A DELAYED SIR EPIDEMIC MODEL WITH NONLINEAR INCIDENCE RATE AND PULSE VACCINATION

YANKE DU*, RUI XU

ABSTRACT. An SIR epidemic model with pulse vaccination and time delay describing infection period is investigated. The global attractiveness of the infection-free periodic solution is discussed, and sufficient condition is obtained for the permanence of the system. Our results indicate that a large vaccination rate or a short period of pulsing leads to the eradication of the disease.

AMS Mathematics Subject Classification : 34D05, 34C25, 92D30. Key words and phrases : SIR epidemic model, Delay, Pulse vaccination, Global attractiveness, Permanence.

1. Introduction

Infectious diseases have a great influence on the human life and socio-economy, which compel scientists to design and implement more effective control and preparedness programs. Pulse vaccination strategy (PVS), its theoretical study was firstly proposed by Agur et al. in [1], consists of periodical repetitions of impulsive vaccinations in a population, on all the age cohorts, differently from the traditional constant vaccination. At each vaccination time, a constant fraction of susceptible individuals is vaccinated. This kind of vaccination is called impulsive vaccination since all the vaccine doses are applied in a time which is very short with respect to the dynamics of the disease. Pulse vaccination is gaining prominence as a strategy for the elimination of infectious diseases such as measles, hepatitis, parotitis, smallpox and phthisis, and was considered in many literatures (see, for example, [3, 4, 5, 6, 9, 10, 11]). Known theoretical results showed that the pulse vaccination strategy can be distinguished from the conventional strategies in leading to disease eradiation at relatively low value of vaccination.

Received October 12, 2009. Revised December 30, 2009. Accepted January 16, 2010. * Corresponding author.

 $^{\ \, \}textcircled{o}$ 2010 Korean SIGCAM and KSCAM.

Incidence plays a very important role in the research of epidemic models. In many epidemic models, bilinear incidence βSI and standard incidence $\beta SI/N$ are frequently used. Bilinear incidence is based on the law of mass action. This contact law is more appropriate for communicable diseases such as influenza etc., but not for sexually transmitted diseases. For standard incidence, it may be a good approximation if the number of available partners is large enough and everybody could not make more contacts than is practically feasible. In [2], Capasso and Serio introduced a saturated incidence rate $\beta SI/(1+\alpha I)$ into epidemic models after studying the cholera epidemic spread in Bari in 1973, where βI measures the infection force of the disease and $1/(1+\alpha I)$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases. $\beta I/(1+\alpha I)$ tends to a saturation level when I gets large. Comparing with bilinear and standard incidence, saturation incidence may be more suitable for our real world.

The susceptible-infective-removed (SIR) model, initially proposed by Kermack and Mckendrik [8], is of great historical importance. The model has been extended in many ways. For example, we can consider the effects of population structure by age, transmission, time delay, and so on. In the natural world, there are many diseases which the infected population recover and become susceptible or removed population by itself after they are infected through some certain time. This phenomenon was studied by Hethcote and Driessche in [7]. Now, we assume that, when a susceptible individual is infected, there is a time $\tau > 0$, during which the infectious individual develops, and only after that time he or she becomes the removed one. The time τ is called infection period. In the present paper, we are concerned with the effects of time delay, nonlinear incidence rate, and pulse vaccination on the dynamics of an SIR epidemic model. To this end, we propose the following mathematical model:

$$\begin{cases}
\dot{S}(t) = A - \mu_1 S(t) - \frac{\beta S(t)I(t)}{1+\alpha I(t)} \\
\dot{I}(t) = \frac{\beta S(t)I(t)}{1+\alpha I(t)} - \frac{\beta e^{-\mu_2 \tau} S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)} - \mu_2 I(t) \\
\dot{R}(t) = \frac{\beta e^{-\mu_2 \tau} S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)} - \mu_3 R(t)
\end{cases} t \neq kT, \ k \in Z_+$$

$$S(t^+) = (1 - \theta)S(t)$$

$$I(t^+) = I(t)$$

$$R(t^+) = R(t) + \theta S(t)$$

$$t = kT, \ k \in Z_+$$
(1)

Here, all coefficients are positive constants. Z_+ denotes the set of positive integer. A represents the recruitment rate assuming all newborns to be susceptible. $\mu_1, \ \mu_2, \ \mu_3$ represent the death rates of susceptible, infectious, and recovered, respectively. θ (0 < θ < 1) is the proportion of those vaccinated successfully, which is called impulsive vaccination rate. T > 0 is the period of pulsing. Consider the death of infectious individuals during the infection period, that is, $\beta e^{-\mu_2 \tau} S(t-\tau) I(t-\tau)/(1+\alpha I(t-\tau))$ term. It is natural biologically to assume that $\mu_1 \leq \min\{\mu_2, \mu_3\}$.

