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VARYING PULSE CONTROL SCHEMES FOR CITRUS HUANGLONGBING EPIDEMIC MODEL WITH GENERAL INCIDENCE

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Abstract. In this paper a mathematical model for citrus Huanglongbing transmission including impulsive roguing control strategy and general incidence is proposed and analyzed. The global dynamics of disease-free periodic solution and the permanence of the system are investigated. Numerical simulations support our analytical conclusions.

Keywords: Citrus Huanglongbing model; Varying pulse control; General incidence; Extinction; Permanence.

2010 AMS Subject Classification: 34K45, 34K25.

1. Introduction

Huanglongbing (HLB), a vector-transmitted bacterial infection and caused by the bacteria Candidatus Liberibacter spp., is becoming one of the most serious problem of citrus worldwide. The report from the University of Florida's Institute of Food and Agricultural Sciences showed that HLB had caused 3.63 billion dolors in lost revenue and over 6,000 lost jobs in the state

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of Florida from 2006 to 2011 [1]. Since HLB was first discovered in China in the late 1800s [2], it is known to occur in African, Oceanian, South and North American countries and so on. HLB scarcely cures and affects all citrus varieties. Once the susceptible citrus is infected, then the infected citrus shows a blotchy mottle condition of the leaves that result in the development of yellow shoots the early and very characteristic symptom of the disease and moreover it can reduce the productivity of orchards, even poor quality and infection leads to plant death in extreme cases [3]. So HLB has become an important issue to study.

HLB is transmitted mainly by the psyllid Diaphorina citri, known as the Asian citrus psyllid [4]. As a general rule, the bacteria are carried in the saliva of a psyllid and when an infected psyllid feeds on the leaves of a tree, it passes the disease to the phloem within the veins of that tree. Similarly, a healthy psyllid can acquire the infection by feeding on an infected tree. Besides, we also find that a single psyllid, lasting 45 days, will lay around 800 eggs during its lifetime. It means that the population of psyllid is very large and at the same time, we need better control methods to treat and prevent the disease or predict the incidence trends. Actually, mathematical models have been an important research tool to understand the dynamics of vector-transmitted plant pathogens [3-5]. The applications of mathematical approach to plant epidemics were reviewed by Van der Plank [5] and Kranz [6].

To predict the spread of HLB and explore the effective control strategies, many continuous mathematical models have been established. A mathematical model of the transmission of HLB between its psyllid vector and citrus host [7] was developed to characterize the dynamics of the vector and disease development, focusing on the spread of the pathogen from flush to flush within a tree. Dynamics of vector and host populations were simulated. The result showed that the effect of spraying of psyllids depends on time of initial spraying, frequency, and efficacy of the insecticides. Raphael et al. [8] proposed a model for HLB spread between citrus plants which a delay period on the nymphal stage of Diaphorina citri and human intervention were taken into consideration. Numerical simulations were performed to assess the possible impacts of human detection efficiency of symptomatic plants, as well as the influence of a long incubation period of HLB in the plant. To study the impact of seasonal activity of psyllid on the dynamics of Huanglongbing (HLB) infection, a HLB mathematical model with periodic environment

[9] was developed. By the concept of next generation matrix, the basic reproduction number R_0 was obtained, and proofed that the disease-free periodic solution is globally asymptotically stable if $R_0 < 1$, whereas the disease persists if $R_0 > 1$.

However, the common assumption about the continuity of control activities is contradictory for the reality that the control behavior usually occurs in regular pulses [10-12]. For example, for the spread of citrus tristeza disease, Fishman and Marcus [13] investigated a model with periodic removals and mainly discussed the case of two interacting populations where infection can be transmitted from one population to another. By analyzing the properties of solutions of the model, they studied the effectiveness of removal. In this paper, according to the above biological background, we develop a time-varying impulsive control model with general nonlinear incidence rate, in which removing infected plants at fixed moments is considered.

This paper is structured as follows. In the next section, we mainly investigate a mathematical model with general nonlinear incidence rate and time-varying pulse control, under some assumptions and the biological interpretation. In Sections 3, we show that global attractivity of the disease-free periodic solution is determined by the threshold parameter R_1. In Section 4, we give another expression of threshold parameter R_2, and show that if $R_2 > 1$, the disease is permanence. Numerical simulations which demonstrate the theoretical analysis and a brief conclusion are given in the last section.

