of parameters of the model are evaluated taken from the literatures. Furthermore, numerical simulations support our analytical conclusions and the sensitive analysis on the basic reproduction number to the changes of average and amplitude values of the recruitment function of citrus are shown. Finally, some useful comments on controlling the transmission of HLB are given.

1. Introduction

Plant disease is an important constraint to crop production. Due to plant diseases, more than 10% of global food production is lost and 800 million people do not have adequate food in the world [1–3]. Plant pathologists cannot ignore the juxtaposition of these figures for food shortage and the reduction of crops caused by plant disease.

Nowadays, Huanglongbing (HLB) which is a century old disease caused by the bacteria *Candidatus* Liberibacter spp is one of the most serious problems of citrus worldwide [4]. HLB has been responsible for the near destruction of citrus industries in Asia and Africa [4]. The main symptoms on HLB-infected citrus trees are yellow shoots, leaves with blotchy mottle, and small lopsided fruits [4, 5]. The HLB is a phloem-restricted, noncultured, Gram-negative bacterium causing crippling diseases denoting "greening" in South Africa, "mottle leaf" in the Philippines, "dieback" in India, and "vein phloem degeneration" in Indonesia. The infected citrus orchards are usually destroyed or become unproductive in 5 to 8 years [4].

Most of the known plant viruses are transmitted by insect vectors and entirely dependent on the behaviour and dispersal capacity of their vectors to spread from plant to plant. HLB, a destructive disease of citrus, can be transmitted by grafting from citrus to citrus and by dodder to periwinkle. The citrus psyllid (Diaphorina Citri Kuwayama) is natural and mainly vector [4]. In this paper, we mainly consider that HLB transmitted from tree to tree by Asian citrus psyllid insect vectors.

Mathematical models play an important role in understanding the epidemiology of vector-transmitted plant diseases. Since the introduction of HLB, a lot of researches have been conducted on the epidemiology of the disease and on the vector, but the result of these two lines of inquiry integrated is very few. Analytical models have also been developed for the spread of citrus canker [6], but models for vector-transmitted bacterial pathogens are still preliminary [7]. In [8], the authors proposed a deterministic compartmental mathematic model to analyze HLB spread between citrus plants. They assumed that all coefficients of the model are constant (autonomous systems). However, in the real world, actual data and evidence show that dynamics of disease transmission are not as simple as shown in the model. In [9], Hall and Hentz have studied seasonal activity of psyllid insect vectors which is correlated with humidity. Seasonal fluctuations in the transmission of infectious diseases imply that the corresponding mathematical models may admit periodic solutions. It is interesting and important to study the globally dynamics which are determined by threshold parameter R_0 in periodic epidemiological models.

Based on above introduction, we propose a model with periodic transmission rates to investigate the seasonal HLB epidemics [10, 11]. In this model, we consider Logistic growth term for dynamics of susceptible psyllid vector. Furthermore, we assumed that the infective citrus population is generated through susceptible citrus which was bit by infective psyllid and the susceptible psyllid bit the infective citrus which will become infective psyllid. Then, the periodic system is as follows:

$$\begin{aligned}\frac{dS_h(t)}{dt} &= \Lambda(t) - \beta_1(t) S_h(t) I_v(t) - \mu_1(t) S_h(t),\\ \frac{dI_h(t)}{dt} &= \beta_1(t) S_h(t) I_v(t) - \mu_1(t) I_h(t) - d(t) I_h(t),\\ \frac{dS_v(t)}{dt} &= b(t)\left(S_v(t)+I_v(t)\right)\left[1 - \frac{S_v(t)+I_v(t)}{m\left(S_h(t)+I_h(t)\right)}\right]\\ &\quad - \beta_2(t) S_v(t) I_h(t),\\ \frac{dI_v(t)}{dt} &= \beta_2(t) S_v(t) I_h(t) - \mu_2(t) I_v(t),\end{aligned} \tag{1}$$

with initial condition

$$S_h(0) > 0, \qquad I_h(0) > 0, \qquad S_v(0) > 0, \qquad I_v(0) > 0. \tag{2}$$

Here, $S_h(t)$, $I_h(t)$, $S_v(t)$, and $I_v(t)$ represent susceptible citrus host, infected citrus host, susceptible psyllid, and infected psyllid, respectively. We can easily see that $N_h(t) = S_h(t) + I_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$ are the number of citrus population and psyllid population, respectively. $\Lambda(t)$ is the recruitment rate of citrus at time t, $\beta_1(t)$ is the infected rate of citrus host at time t, $\mu_1(t)$ and $d(t)$ are the nature death and disease induced death rate of citrus host at time t, respectively, $b(t)$ is the intrinsic growth rate of psyllid at time t, $\beta_2(t)$ and $\mu_2(t)$ are the infected rate and the nature death rate of psyllid at time t, respectively, and $m(>0)$ is the maximum abundance of psyllid per citrus. $\Lambda(t)$, $\beta_1(t)$, $\mu_1(t)$, $d(t)$, $b(t)$, $\beta_2(t)$, and $\mu_2(t)$ are continuous, positive ω-periodic functions.

The paper is organized as follows. In the next section, we give the basic reproduction number of (1). In Sections 3 and 4, the results show that the dynamical properties of the model are completely determined by R_0. That is, if $R_0 < 1$, the disease-free periodic solution is globally asymptotically stable, and if $R_0 > 1$, the model is permanence. In Section 5, we present numerical simulations which demonstrate the theoretical analysis and a brief discussion of our main results.

2. Basic Reproduction Number

In the following, we introduce some notations and lemmas which will be used for our further argument.

Let (R^k, R^k_+) be the standard ordered k-dimensional Euclidean space with a norm $\|\cdot\|$. For $u, v \in R^k$; we denote $u \geq v$ if $u - v \in R^k_+$, $u > v$ if $u - v \in R^k_+ \setminus \{0\}$, and $u \gg v$ if $u - v \in \operatorname{Int}(R^k_+)$, respectively.

Define $g^L = \max_{t\in[0,\omega)} g(t)$ and $g^M = \min_{t\in[0,\omega)} g(t)$, where $g(t)$ is a continuous, positive, ω-periodic function.

Consider the following linear ordinary differential system:

$$\frac{dx(t)}{dt} = A(t)\, x(t), \tag{3}$$

where $A(t)$ is a continuous, cooperative, irreducible, and ω-periodic $k \times k$ matrix function. Denote $\Phi_A(t)$ be the fundamental solution matrix of (3) and $r(\Phi_A(\omega))$ be the spectral radius of $\Phi_A(\omega)$. By the Perron-Frobenius Theorem, we know that $r(\Phi_A(\omega))$ is the principle eigenvalue of $\Phi_A(\omega)$; that is, it is simple and admits an eigenvector $v^* \gg 0$.