Since the first two equations are independent of the third one, we restrict our attention to the following subsystem:

$$\begin{cases}
\dot{S}(t) = A - \mu_1 S(t) - \frac{\beta S(t)I(t)}{1+\alpha I(t)} \\
\dot{I}(t) = \frac{\beta S(t)I(t)}{1+\alpha I(t)} - \frac{\beta e^{-\mu_2 \tau} S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)} - \mu_2 I(t)
\end{cases} t \neq kT, k \in Z_+$$

$$S(t^+) = (1-\theta)S(t) \\
I(t^+) = I(t)
\end{cases} t = kT, k \in Z_+$$
(2)

The initial conditions for (2) are

$$(\phi_1(\theta), \phi_2(\theta)) \in C_+ = C([-\tau, 0], R_+^2), \ \phi_i(0) > 0, \ i = 1, 2.$$
 (3)

Let N(t) = S(t) + I(t) + R(t). It follows that

$$\dot{N}(t) = A - \mu_1 S(t) - \mu_2 I(t) - \mu_3 R(t) \le A - \mu_1 N(t),$$

which yields

$$\lim_{t \to \infty} \sup N(t) \le A/\mu_1. \tag{4}$$

It is easy to show that system (2) is positively invariant in the closed set

$$\Omega = \{ (S, I) | S \ge 0, I \ge 0, S + I \le A/\mu_1 \}.$$

The organization of this paper is as follows. In the next section, we establish sufficient condition for the global attractiveness of infection-free periodic solution of system (2). In Section 3, the permanence of system (2) is discussed. In Section 4, we illustrate our theory results by numerical simulations, and some discussions are given.

2. Global attractiveness of infection-free periodic solution

Firstly, we introduce two lemmas which are useful in the subsequent discussions.

Lemma 1. (see [6]) Consider the following impulsive system

$$\begin{cases} \dot{u}(t) = a - bu(t), \ t \neq kT, \\ u(t^+) = (1 - \theta)u(t), \ t = kT, \end{cases}$$

$$\tag{5}$$

where $a, b > 0, 0 < \theta < 1$. Then there exists a unique positive periodic solution of system (5)

$$\widetilde{u}_{a,b}(t) = \frac{a}{b} + \left(u_{a,b}^* - \frac{a}{b}\right)e^{-b(t-kT)}, \ kT < t \le (k+1)T$$

which is globally asymptotically stable, where

$$u_{a,b}^* = \frac{a(1-\theta)(1-e^{-bT})}{b(1-(1-\theta)e^{-bT})}.$$

We deduce from (4) and the first equation of system (2) that $\dot{S}(t) \geq A - (\mu_1 + \frac{A\beta}{\mu_1 + A\alpha})S(t)$. By Lemma 1 and the comparison theorem in impulsive differential equation, we have the following lemma.

Lemma 2. For any solution (S(t), I(t)) of (2) with (3), we have

$$\lim_{t \to \infty} \inf S(t) \ge u_{A,\mu_1 + \frac{A\beta}{\mu_1 + A\alpha}}^*.$$

Secondly, we show the existence of the infection-free periodic solution of system (2), in which infectious individuals are entirely absent from the population permanently, i.e., I(t) = 0 for all $t \ge 0$. Thus, the growth of susceptible individuals must satisfy

$$\begin{cases} \dot{S}(t) = A - \mu_1 S(t), & t \neq kT, \\ S(t^+) = (1 - \theta) S(t), & t = kT. \end{cases}$$

$$\tag{6}$$

By Lemma 1, we know the periodic solution of system (6)

$$\widetilde{S}_e(t) = \widetilde{u}_{A,\mu_1}(t) \tag{7}$$

is globally asymptotically stable.

In the following, we discuss the global attractiveness of the infection-free periodic solution $(\widetilde{S}_e(t), 0)$ of system (2).

Theorem 1. If $R^* < 1$, then the infection-free periodic solution $(\widetilde{S}_e(t), 0)$ of system (2) is globally attractive, where $R^* = A\beta(1 - e^{-\mu_1 T})/(\mu_1 \mu_2 (1 - (1 - \theta)e^{-\mu_1 T}))$.