2. **Model formulation and preliminary**

The total citrus population is divided into two groups: susceptible citrus (S) and infected citrus (I). Based on the above works, now we establish an impulsive model with general incidence. The system is modeled by the following equations:

$$\begin{cases} \left.\begin{aligned} \frac{dS(t)}{dt} &= \alpha(K - S(t) - I(t)) - f(t,S,I) - \mu S(t), \\ \frac{dI(t)}{dt} &= f(t,S,I) - \mu I(t) - \gamma I(t), \end{aligned}\right\} \ t \neq t_n, \\ \left.\begin{aligned} S(t^+) &= S(t), \\ I(t^+) &= (1-\theta_n) I(t), \end{aligned}\right\} \ t = t_n, (n \in N). \end{cases} \tag{2.1}$$

The model is derived from the following assumptions.

• $S(t)$ and $I(t)$ are left continuous for $[t_0,+\infty)$, that is, $S(t)=\lim_{h\to 0^+} S(t-h)$ and $I(t)=\lim_{h\to 0^+} I(t-h)$.

• There is maximum plant population size $K>0$. Recruitment to the population is by replanting at a rate proportional $\alpha>0$ to the difference between the actual number of plants present $S+I$ and maximum population size K.

• $\mu>0$ denotes the natural death rate of susceptible and infected citrus, $\gamma\geq 0$ is the HLB-induced death rate.

• There exist a positive integer r and positive number ω such that $t_{n+r}=t_n+\omega$ for all $n\in N$. And θ_n $(0\leq\theta_n<1)$ is the proportion of infected citrus removed at each fixed time $t=t_n$, and $\theta_k=\theta_{nr+k}$ for $k=1,2,\cdots,r$.

• The general nonlinear incidence rate $f(t,S,I)$ is a piecewise continuous, nonnegative, periodic function with period ω. The form of $f(t,\ S,I)$ is as follows:

$$f(t,S,I)=\begin{cases} f_1(t,S,I), & t\in(n\omega+t_0,\ n\omega+t_1],\\ \quad\vdots & \\ f_r(t,S,I), & t\in(n\omega+t_{r-1},\ n\omega+t_r], \end{cases} \tag{2.2}$$

for all integer $n\geq 0$, and $f_k(t,0,I)=f_k(t,S,0)=0$ for $k=1,\ 2,\cdots,\ r$.

3. Global attractivity of the disease-free periodic solution

From system (2.1), we can easily obtain that the solution $(\alpha K/(\alpha+\mu),\ 0)$ is the disease-free periodic solution. To discuss the attractivity of the disease-free periodic solution of system (2.1), we firstly assume the following hypothesis:

(A): There exist positive, continuous, periodic functions $\beta_k(t)$ with period ω, such that $f_k(t,S,I)\leq\beta_k(t)SI$, for $k=1,2,\cdots,r$ and $t\geq t_0$.

Theorem 1. *If $R_1 < 1$ and system (2.1) satisfies the hypothesis (A), then the disease-free periodic solution $(\alpha K/(\alpha+\mu),\ 0)$ is globally attractive, where*

$$R_1 = \frac{\dfrac{\alpha K}{\alpha+\mu}\displaystyle\sum_{i=1}^{r}\int_{t_{i-1}}^{t_i}\beta_i(t)\,dt}{\omega(\mu+\gamma)-\displaystyle\sum_{i=1}^{r}\ln(1-\theta_i)}. \tag{3.1}$$

Proof. Let $(S(t),\ I(t))$ be any solution of system (2.1). Since $R_1 < 1$, we can choose a sufficiently small number $\varepsilon_1 > 0$ such that

$$\Lambda \triangleq \exp\left[\sum_{i=i}^{r}\int_{t_{i-1}}^{t_i}[\beta_i(t)(\frac{\alpha K}{\alpha+\mu}+\varepsilon_1)]dt+\sum_{i=i}^{r}\ln(1-\theta_i)-\omega(\mu+\gamma)\right]<1, \tag{3.2}$$

From the first equation of (2.1), we have $\frac{dS(t)}{dt} \le \alpha(K-S(t))-\mu S(t)$. By the comparison theorem, we can get that there exists a constant $t^1(>t_0)$ such that

$$S(t) < \frac{\alpha K}{\alpha+\mu}+\varepsilon_1, \quad \text{for all } t \ge t^1. \tag{3.3}$$