Lemma 1 (see [12]). *Let $p = (1/\omega)\ln r(\Phi_{A(\cdot)}(\omega))$. Then there exists a positive ω-periodic function $v(t)$ such that $\exp(pt)v(t)$ is a solution of (3).*

Consider the following nonautonomous linear equation:

$$\frac{dS_h(t)}{dt} = \Lambda(t) - \mu_1(t) S_h(t), \tag{4}$$

where $\Lambda(t)$ and $\mu_1(t)$ are the same as in System (1). From Zhang and Teng ([13, Lemma 2.1]) and simple calculation, we have the following lemma.

Lemma 2. *System (4) has a unique positive ω-periodic solution $S_h^*(t)$ which is globally asymptotically stable.*

Consider the following nonautonomous Logistic equation:

$$\frac{dS_v(t)}{dt} = b(t) S_v(t)\left(1 - \frac{S_v(t)}{mS_h(t)}\right), \tag{5}$$

where $b(t)$ and m are the same as in system (1). From Teng and Li ([14, Lemma 2]) and simple calculation, we can obtain the following lemma.

Lemma 3. *System (5) has a unique positive ω-periodic solution $S_v^*(t)$ which is globally asymptotically stable, where $S_v^*(t) = mS_h^*(t)$.*

According to Lemmas 2 and 3, it is easy to see that (1) has a unique disease-free periodic solution $(S_h^*(t), 0, S_v^*(t), 0)$.

Now, we use the generation operator approach (see [15]) to derive the basic reproduction number. Applying the symbol of the theory in Wang and Zhao [15], for system (1), we have

$$\mathscr{F}(t,x) = \begin{pmatrix} \beta_1(t) S_h(t) I_v(t) \\ \beta_2(t) S_v(t) I_h(t) \\ 0 \\ 0 \end{pmatrix},$$

$$\mathscr{V}^{+}(t,x)=\begin{pmatrix}0\\0\\\Lambda(t)\\b(t)\left(S_v(t)+I_v(t)\right)\end{pmatrix},$$

$$\mathscr{V}^{-}(t,x)=\begin{pmatrix}\left(\mu_1(t)+d(t)\right)I_h(t)\\\mu_2(t)I_v(t)\\\mu_1(t)S_h+\beta_1(t)S_h(t)I_v(t)\\b(t)\dfrac{\left(S_v(t)+I_v(t)\right)^2}{m\left(S_h(t)+I_h(t)\right)}+\beta_2(t)S_v(t)I_h(t)\end{pmatrix}, \tag{6}$$

where $x=(I_h(t),I_v(t),S_h(t),S_v(t))^T$. Then System (1) can be written as the following form:

$$\frac{dx(t)}{dt}=\mathscr{F}(t,x(t))-\mathscr{V}(t,x(t)), \tag{7}$$

where $\mathscr{V}(t,x(t))=\mathscr{V}^{-}(t,x(t))-\mathscr{V}^{+}(t,x(t))$.

It is easy to obtain that the conditions (A1)–(A5) in [15] hold. In the following, we will check the conditions (A6) and (A7) in [15].

We know that $x^*(t)=(0,0,S_h^*(t),S_v^*(t))$ is the disease-free periodic solution of system (7). Denote

$$\begin{aligned}f(t,x(t))&=\mathscr{F}(t,x(t))-\mathscr{V}(t,x(t)),\\M(t)&=\left(\frac{\partial f_i(t,x^*(t))}{\partial x_j}\right)_{3\le i,j\le 4},\end{aligned} \tag{8}$$

where $f_i(t,x(t))$ and x_i are the ith components of $f(t,x(t))$ and x, respectively. According to (6), we have

$$M(t)=\begin{pmatrix}-\mu_1(t)&0\\\dfrac{b(t)S_v^{*2}(t)}{mS_h^{*2}(t)}&-b(t)\end{pmatrix}. \tag{9}$$

It is easy to see that $r(\Phi_M(\omega))<1$, where $r(\Phi_M(\omega))$ is the spectral radius of $\Phi_M(\omega)$. This implies that $x^*(t)$ is linearly asymptotically stable in the disease-free subspace $X_S=\{(0,0,S_h,S_v)\in R_+^4\}$. Thus, condition (A6) in [15] holds.

We further define

$$\begin{aligned}F(t)&=\left(\frac{\partial\mathscr{F}_i(t,x^*(t))}{\partial x_j}\right)_{1\le i,j\le 2},\\V(t)&=\left(\frac{\partial\mathscr{V}_i(t,x^*(t))}{\partial x_j}\right)_{1\le i,j\le 2},\end{aligned} \tag{10}$$

where $\mathscr{F}_i(t,x)$ and $\mathscr{V}_i(t,x)$ are the ith components of $\mathscr{F}(t,x)$ and $\mathscr{V}(t,x)$, respectively. Then, from (6), we obtain that

$$\begin{aligned}F(t)&=\begin{pmatrix}0&\beta_1(t)S_h^*(t)\\\beta_2(t)S_v^*(t)&0\end{pmatrix},\\V(t)&=\begin{pmatrix}\mu_1(t)+d(t)&0\\0&\mu_2(t)\end{pmatrix}.\end{aligned} \tag{11}$$

Let $Y(t,s)$ be a 2×2 matrix solution of the system:

$$\begin{aligned}\frac{dY(t,s)}{dt}&=-V(t)Y(t,s),\quad\forall t\ge s,\\Y(s,s)&=I,\end{aligned} \tag{12}$$

where I is 2×2 identity matrix. From (11) and (12), we have $r(\Phi_{-V}(\omega))<1$. Therefore, the condition (A7) in [15] also holds.

Let C_ω be the ordered Banach space of all ω-periodic function from $R\to R^2$, which is equipped with maximum norm $\|\cdot\|_\infty$ and the positive cone $C_\omega^+=\{\phi\in C_\omega:\phi(t)\ge 0$, for all $t\in R\}$. Define the following linear operator $L:C_\omega\to C_\omega$ by

$$\left(L\phi\right)(t)=\int_0^{+\infty}Y(t,t-a)F(t-a)\phi(t-a)\,da,\qquad\forall t\in R,\quad\phi\in C_\omega. \tag{13}$$

Based on the assumptions above and the results of Wang and Zhang [15], we can derive the basic reproduction number R_0 of system (1) as follows:

$$R_0=r(L), \tag{14}$$

and obtain the following conclusion.