Proof. Since $R^* < 1$, we can choose $\varepsilon > 0$ sufficiently small such that

$$\beta \left(\frac{A(1 - e^{-\mu_1 T})}{\mu_1 [1 - (1 - \theta)e^{-\mu_1 T}]} + \varepsilon \right) < \mu_2.$$
 (8)

From the first equation of system (2), we have $\dot{S}(t) \leq A - \mu_1 S(t)$. By (7) and the comparison theorem in impulsive differential equation, there exists an integer $k_1 > 0$ such that

$$S(t) < \widetilde{S}_e(t) + \varepsilon \le \frac{A(1 - e^{-\mu_1 T})}{\mu_1 [1 - (1 - \theta)e^{-\mu_1 T}]} + \varepsilon := \sigma, \ t > k_1 T.$$
 (9)

We deduce from (9) and the second equation of system (2) that

$$\dot{I}(t) \le \frac{\beta S(t)I(t)}{1 + \alpha I(t)} - \mu_2 I(t) \le (\beta \sigma - \mu_2)I(t), \ t > k_1 T.$$

Noting that (8) implies $\beta \sigma - \mu_2 < 0$, we get $\lim_{t\to\infty} \sup I(t) \leq 0$. Considering the positivity of I(t), we know that $\lim_{t\to\infty} I(t) = 0$. Therefore, there exists

an integer $k_2 > k_1$ such that $I(t) < \varepsilon$ for all $t > k_2T$. It follows from the first equation of system (2) that

$$\dot{S}(t) \ge A - (\mu_1 + \frac{\beta \varepsilon}{1 + \alpha \varepsilon}) S(t), \ t > k_2 T,$$

which implies that there exists $k_3 > k_2$ such that

$$S(t) > \widetilde{u}_{A,\mu_1 + \frac{\beta \varepsilon}{1 - \varepsilon}}(t) - \varepsilon, \ t > k_3 T.$$
 (10)

Because ε is sufficiently small, we derive from (9) and (10) that $\widetilde{S}_e(t)$ is globally attractive. Therefore, the infection-free solution $(\widetilde{S}_e(t), 0)$ is globally attractive. The proof is complete.

Set

$$\theta^* = \frac{(e^{\mu_1 T} - 1)(A\beta - \mu_1 \mu_2)}{\mu_1 \mu_2}, \ T_* = \frac{1}{\mu_1} \ln \left[1 + \frac{\theta \mu_1 \mu_2}{A\beta - \mu_1 \mu_2} \right].$$

According to Theorem 1, we can easily obtain the following results.

Corollary 1. If $A\beta < \mu_1\mu_2$, the infection-free periodic solution $(\widetilde{S}_e(t),0)$ is globally attractive. If $A\beta > \mu_1\mu_2$, the infection-free periodic solution $(\widetilde{S}_e(t),0)$ is globally attractive provided that $\theta > \theta^*$ or $T < T_*$.

3. Permanence

In this section, we discuss the permanence of system (2). We first give the following definition.

Definition 1. System (2) is said to be permanent if there exists a compact region $\Omega_0 \in \operatorname{int}\Omega$ such that every solution of system (2) with initial data (3) will eventually enter and remain in region Ω_0 .

Denote

$$R_* = (1 - \theta)(1 - e^{-\mu_2 \tau})R^*, \ I^* = \frac{A\beta(1 - \theta)(1 - e^{-\mu_1 T})(1 - e^{\mu_2 \tau})}{\mu_2(\beta + \alpha \mu_1)[1 - (1 - \theta)e^{-\mu_1 T}]} - \frac{\mu_1}{\beta + \alpha \mu_1}.$$

It is readily seen that $I^* > 0$ if $R_* > 1$.

Lemma 3. If $R_* > 1$, there does not exist any $t_0 > 0$ such that $I(t) < I^*$ for all $t \ge t_0$.

Proof. Suppose that the conclusion is not valid. Then there exists a $t_0 > 0$ such that $I(t) < I^*$ for all $t \ge t_0$. It follows from the first equation of (2) that

$$\dot{S}(t) > A - (\mu_1 + \frac{\beta I^*}{1 + \alpha I^*}) S(t), \ t \ge t_0.$$

Again by Lemma 1 and the comparison theorem in impulsive differential equation, there exists a $t_1 > t_0 + \tau$ such that, for $\varepsilon > 0$ sufficiently small,

$$S(t) > \widetilde{u}_{A,\mu_1 + \frac{\beta I^*}{1 + \alpha I^*}}(t) - \varepsilon > u^*_{A,\mu_1 + \frac{\beta I^*}{1 + \alpha I^*}} - \varepsilon := \delta, \ t \ge t_1.$$
 (11)