It follows from (3.3) and the second equation of system (2.1) that, for $t \in (n\omega+t_{k-1}, n\omega+t_k]$ $(k=1,\ 2,\ \cdots,\ r)$ and $t \ge t^1$,

$$\begin{aligned}\frac{dI(t)}{dt} &= f(t,S,I)-(\mu+\gamma)I(t)\\ &\le \beta_k(t)S(t)I(t)-(\mu+\gamma)I(t)\\ &\le \left[\beta_k(t)(\frac{\alpha K}{\alpha+\mu}+\varepsilon_1)-(\mu+\gamma)\right]I(t).\end{aligned}$$

Thus,

$$\begin{aligned}I(t) &\le I((n\omega+t_{k-1})^+)\exp\left(\int_{n\omega+t_{k-1}}^{t}\left[\beta_k(\tau)(\frac{\alpha K}{\alpha+\mu}+\varepsilon_1)-(\mu+\gamma)\right]d\tau\right)\\ &= (1-\theta_{k-1})I(n\omega+t_{k-1})\exp\left(\int_{t_{k-1}+n\omega}^{t}\left[\beta_k(\tau)(\frac{\alpha K}{\alpha+\mu}+\varepsilon_1)-(\mu+\gamma)\right]d\tau.\right)\end{aligned}$$

By using the similar method, we can deduce that for $t \in (n\omega+t_{k-1}, n\omega+t_k]$

$$\begin{aligned}I(t) &\le \prod_{i=1}^{k-1}(1-\theta_i)I((n\omega+t_0)^+)\\ &\times\exp\left\{(\frac{\alpha K}{\alpha+\mu}+\varepsilon_1)\left[\int_{n\omega+t_0}^{n\omega+t_1}\beta_1(\tau)d\tau+\cdots+\int_{n\omega+t_{k-1}}^{t}\beta_k(\tau)d\tau\right]-(\mu+\gamma)(t-n\omega-t_0)\right\}.\end{aligned} \tag{3.4}$$

Especially, when $t=(n+1)\omega+t_0$, we have

$$\begin{aligned}
&I(((n+1)\omega+t_0)^+)=I((n\omega+t_r)^+)=(1-\theta_r)I(n\omega+t_r)\\
&\le\prod_{i=1}^{r}(1-\theta_i)I((t_0+n\omega)^+)\exp\left[\sum_{i=1}^{r}\int_{t_{i-1}}^{t_i}[\beta_i(\tau)(\frac{\alpha K}{\alpha+\mu}+\varepsilon_1)]d\tau-(t_r-t_0)(\mu+\gamma)\right]\\
&=I((n\omega+t_0)^+)\exp\left[\sum_{i=1}^{r}\int_{t_{i-1}}^{t_i}[\beta_i(\tau)(\frac{\alpha K}{\alpha+\mu}+\varepsilon_1)]d\tau+\sum_{i=1}^{r}\ln(1-\theta_i)-\omega(\mu+\gamma)\right]\\
&=\Lambda I((n\omega+t_0)^+).
\end{aligned}$$

Thus, for any positive integer q, we have $I(((n+q)\omega+t_0)^+)\le\Lambda^q I((n\omega+t_0)^+)$. It follows from (3.2) that

$$I[((n+q)\omega+t_0)^+]\to 0,\ \text{as } q\to\infty. \tag{3.5}$$

From (3.4) and (3.5), we have

$$\lim_{t\to\infty}I(t)=0 \tag{3.6}$$

Therefore, for above mentioned ε_1, there exists t^2 $(>t^1)$, we have

$$I(t)<\varepsilon_1 \quad \text{for all} \quad t>t^2. \tag{3.7}$$

From the first equation of system (2.1) and (3.7), we have for $t>t^2$,

$$\begin{aligned}
\frac{dS(t)}{dt}&=\alpha(K-S(t)-I(t))-f(t,S,I)-\mu S(t)\\
&\ge\alpha(K-\varepsilon_1)-(\alpha+\mu+\beta^*\varepsilon_1)S(t),
\end{aligned}$$

where $\beta^*=\max\{\beta_i(t),\ t_0\le t\le t_0+\omega,\ i=1,\cdots,r\}$. Solving the above differential inequality, we have

$$S(t)\ge\frac{\alpha(K-\varepsilon_1)}{\alpha+\mu+\beta^*\varepsilon_1}+\left[S(t^2)-\frac{\alpha(K-\varepsilon_1)}{\alpha+\mu+\beta^*\varepsilon_1}\right]e^{-(\alpha+\mu+\beta^*\varepsilon_1)(t-t^2)}\doteq\hat{S}(t),\quad \text{for } t>t^2. \tag{3.8}$$