Theorem 4. *For system* (1)*, the following statements are valid:*

(i) $R_0=1$ *if and only if* $r(\Phi_{F-V}(\omega))=1$,

(ii) $R_0>1$ *if and only if* $r(\Phi_{F-V}(\omega))>1$,

(iii) $R_0<1$ *if and only if* $r(\Phi_{F-V}(\omega))<1$,

where $F(t)$ *and* $V(t)$ *are defined in* (11).

It follows from Theorem 4 that the disease-free periodic solution $(S_h^*(t),0,S_v^*(t),0)$ of system (1) is asymptotically stable if $R_0<1$, and it is unstable if $R_0>1$.

In order to calculate R_0, we consider the following linear ω-periodic system:

$$\frac{dw}{dt}=\left(-V(t)+\frac{1}{\lambda}F(t)\right)w,\quad\lambda\in(0,\infty). \tag{15}$$

Let $W(t,s,\lambda)$, $t\geqslant s$, $s\in R$, be the evolution operator of the System (15) on R^2. Since $F(t)$ is nonnegative and $-V(t)$ is cooperative, then $r(W(\omega,0,\lambda))$ is continuous and nonincreasing for $\lambda\in(0,\infty)$, and $\lim_{\lambda\to\infty}r(W(\omega,0,\lambda))<1$. Thus, we have the following result, which will be used in our numerical calculation of the basic reproduction ratio R_0 in Section 5.

Lemma 5 (see [15]). *The following statements are valid.*

(i) *If* $r(W(\omega,0,\lambda))=1$ *has a positive solution,* λ_0 *is an eigenvalue of* L*, and hence* $R_0>0$.

(ii) *If* $R_0>0$*, then* $\lambda=R_0$ *is the unique solution of* $r(W(\omega,0,\lambda))=1$.

(iii) $R_0=0$ *if and only if* $r(W(\omega,0,\lambda))<1$ *for all* $\lambda>0$.

3. Global Stability of Disease-Free Periodic Solution

In this section we will prove the global asymptotical stability of the disease-free periodic solution $(S_h^*(t), 0, S_v^*(t), 0)$.

Let $N_h(t) = S_h(t) + I_h(t)$, $N_v(t) = S_v(t) + I_v(t)$. Denote

$$\begin{aligned}\Omega = \Big\{ & (S_h, I_h, S_v, I_v) \in R_+^4 \mid 0 \le S_h + I_h \le N_1 < +\infty, \\ & 0 \le S_v + I_v \le N_2 < +\infty \Big\},\end{aligned} \tag{16}$$

where $N_1 = \Lambda^L/\mu_1^M$ and $N_2 = mN_1$. Similar to [16, 17], we firstly prove the following lemmas.

Lemma 6. *Ω is a positively invariant set for* (1).

Proof. From the equations in (1), we have

$$\begin{aligned}\frac{dN_h(t)}{dt} &= \Lambda(t) - \mu_1(t) N_h(t) \\ &\le \Lambda^L - \mu_1^M N_h(t) \\ &\le 0 \quad \text{if } N_h(t) \ge N_1, \\ \frac{dN_v(t)}{dt} &= b(t) N_v \left(1 - \frac{N_v}{mN_h}\right) - \mu_2(t) I_v(t) \\ &\le b(t) N_v(t) \left(1 - \frac{N_v(t)}{mN_h}\right) \\ &\le 0 \quad \text{if } N_v(t) \ge N_2\end{aligned} \tag{17}$$

which implies that Ω is a positive invariant compact set for (1). The proof is completed. □

Lemma 7. *Let $(S_h(t), I_h(t), S_v(t), I_v(t))$ be any solution of system* (1). *It holds that*

$$\begin{aligned}\lim_{t\to+\infty} (N_h(t) - S_h^*(t)) &= 0, \\ \lim_{t\to+\infty} (N_v(t) - S_v^*(t)) &= 0,\end{aligned} \tag{18}$$

where $S_h^*(t)$, $S_v^*(t)$ are defined in Lemmas 2 and 3, respectively.

Proof. We denote that $y_1(t) = N_h(t) - S_h^*(t)$. It follows from the first equation of (17) that $dy_1(t)/dt \le -\mu_1(t) y_1(t)$, which implies that $\lim_{t\to+\infty} y_1(t) = \lim_{t\to+\infty}(N_h(t) - S_h^*(t)) = 0$. Further, from Lemma 6, we obtain that for any $\varepsilon > 0$, there exists a $T > 0$ such that

$$S_h^*(t) - \varepsilon \le N_h(t) \le S_h^*(t) + \varepsilon, \quad N_v(t) < N_2, \quad \forall t \ge T. \tag{19}$$

Let $y_2(t) = N_v(t) - S_v^*(t)$. From the second equation of (17) and (19), we get

$$\begin{aligned}\frac{dy_2(t)}{dt} &= b(t) N_v(t) \left[1 - \frac{N_v(t)}{mN_h(t)}\right] - \mu_2(t) I_v(t) \\ &\quad - b(t) S_v^*(t) \left[1 - \frac{S_v^*(t)}{mS_h^*(t)}\right] \\ &\le b(t) N_v(t) \left[1 - \frac{N_v(t)}{m(S_h^*(t) + \varepsilon)}\right] \\ &\quad - b(t) S_v^*(t) \left[1 - \frac{S_v^*(t)}{mS_h^*(t)}\right] \\ &= b(t) (N_v(t) - S_v^*(t)) \left[1 - \frac{S_h^*(t)}{S_h^*(t) + \varepsilon}\right] \\ &\quad - b(t) (N_v^*(t) - S_v^*(t)) \frac{N_v(t)}{m(S_h^*(t) + \varepsilon)} \\ &\quad + b(t) \frac{S_v^{*2}(t)\varepsilon}{mS_h^*(t)(S_h^*(t) + \varepsilon)} \\ &= -b(t) \frac{N_v(t)}{m(S_h^*(t) + \varepsilon)} y_2(t) + \Delta(\varepsilon)\end{aligned} \tag{20}$$

for all $t > T$, where

$$\begin{aligned}\Delta(\varepsilon) &= b(t) (N_v(t) - S_v^*(t)) \left[1 - \frac{S_h^*(t)}{S_h^* + \varepsilon}\right] \\ &\quad + b(t) \frac{S_v^{*2}(t)\varepsilon}{mS_h^*(t)(S_h^* + \varepsilon)}.\end{aligned} \tag{21}$$

Obviously, $\lim_{\varepsilon\to 0} \Delta(\varepsilon) = 0$. Because ε is arbitrarily small, then $\lim_{t\to+\infty} y_2(t) = \lim_{t\to+\infty}(N_v(t) - S_v^*(t)) = 0$. Hence, the proof is completed. □

Theorem 8. *The disease-free periodic solution $(S_h^*(t), 0, S_v^*(t), 0)$ is globally asymptotically stable if $R_0 < 1$, whereas it is unstable if $R_0 > 1$.*

Proof. From Theorem 4, we have that $(S_h^*(t), 0, S_v^*(t), 0)$ is unstable if $R_0 > 1$, and $(S_h^*(t), 0, S_v^*(t), 0)$ is locally stable if $R_0 < 1$. Therefore, we only need to show the global attractivity of $(S_h^*(t), 0, S_v^*(t), 0)$ for $R_0 < 1$.

Since $R_0 < 1$, by Theorem 4, we can choose $\epsilon_1 > 0$ sufficiently small such that

$$r\left(\Phi_{F-V+M_{\epsilon_1}}(\omega)\right) < 1, \tag{22}$$

where

$$M_{\epsilon_1}(t) = \begin{pmatrix} 0 & \epsilon_1 \\ \epsilon_1 & 0 \end{pmatrix}. \tag{23}$$

From Lemma 6 and (18), we have that, for above mentioned $\epsilon_1 > 0$, there exists a $T_1 > 0$ such that $S_h(t) \le S_h^*(t) + \epsilon_1$, $S_v(t) \le S_v^*(t) + \epsilon_1$ for $t > T_1$. It follows from the second and fourth equations that for $t > T_1$,

$$\begin{aligned}\frac{dI_h(t)}{dt} &\le \beta_1(t)(S_h^*(t) + \epsilon_1) I_v(t) - (\mu_1(t) + d(t)) I_h(t), \\ \frac{dI_v(t)}{dt} &\le \beta_2(t)(S_v^*(t) + \epsilon_1) I_h(t) - \mu_2(t) I_v(t).\end{aligned} \tag{24}$$

Consider the following comparison system:

$$\begin{aligned}\frac{d\widetilde{I}_h(t)}{dt} &= \beta_1(t)\left(S_h^*(t)+\epsilon_1\right)\widetilde{I}_v(t) - \left(\mu_1(t)+d(t)\right)\widetilde{I}_h(t),\\ \frac{d\widetilde{I}_v(t)}{dt} &= \beta_2(t)\left(S_v^*(t)+\epsilon_1\right)\widetilde{I}_h(t) - \mu_2(t)\widetilde{I}_v(t).\end{aligned} \tag{25}$$

In view of Lemma 1, we know that there exists a positive ω-periodic function $v_1(t)$ such that $J(t) \le v_1(t)\exp(p_1 t)$, where $J(t) = (\widetilde{I}_h(t), \widetilde{I}_v(t))^T$ and $p_1 = (1/\omega)\ln r(\Phi_{F-V+M_\epsilon}(\omega)) < 0$. It follows from (22) that $\lim_{t\to+\infty}\widetilde{I}_h(t) = 0$ and $\lim_{t\to+\infty}\widetilde{I}_v(t) = 0$. By the comparison of theorem [18], we have $\lim_{t\to+\infty} I_h(t) = 0$ and $\lim_{t\to+\infty} I_v(t) = 0$. From (18), we have

$$\begin{aligned}\lim_{t\to+\infty}\left(S_h(t)-S_h^*(t)\right) &= 0,\\ \lim_{t\to+\infty}\left(S_v(t)-S_v^*(t)\right) &= 0.\end{aligned} \tag{26}$$

Hence, the disease free periodic solution $(S_h^*(t), 0, S_v^*(t), 0)$ is globally attractive. This completes the proof. □

4. Permanence

In this section, we show that if $R_0 > 1$, then the disease persists.

Firstly, we define $X = \{(S_h, I_h, S_v, I_v) \in R_+^4\}$, $X_0 = \{(S_h, I_h, S_v, I_v) \in X : S_h \ge 0,\ I_h > 0,\ S_v \ge 0,\ I_v > 0\}$, and $\partial X_0 = X \backslash X_0$, and we denote $u(t, x_0)$ as the unique solution of System (1) with the initial value $x_0 = (S_h^0, I_h^0, S_v^0, I_v^0)$.

Define Poincaré map $P : X \to X$ associated with System (1) as follows:

$$P(x_0) = u(\omega, x_0), \quad \forall x_0 \in X. \tag{27}$$

By Lemma 6, it is easy to see that both X and X_0 are positively invariant and P is point dissipative. Set

$$\begin{aligned}M_\partial = \Big\{&\left(S_h^0, I_h^0, S_v^0, I_v^0\right) \in \partial X_0 \mid P^m\left(S_h^0, I_h^0, S_v^0, I_v^0\right) \in \partial X_0,\\ &m \in Z^+\Big\},\end{aligned} \tag{28}$$

where $Z^+ = \{0, 1, 2, \ldots\}$. We claim that

$$M_\partial = \left\{(S_h, 0, S_v, 0),\ S_h \ge 0,\ S_v \ge 0\right\}. \tag{29}$$

Obviously, $M_\partial \supseteq \{(S_h, 0, S_v, 0),\ S_h \ge 0,\ S_v \ge 0\}$. Next we want to show $M_\partial \setminus \{(S_h, 0, S_v, 0),\ S_h \ge 0,\ S_v \ge 0\} = \emptyset$. If it does not hold, then there exists a point $(S_h^0, I_h^0, S_v^0, I_v^0) \in M_\partial \setminus \{(S_h, 0, S_v, 0),\ S_h \ge 0,\ S_v \ge 0\}$.

Case 1. $I_h^0 = 0$ and $I_v^0 > 0$. It is obvious that $I_v(t) > 0$ and $S_h(t) > 0$ for any $t > 0$. Then, from the second equation of System (1), $dI_h(t)/dt|_{t=0} = \beta_1(0)S_h(0)I_v(0) > 0$ holds. It follows that $(S_h(t), I_h(t), S_v(t), I_v(t)) \notin \partial X_0$ for $0 < t \ll 1$. This is a contradiction.

Case 2. $I_h^0 > 0$ and $I_v^0 = 0$. It is obvious that $I_h(t) > 0$ and $S_v(t) > 0$ for any $t > 0$. Then, from the fourth equation of System (1), $dI_v(t)/dt|_{t=0} = \beta_2(0)S_v(0)I_h(0) > 0$ holds. It follows that $(S_h(t), I_h(t), S_v(t), I_v(t)) \notin \partial X_0$ for $0 < t \ll 1$. This is a contradiction.

That is to say, for any $(S_h^0, I_h^0, S_v^0, I_v^0) \notin \{(S_h, 0, S_v, 0) : S_h \ge 0,\ S_v \ge 0\}$, then $(S_h^0, I_h^0, S_v^0, I_v^0) \notin M_\partial$. Therefore we have $M_\partial = \{(S_h, 0, S_v, 0) : S_h \ge 0,\ S_v \ge 0\}$.