From (3.3) and (3.8), we have

$$\hat{S}(t)\le S(t)\le\frac{\alpha K}{\alpha+\mu}+\varepsilon_1,\quad \text{for } t>t^2. \tag{3.9}$$

Because ε_1 is arbitrarily small, (3.9) implies that

$$\lim_{t\to\infty}S(t)=\frac{\alpha K}{\alpha+\mu} \tag{3.10}$$

From (3.6) and (3.10), we obtain that the disease-free periodic solution $(\alpha K/(\alpha+\mu),0)$ is global attractive.

□

4. **Permanence**

In this section, we mainly obtain the sufficient conditions for the permanence of system (2.1). So we give the following hypothesis (B) at first.

(B): There exist positive, continuous, periodic functions $\varphi_k(t)$ with period ω, such that $f_k(t,S,I) \geq \varphi_k(t)SI$, for $k = 1,2,\cdots,r$ and $t \geq t_0$.

Theorem 2. *If $R_2 > 1$ and system (2.1) satisfies the hypotheses (A) and (B), then system (2.1) is permanent, where*

$$R_2 = \frac{\dfrac{\alpha K}{\alpha+\mu}\displaystyle\sum_{i=1}^{r}\int_{t_{i-1}}^{t_i}\varphi_i(t)\,dt}{(\mu+\gamma)\omega - \displaystyle\sum_{i=1}^{r}\ln(1-\theta_i)}.$$

Proof. Since $R_2 > 1$, we can easily see that there exists a sufficiently small $\varepsilon > 0$ such that

$$\Omega \doteq \prod_{i=1}^{r}(1-\theta_i)\exp\left[\left(\frac{\alpha(K-\varepsilon)}{\beta^*\varepsilon+\alpha+\mu}-\varepsilon\right)\sum_{i=1}^{r}\int_{t_{i-1}}^{t_i}\varphi_i(t)dt-(\mu+\gamma)\omega\right] > 1. \tag{4.1}$$

In order to illustrate the conclusion, we firstly obtain the disease is uniformly weakly persistent, that is, there exists a positive constant $\eta > 0$, such that $\limsup_{t\to+\infty} I(t) \geq \eta$. By contradiction, we have that for above given $\varepsilon > 0$, there exists a $t^3 > 0$ such that $I(t) < \varepsilon$ for all $t > t^3$.

In view of the hypothesis (A) and the first equation of system (2.1), we have

$$\frac{dS(t)}{dt} = \alpha(K-S(t)-I(t)) - f(t,S,I) - \mu S(t) \geq \alpha(K-\varepsilon) - (\beta^*\varepsilon+\alpha+\mu)S(t) \ \text{ for all } t > t^3,$$

where $\beta^* = \max\{\beta_i(t),\ t_0 \leq t \leq t_0+\omega,\ i = 1,\ \cdots,\ r\}$.

By comparison theorem, we have $S(t) \geq y_1(t)$ and $y_1(t) \to \dfrac{\alpha(K-\varepsilon)}{\beta^*\varepsilon+\alpha+\mu}$ as $t \to +\infty$, where $y_1(t)$ is the solution of the following comparison system:

$$\frac{dy_1(t)}{dt} = \alpha(K-\varepsilon) - (\beta^*\varepsilon+\alpha+\mu)y_1(t).$$

Therefore, for above mentioned ε, there exists a $n^* > 0$, such that

$$S(t) \geq y_1(t) \geq \frac{\alpha(K-\varepsilon)}{\beta^*\varepsilon+\alpha+\mu} - \varepsilon \quad \text{for all } t > t^3 + n^*\omega. \tag{4.2}$$

For above mentioned $t^3 + n^*\omega$, we know that there exists a positive integer n_1 such that $n_1\omega \geq t^3 + n^*\omega$. Then, for all $n\omega + t_{s-1} < t < n\omega + t_s$ ($n \geq n_1$, $s = 1, \cdots, r$), by (4.2) and the second equation of system (2.1) yields

$$\begin{aligned} \frac{dI(t)}{dt} &= f(t,S,I) - (\mu+\gamma)I(t) \\ &\geq \left[\left(\frac{\alpha(K-\varepsilon)}{\beta^*\varepsilon+\alpha+\mu} - \varepsilon\right)\varphi_s(t) - (\mu+\gamma)\right] I(t), \end{aligned} \tag{4.3}$$