Next, we present the following result of the uniform persistence of the disease.

Theorem 9. *Suppose* $R_0 > 1$. *Then there is a positive constant* $\epsilon > 0$ *such that each positive solution* $(S_h(t), I_h(t), S_v(t), I_v(t))$ *of System* (1) *satisfies*

$$\liminf_{t\to+\infty} I_h(t) \ge \epsilon, \qquad \liminf_{t\to+\infty} I_v(t) \ge \epsilon. \tag{30}$$

Proof. By Theorem 4, we obtain $r(\Phi_{F-V}(\omega)) > 1$. So we can choose $\eta > 0$ small enough such that $r(\Phi_{F-V-M_\eta}) > 1$, where

$$M_\eta = \begin{pmatrix} 0 & \eta \\ \eta & 0 \end{pmatrix}. \tag{31}$$

Put $P_0 = \{S_h^*(0), 0, S_v^*(0), 0\}$. Now we proceed by contradiction to prove that

$$\limsup_{m\to+\infty} d\left(P^m\left(S_h^0, I_h^0, S_v^0, I_v^0\right), P_0\right) \ge \delta. \tag{32}$$

If it does not hold, then

$$\limsup_{m\to+\infty} d\left(P^m\left(S_h^0, I_h^0, S_v^0, I_v^0\right), P_0\right) < \delta \tag{33}$$

for some $(S_h^0, I_h^0, S_v^0, I_v^0) \in X_0$. Without loss of generality, suppose that

$$d\left(P^m\left(S_h^0, I_h^0, S_v^0, I_v^0\right), P_0\right) < \delta, \quad \forall m \in Z_+. \tag{34}$$

By the continuity of the solutions with respect to the initial values, we obtain

$$\begin{aligned}&\left\|u\left(t, P^m\left(S_h^0, I_h^0, S_v^0, I_v^0\right)\right) - u\left(t, P_0\right)\right\| \le \eta,\\ &\qquad \forall t \in [0, \omega], \quad \forall m \in Z_+.\end{aligned} \tag{35}$$

For any $t \ge 0$, there exists a $m \in Z_+$ such that $t = m\omega + t_1$, where $t_1 \in [0, \omega]$. Then we have

$$\begin{aligned}&\left\|u\left(t, \left(S_h^0, I_h^0, S_v^0, I_v^0\right)\right) - u\left(t, P_0\right)\right\|\\ &\quad = \left\|u\left(t_1, P^m\left(S_h^0, I_h^0, S_v^0, I_v^0\right)\right) - u\left(t_1, P_0\right)\right\| \le \eta\end{aligned} \tag{36}$$

for all $t \ge 0$, which implies that $S_h^*(t) - \eta \le S_h(t) \le S_h^*(t) + \eta$, $S_v^*(t) - \eta \le S_v(t) \le S_v^*(t) + \eta$. Then from (1) we have

$$\begin{aligned}\frac{dI_h(t)}{dt} &\ge \beta_1(t)\left(S_h^*(t) - \eta\right)I_v(t) - \left(\mu_1(t) + d(t)\right)I_h(t),\\ \frac{dI_v(t)}{dt} &\ge \beta_2(t)\left(S_v^*(t) - \eta\right)I_h(t) - \mu_2(t)I_v(t).\end{aligned} \tag{37}$$

Table 1: Parameter definitions and values used for numerical simulations of the Huanglongbing model.

Parameter	Definition	Average value	Unit	Reference
Λ	The recruitment rate of citrus	—	month^{-1}	Estimate
β_1	Infected rate of citrus	—	month^{-1}	Estimate
μ_1	Nature death rate of citrus	0.00275–0.004167	month^{-1}	[20]
d	Disease induced death rate of citrus	0.016667–0.027775	month^{-1}	[21]
D	Birth rate of psyllid	3.78327–33.526137	month^{-1}	[20, 22]
β_2	Infected rate of psyllid	—	month^{-1}	Estimate
μ_2	Nature death rate of psyllid	0.1169825–0.95052	month^{-1}	[23]
m	Max abundance of psyllid per citrus	120–1000	—	[24]

Table 2: Parameter functions for model (1) according to the values of Table 1.

Parameter functions	Value	Reference
$\beta_1(t)$	$0.0042925 + 0.003543\cos(2\pi t/12)$	Estimate
$\mu_1(t)$	$0.0034585 + 0.0007085\cos(2\pi t/12)$	[20]
$d(t)$	$0.022221 + 0.005554\cos(2\pi t/12)$	[21]
$D(t)$	$18.6547035 + 14.8714335\cos(2\pi t/12)$	[20, 22]
$\beta_2(t)$	$0.008779171 + 0.004838437\cos(2\pi t/12)$	Estimate
$\mu_2(t)$	$0.53375125 + 0.41676875\cos(2\pi t/12)$	[23]
$b(t) = D(t) - \mu_2(t)$	$18.120952 + 14.45466475\cos(2\pi t/12)$	[20, 22, 23]
m	560	[24]

Consider the linear system

$$\begin{aligned}\frac{d\widehat{I}_h(t)}{dt} &= \beta_1(t)\left(S_h^*(t)-\eta\right)\widehat{I}_v(t) - \left(\mu_1(t)+d(t)\right)\widehat{I}_h(t),\\ \frac{d\widehat{I}_v(t)}{dt} &= \beta_2(t)\left(S_v^*(t)-\eta\right)\widehat{I}_h(t) - \mu_2(t)\widehat{I}_v(t).\end{aligned} \tag{38}$$

By Lemma 1 and the standard comparison principle, we have that there exists a positive ω-periodic function $v_2(t)$ such that $J(t) = \exp(p_2 t)v_2(t)$ is a solution of System (38), where $J(t) = (\widehat{I}_h(t), \widehat{I}_v(t))^T$ and

$$p_2 = \frac{1}{\omega}\ln r\left(\Phi_{F-V-M_\eta}(\omega)\right). \tag{39}$$

It follows from $r(\Phi_{F-V-M_\eta}(\omega)) > 1$ that $p_2 > 0$ and $J(t) \to +\infty$ as $t \to +\infty$. Applying the comparison principle [18], we know that $I_h(t) \to +\infty$ and $I_v(t) \to +\infty$ as $t \to +\infty$. This is a contradiction. Thus, we have proved that (32) holds and P is weakly uniformly persistent with respect to $(X_0, \partial X_0)$. □

According to the results of Lemma 7, we can easily obtain that P has a global attractor P_0. It is easy to obtain that P_0 is an isolated invariant set in X and $W^s(P_0) \cap X_0 = \emptyset$. We know that P_0 is acyclic in M_∂ and every solution in M_∂ converges to P_0. According to Zhao [19], we have that P is uniformly persistent with respect to $(X_0, \partial X_0)$. This implies that the solution of (1) is uniformly persistent with respect to $(X_0, \partial X_0)$. Thus we have that there exists a $\epsilon > 0$ such that $\liminf_{t\to+\infty} I_h(t) \geq \epsilon$, $\liminf_{t\to+\infty} I_v(t) \geq \epsilon$.

5. Numerical Simulations and Sensitivity Analysis

In this section, we will make numerical simulations by means of Matlab 7.1 to support our theoretical results, to predict the trend of the disease, and to explore some control and prevention measures. Numerical values of parameters of system (1) are given in Table 1 (most of the data are taken from the literatures ([20–24])).

According to the periodicity of System (1) and Table 1, we set $\mu_1(t) = \alpha_1^0 + \alpha_2^0\cos(2\pi t/12)$, where $\alpha_2^0 = (0.004167 - 0.00275)/2 = 0.0007085$ and $\alpha_1^0 = 0.00275 + \alpha_2^0 = 0.0034585$. By the similar method, we can obtain the other parameter functions of model (1) (see Table 2). For the simulations that follows, we apply the parameters in Table 2 unless otherwise stated.

Choose $\Lambda(t) = 0.00265 + 0.00235\cos(2\pi t/12)$. Then from Lemma 5, we can compute $R_0 = 0.9844 < 1$ by means of Matlab 7.1. From Theorem 8 we obtain that the infected citrus population $I_h(t)$ and the infected psyllid population $I_v(t)$ of system (1) are extinct (see Figures 1 and 2).

Choose $\Lambda(t) = 0.005 + 0.0035\cos(2\pi t/12)$. Then from Lemma 5, we obtain that $R_0 = 1.8342 > 1$. From Theorem 9 we have that the infected citrus population $I_h(t)$ and the infected psyllid population $I_v(t)$ of System (1) are permanence (see Figures 3 and 4).

From the formulae for R_0, we can predict the general tendency of the epidemic in a long term according to the current situation, which is presented in Figures 1, 2, 3, and 4. From the first two figures we know that the epidemic of

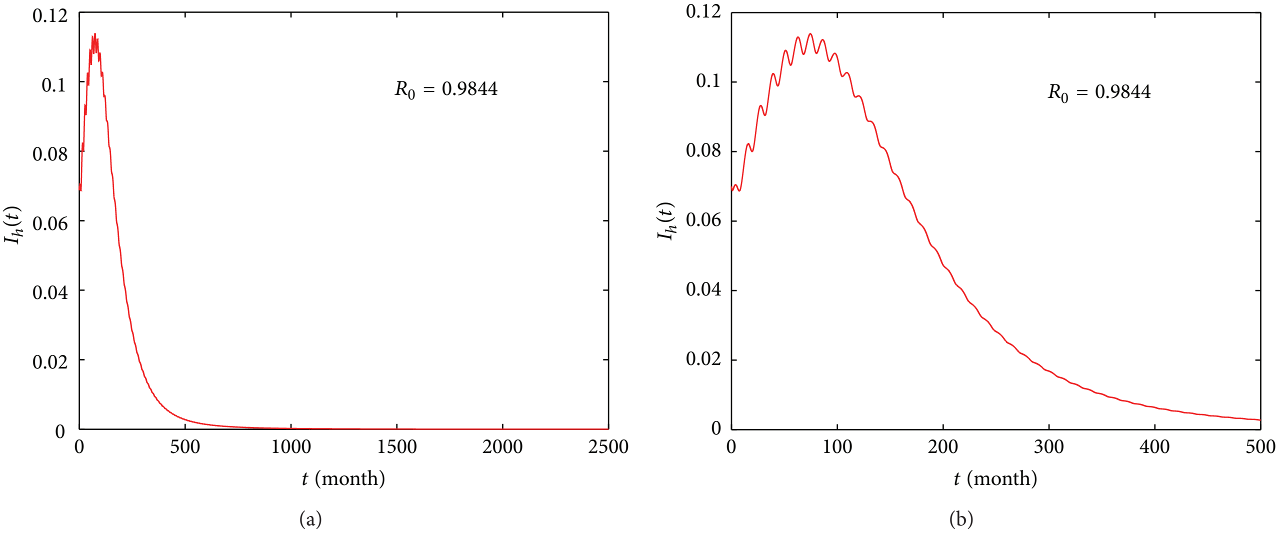


Figure 1: Graphs of numerical simulations of (1) showings the tendency of the infected citrus population. (a) $t \in [0, 2500]$; (b) $t \in [0, 500]$.

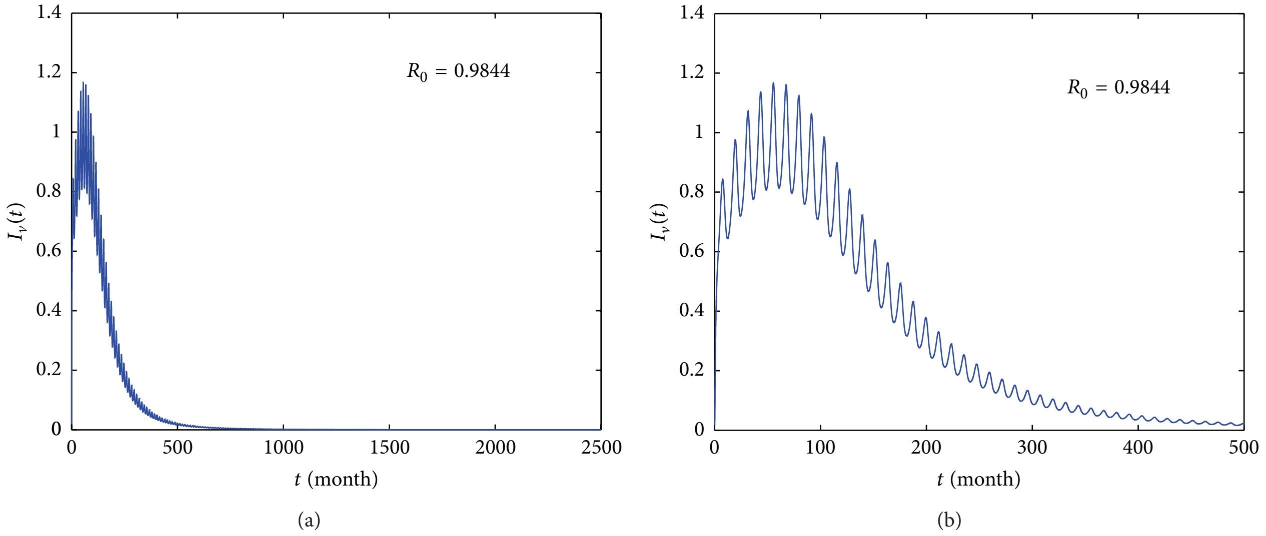


Figure 2: It is similar to Figure 1.