Consider the following auxiliary impulsive system:

$$\begin{cases} \dfrac{dy_2(t)}{dt} = \left[\left(\dfrac{\alpha(K-\varepsilon)}{\beta^*\varepsilon+\alpha+\mu} - \varepsilon\right)\varphi_s(t) - (\mu+\gamma)\right] y_2(t), \ \ n\omega + t_{s-1} < t < n\omega + t_s, \\ y_2(t^+) = (1-\theta_s)y_2(t), \ \ t = n\omega + t_s, \\ y_2(t_0^+) = I_0 > 0. \end{cases} \tag{4.4}$$

Calculating (4.4), we derive that for $n\omega + t_{s-1} < t \leq n\omega + t_s$ ($n \geq n_1$, $s = 1, \cdots, r$)

$$y_2(t) = I_0\Omega^n \times \prod_{i=1}^{s-1}(1-\theta_i) \exp\left[\left(\frac{\alpha(K-\varepsilon)}{\beta^*\varepsilon+\alpha+\mu} - \varepsilon\right)\sum_{i=1}^{s-1}\int_{t_{i-1}}^{t_i}\varphi_i(t)dt - (\mu+\gamma)(t-n\omega-t_0)\right], \tag{4.5}$$

From (4.1) and (4.5), we have

$$y_2(t) \to \infty, \text{ as } n \to \infty.$$

That is to say, as $t \to \infty$, we get $y_2(t) \to \infty$. By the comparison theorem we have $\lim\limits_{t\to\infty} I(t) = \infty$, which is a contradiction to $0 < I(t) < \varepsilon$. Thus the claim is proved, that is, there is a $\eta > 0$ such that $\limsup\limits_{t\to\infty} I(t) \geq \eta$.

By the claim, we are left the following two possibilities:

Case 1. $I(t) > \varepsilon$ for all large t;

Case 2. $I(t)$ oscillates about ε for all large t.

The conclusion is evident in the first case. Next we will consider the second possibility. At first, let $\underline{t}$ and $\bar{t}$ be large enough such that

$$I(\underline{t}) \geq \varepsilon, \quad I(\bar{t}) = \varepsilon, \quad \text{and } I(t) < \varepsilon, \text{ for } t \in (\underline{t}, \bar{t}).$$

There are two possible cases for $\underline{t}$.

Case A. If $\underline{t} = t_k + n\omega$ (n is a positive integer and $k = 1, \cdots, r$), then $I(\underline{t}) > \varepsilon$ and $(1-\theta_k)\varepsilon < I(\underline{t}^+) = (1-\theta_k)I(\underline{t}) < \varepsilon$. We claim that there must exists a positive constant m, such that $I(t) \geq m,$ for $t \in (\underline{t},\, \bar{t})$. Flowing, we will consider two possible subcases in term of the size of $\underline{t}$ and $\bar{t}$.

(a) If $\bar{t} - \underline{t} \leq n^*\omega$ (where n^* is defined in (4.2)), then from system (2.1), we have

$$\begin{cases} \dfrac{dI(t)}{dt} = f(t,S,I) - (\mu+\gamma)I(t) \\ \qquad\qquad \geq -(\mu+\gamma)I(t), \;\; t \neq t_n, \\ I(t^+) = (1-\theta_n)I(t), \;\; t = t_n, \end{cases} \tag{4.6}$$

It follows from (4.6) that

$$I(t) \geq \left[\prod_{i=1}^{r}(1-\theta_i)\right]^{n^*+1} \varepsilon \exp[-(\mu+\gamma)n^*\omega] \doteq m \quad \text{for all } t \in [\underline{t},\, \bar{t}].$$

(b) If $\bar{t} - \underline{t} > n^*\omega$, then from the discussion in subcase (a), we have $I(t) \geq m$ for all $t \in [\underline{t},\, \underline{t} + n^*\omega]$. Next, we show that $I(t) \geq m$ for all $t \in (\underline{t} + n^*\omega,\, \bar{t}]$. Otherwise, there exists a constant $t^* > 0$ such that

$$\begin{aligned} &I(t) \geq m, \qquad \text{for all } t \in [\underline{t},\, \underline{t} + t^* + n^*\omega), \\ &I(\underline{t} + t^* + n^*\omega) \geq m, \;\; I(t) < m, \quad \text{for } 0 < t - (\underline{t} + t^* + n^*\omega) \ll 1. \end{aligned}$$