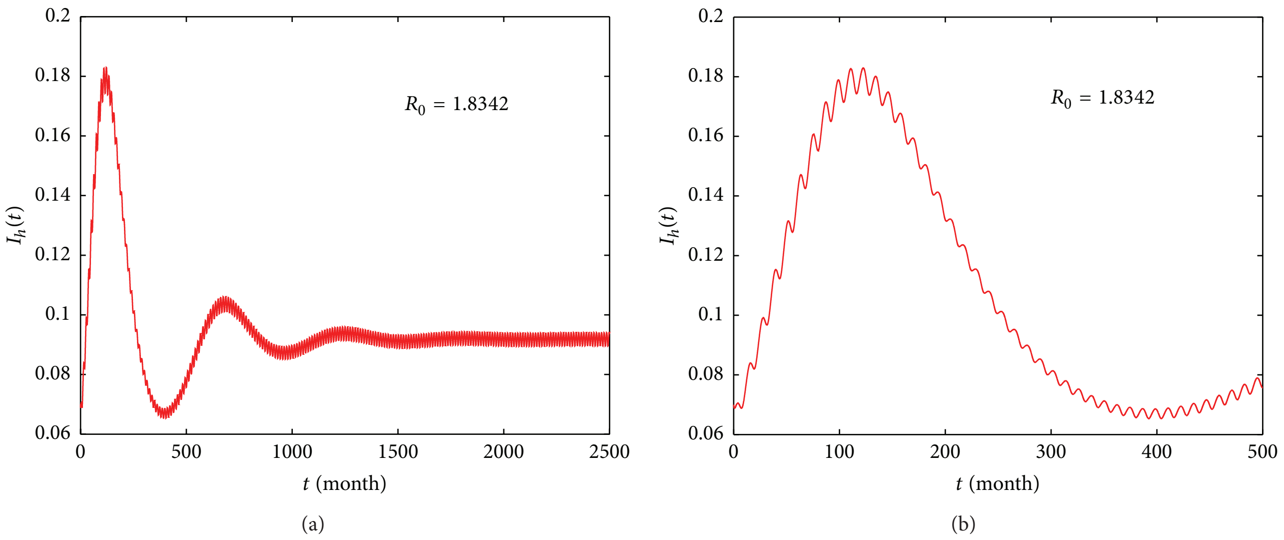
Huanglongbing can be rising in a short time but cannot be outbreak with the current prevention and control measures. From Figures 3 and 4, we can see that the epidemic of Huanglongbing dropped heavily after 100 months, while there is still tendency to a stable periodic solution in a long time.

Next, we perform some sensitivity analysis to determine the influence R_0 on the parameters $\Lambda(t)$, $\beta_1(t)$, and $\beta_2(t)$.

We choose function $\Lambda(t) = \Lambda_1^0 + \Lambda_2^0 \cos(2\pi t/12)$, where Λ_1^0, Λ_2^0 denote the average and amplitude values of $\Lambda(t)$, respectively, and $\Lambda_1^0 = (1/12)\int_0^{12} \Lambda(t)dt$. From Figure 5, we can observe that the blue line is linear relation between R_0 and Λ_2^0, and R_0 increases as Λ_2^0 increases. The red curve reflects the influence of the average value of $\Lambda(t)$ on R_0. Figure 5 shows that Λ_1^0 is more sensitive than Λ_2^0 on the basic reproduction number R_0. Therefore, in the real world, decreasing the average recruitment rate of citrus is the valuable way to control Huanglongbing.

Now, we consider the combined influence of $\beta_1(t)$ and $\beta_2(t)$ on R_0. Set $\Lambda(t) = 0.0027 + 0.00235\cos(2\pi t/12)$, $\beta_1(t) = a_1 + b_1\cos(2\pi t/12)$ and $\beta_2(t) = a_2 + b_2\cos(2\pi t/12)$. Moreover, we know that $a_1 = (1/12)\int_0^{12} \beta_1(t)dt$ and $a_2 = (1/12)\int_0^{12} \beta_2(t)dt$. Other parameters can be seen in Table 2.

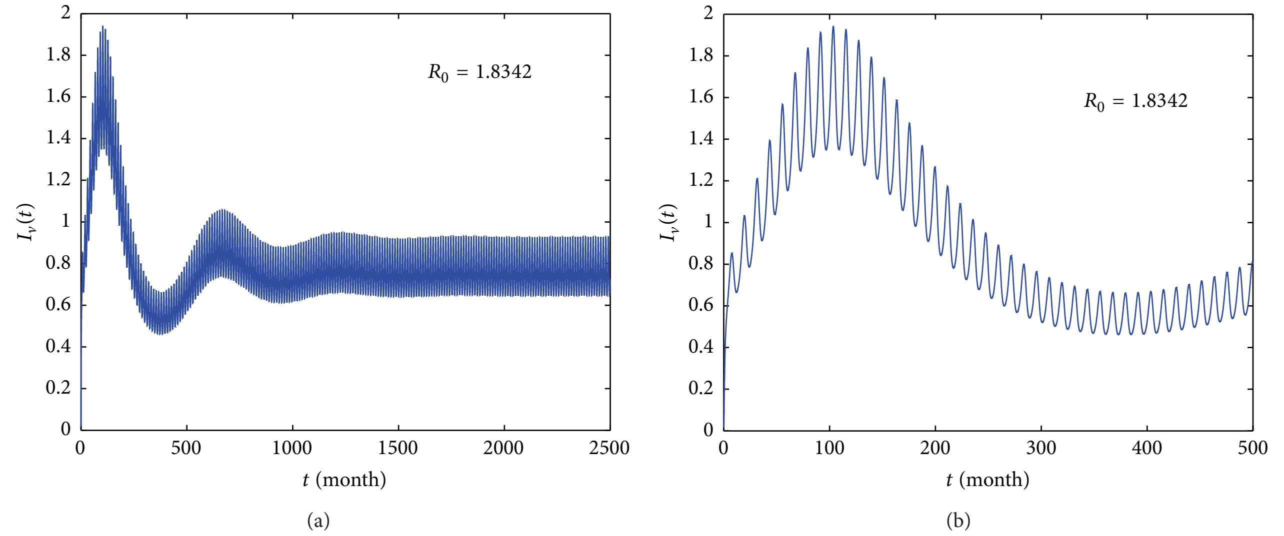
Case (I). We fix $b_1 = 0.003543$ and $b_2 = 0.004838437$, and let a_1 vary from 0.00001 to 0.015 and a_2 from 0.00001 to 0.02. For this case, it is interesting to examine how the average values of adequate contact rate $\beta_1(t)$ and $\beta_2(t)$ affect the basic reproduction number R_0. Numerical results shown



(a)

(b)

FIGURE 3: The figures show that the infected citrus population is permanence. (a) $t \in [0, 2500]$; (b) $t \in [0, 500]$.