On the other hand, similar to discussion in subcase (a), it is easy to know that we can choose a proper $\rho > 0$, such that $I(\underline{t} + t^* + n^*\omega) \geq \rho I(\underline{t}) \exp[-(\mu+\gamma)(n^*\omega + t^*)] > m$. Since $e^{-(\mu+\gamma)t}$ is a continuous function, that is $\rho I(\underline{t}) \exp\{-(\mu+\gamma)t\} \geq m$ for $0 < t \ll 1$ hold. Then for $0 < t - (\underline{t} + t^* + n^*\omega \ll 1)$, we have

$$\begin{aligned} I(t) &\geq I(\underline{t} + t^* + n^*\omega) \exp\{-(\mu+\gamma)(t - \underline{t} - t^* - n^*\omega)\} \\ &\geq \rho I(\underline{t}) \exp[-(\mu+\gamma)(\underline{t} + n^*\omega + t^* - \underline{t})] \exp\{-(\mu+\gamma)(t - \underline{t} - t^* - n^*\omega)\} \\ &\geq m \end{aligned}$$

Then, $I(t) \geq m$, for $0 < t - (\underline{t} + t^* + n^*\omega) \ll 1$, which is a contraction. Therefore, $I(t) \geq m$ for any $t \in [\underline{t},\, \bar{t}]$.

Case B. If $\underline{t} \neq t_k + n\omega$, then $I(\underline{t}) = \varepsilon$. Using the analogous methods of Case A, we can easily

get $I(t) \geq \left[\prod_{i=1}^{r}(1-\theta_i)\right]^{n^*+1} \varepsilon \exp[-(\mu+\gamma)n^*\omega]$=m, for all $t \in [\underline{t},\ \bar{t}]$.

Thus, we see that $I(t) \geq m$ for any $t \in [\underline{t},\ \bar{t}]$. Since this kind of interval $[\underline{t},\ \bar{t}]$ is chosen in an arbitrary way, we conclude that $I(t) \geq m$ for all large t.

According to our above discussion, the choice of m is independent of the positive solution of system (2.1), and we have proved that any solution of system (2.1) satisfies $I(t) \geq m$ for sufficiently large t, that is, $\liminf_{t\to+\infty} I(t) \geq m$. It is easy to obtain that, there exists a positive constant S_* such that $\liminf_{t\to+\infty} S(t) \geq S_*$. Therefore, system (2.1) is permanent. □

5. Numerical simulation and conclusion

In this paper, we show that the disease will go to extinction if $R_1 < 1$, and the disease persists if $R_2 > 1$. We provide numerical simulations of system (2.1) to support the conclusions of previous sections. Numerical analysis of system (2.2) is being done using Matlab.

In model (2.1), let $\omega = 12,\ r = 4,\ \alpha = 0.03,\ K = 1,\ \mu = 0.004, \gamma = 0.025,\ t_0 = 3,\ t_1 = 5,\ t_2 = 7,\ t_3 = 10,\ t_4 = 15$. We take $f_k(t,\ S,\ I) = \beta_k(t)SI = \psi_k(t)SI\ (k = 1,2,3,4)$ and $\beta_1(t) = 0.10 + 0.03\sin(\pi t/6),\ \beta_2(t) = 0.05 + 0.02\sin(\pi t/6),\ \beta_3(t) = 0.30 + 0.05\sin(\pi t/6),\ \beta_4(t) = 0.25 + 0.05\sin(\pi t/6)$.

In Fig. 1 we fix $\theta_1 = 0.1,\ \theta_2 = 0.5,\ \theta_3 = 0.5$ and $\theta_4 = 0.1$, resulting in $R_1 = 0.991 < 1$. This, of course, leads to the extinction of the disease, as clearly indicated by the graph.

However, in Fig.2 we use $\theta_1 = 0.1,\ \theta_2 = 0.5,\ \theta_3 = 0.5$ and $\theta_4 = 0.1$, giving $R_2 = 1.032 > 1$. Computer observation shows that the disease is permanent (see Fig.2).

Note that, in the hypotheses (A) and (B), if $\beta_k(t) = \psi_k(t)\ (k = 1,\ 2,\ \cdots,\ r)$, that is $f_k(t,\ S,\ I) = \beta_k SI$, then $R_1 = R_2$. In this case, R_1 or R_2 is the basic reproductive number which determines the extinction and the uniform persistence of diseases. Whereas, if $\beta_k(t) < f_k(t,\ S,\ I) < \psi_k(t)\ (k = 1,\ 2,\ \cdots,\ r)$, then $R_1 < R_2$. For system (2.1), we think there exists the threshold value R_0, and $R_1 \leq R_0 \leq R_2$. This means that conditions of Theorems 1 and 2 are sufficient, not necessary.

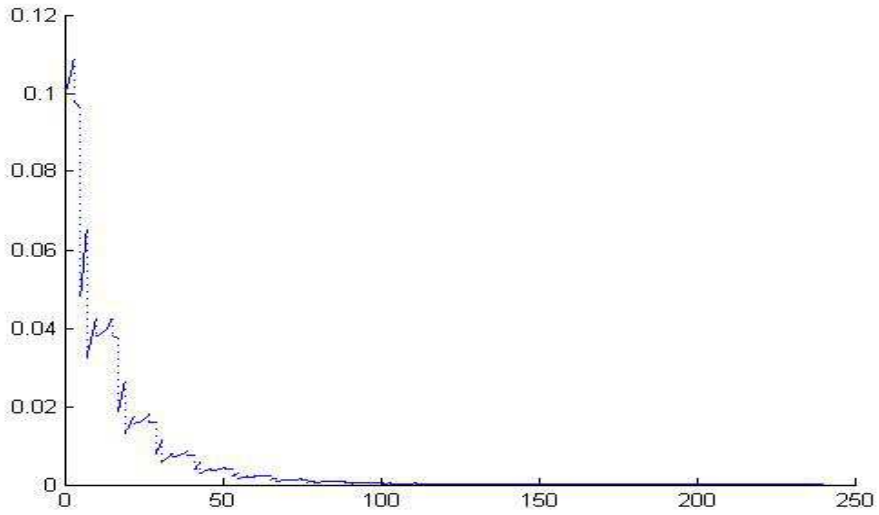

FIGURE 1. This figure shows that movement path of I as functions of time t. $R_0 = 0.991 < 1$, where parameters $\theta_1 = 0.1,\ \theta_2 = 0.5,\ \theta_3 = 0.5$ and $\theta_4 = 0.1$. The disease will be die out.

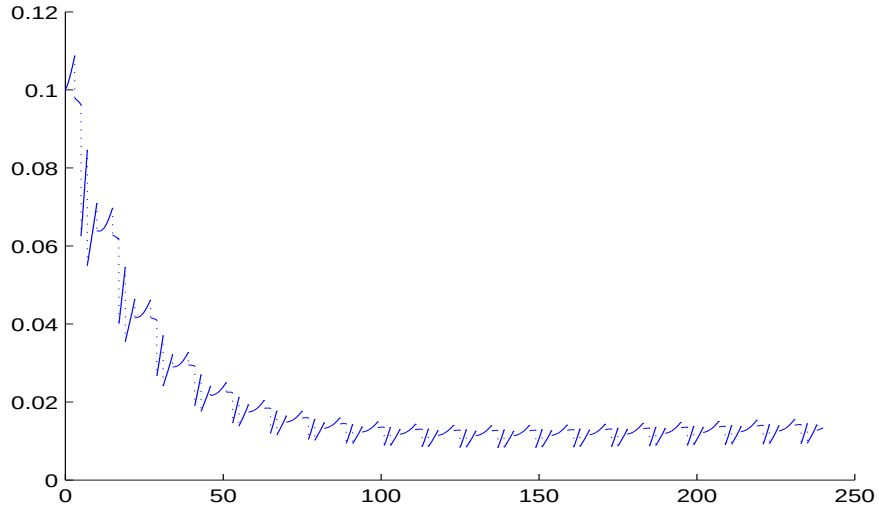

FIGURE 2. This figure shows that movement path of I as functions of time t. $R_0 = 1.032 > 1$, where parameters $\theta_1 = 0.1,\ \theta_2 = 0.45,\ \theta_3 = 0.45$ and $\theta_4 = 0.1$. The disease is permanent.

Conflict of Interests

The authors declare that there is no conflict of interests.

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