(a)

(b)

FIGURE 4: It is similar to Figure 3.

in Figure 6 imply that the basic reproduction number R_0 may be less than 1 when a_1 or a_2 is small enough. And the results also imply that R_0 increases as a_1 and a_2 increase. Further, we can observe that from Figure 6(i)the smaller the values of a_1 or a_2 are, the more sensitive R_0 is; (ii) increasing a_2 may be more sensitive for R_0 when a_1 is fixed; (iii) increasing a_1 may be more sensitive for R_0 when a_2 is fixed.

Case (II). We fix $a_1 = 0.0042925$ and $a_2 = 0.00877917$, and let b_1 vary from 0.000001 to 0.005 and b_2 from 0.000002 to 0.006. Then we obtain the result of numerical simulation and it is shown in Figure 7. Obviously, Figure 7 shows that R_0 is linearly related to both b_1 and b_2 with the pattern that R_0 decreases to a relatively small value (less than 1) only when b_1 and b_2 are very small.

By the above graphs of the basic reproduction number R_0 on the average values of recruitment rate of citrus $\Lambda(t)$ and adequate contact rate $\beta_1(t)$, $\beta_2(t)$, we know that the basic reproduction number R_0 is a monotonic increasing function by the average values. From the sensitivity analysis diagrams, we observe that R_0 falls to less than 1 by decreasing the values of those parameters.

6. Conclusion

In this paper, we have analyzed a HLB transmission model with Logistic growth in periodic environments. It is proved that R_0 is the threshold for distinguishing the disease extinction or permanence. The disease-free periodic solution is

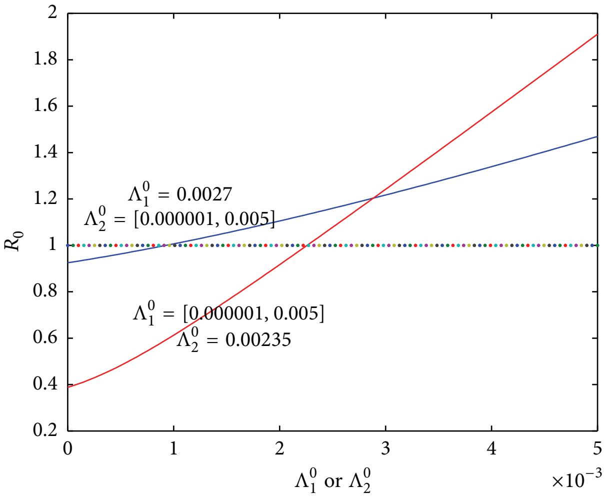


FIGURE 5: The graph shows the sensitivity of the basic reproduction number R_0 to the changes of $\Lambda(t)$.

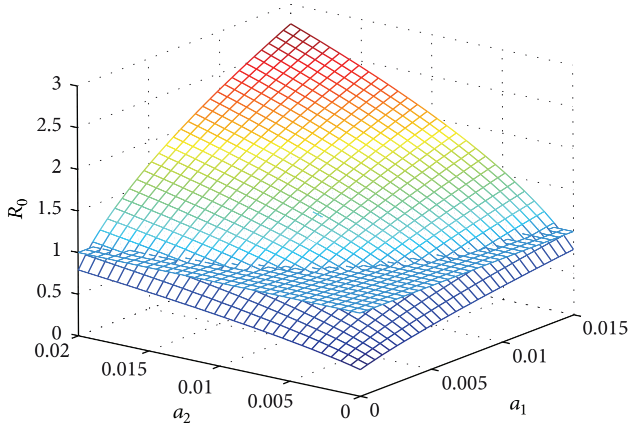


FIGURE 6: The graph of R_0 in terms of a_1 and a_2.

globally asymptotically stable and the disease dies out when $R_0 < 1$. When $R_0 > 1$, the disease persists.

The numerical simulations shown in Figure 5 show that there are some parameter ranges of Λ_1 and Λ_2 such that the threshold parameter R_0 is smaller than 1. It indicates a useful way to eradicate Huanglongbing by limiting the recruitment of citrus, including the average value and amplitude of recruitment function.

The results shown in Figure 6 (Figure 7) show that if the amplitudes of infected functions b_1, b_2 (the average infected rate a_1, a_2) are fixed, we can control the infection of citrus and psyllid by limiting the average infected rates a_1, a_2 (the amplitudes of infected functions b_1, b_2).

According to the above theoretical analysis and numerical simulations, we can conclude that the recruitment of citrus and the infection of citrus and psyllid have significant effects on Huanglongbing transmission. In order to prevent the epidemic disease from generating endemic, making an appropriate reduction of the recruitment rate of citrus and decreasing the contact rate between psyllid and the citrus are effective measures to control Huanglongbing.

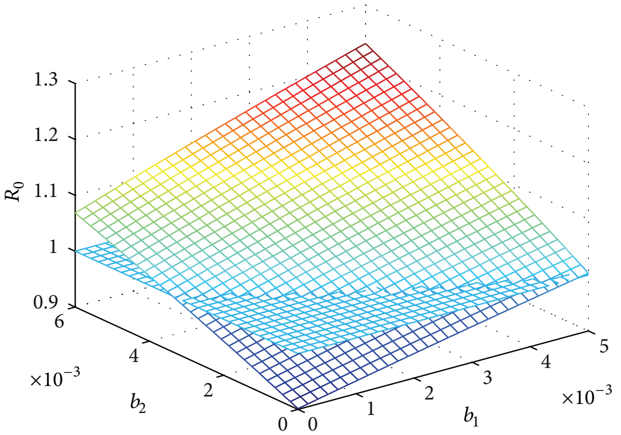


FIGURE 7: The graph of R_0 in terms of b_1 and b_2.

Disclosure

The paper is approved by all authors for publication. The authors would like to declare that the work described was original research that has not been published previously and not under consideration for publication elsewhere.

Conflict of Interests

No conflict of interests exists in the submission of this paper.

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