Noting that the second equation of (2) can be rewritten as

$$I(t) = \int_{t-\tau}^{t} \frac{\beta S(u)I(u)}{1 + \alpha I(u)} e^{-\mu_2(t-u)} du, \ t \ge \tau, \tag{12}$$

we derive from (11) and (12) that

$$I(t) > \beta \delta \int_{t-\tau}^{t} \frac{I(u)}{1 + \alpha I(u)} e^{-\mu_2(t-u)} du, \ t \ge t_1 + \tau.$$
 (13)

Denote $I_l = \min_{t \in [t_1, t_1 + \tau]} I(t)$. We claim that $I(t) \geq I_l$ for all $t \geq t_1$. Otherwise, there must exist a $\bar{t} > t_1 + \tau$ such that $I(\bar{t}) < I_l$. Denote $t_2 = \inf_{t > t_1 + \tau} \{I(t) < I_l\}$. Then we have $I(t_2) = I_l$ and $I(t) \geq I_l$ for $t_1 \leq t \leq t_2$. It follows that

$$I(t_2) > \frac{\beta \delta I_l(1 - e^{-\mu_2 \tau})}{\mu_2(1 + \alpha I_l)} > \frac{\beta \delta I_l(1 - e^{-\mu_2 \tau})}{\mu_2(1 + \alpha I^*)}.$$
 (14)

Noting that $R_* > 1$ implies that for $\varepsilon > 0$ sufficiently small,

$$\beta \delta(1 - e^{-\mu_2 \tau}) > \mu_2 (1 + \alpha I^*),$$
 (15)

we deduce from (14) and (15) that $I(t_2) > I_l$, which is a contradiction. This proves the claim.

Choose a constant \mathcal{R} such that $1 < \mathcal{R} < \frac{\beta \delta (1 - e^{-\mu_2 \tau})}{\mu_2 (1 + \alpha I^*)}$. We now claim that $I(t) > I_l \mathcal{R}$ for all $t \geq t_1 + \tau$. From (13), we have

$$I(t_1 + \tau) > \frac{\beta \delta I_l}{1 + \alpha I_l} \int_{t_1}^{t_1 + \tau} e^{-\mu_2(t_1 + \tau - u)} du$$

= $\frac{\beta \delta I_l (1 - e^{-\mu_2 \tau})}{\mu_2 (1 + \alpha I_l)} \ge \frac{\beta \delta I_l (1 - e^{-\mu_2 \tau})}{\mu_2 (1 + \alpha I^*)} > I_l \mathcal{R}.$

If the claim is not valid, then there is a $t_3 > t_1 + \tau$ such that $I(t_3) = I_l \mathcal{R}$ and $I(t) \geq I_l \mathcal{R}$ for $t_1 + \tau \leq t \leq t_3$, contradicting to

$$I(t_3) \geq \frac{\beta \delta I_l}{1 + \alpha I_l} \int_{t_3 - \tau}^{t_3} e^{-\mu_2(t_3 - u)} du$$

= $\frac{\beta \delta I_l (1 - e^{-\mu_2 \tau})}{\mu_2 (1 + \alpha I_l)} \geq \frac{\beta \delta I_l (1 - e^{-\mu_2 \tau})}{\mu_2 (1 + \alpha I^*)} > I_l \mathcal{R}.$

This proves the claim. By induction method, we conclude that $I(t) \geq I_l \mathcal{R}^k$ for $t \geq t_1 + k\tau$. It follows that $I(t) \geq I^*$ if t is sufficiently large, contradicting $I(t) < I^*$ for all $t \geq t_0$. The proof is complete.

Theorem 2. If $R_* > 1$, system (2) is permanent.

Proof. In view of Lemma 3, we are left to consider two cases for the positive solution of (S(t), I(t)) of system (2). First, there exists a \bar{t}_0 such that $I(t) \geq I^*$ for all $t \geq \bar{t}_0$, which is our expected case. Second, I(t) oscillates about I^* . Under this case, there is a $t_4 > 0$ such that $I(t_4) \geq I^*$. Let $t_5 = \inf_{t \geq t_4} \{I(t) < I^*\}$. I(t) is uniformly continuous since the positive solutions of (2) are ultimately bounded and I(t) is not affected by impulses. Hence, $I(t) \geq I^*$ for $t \in [t_4, t_5)$ and $I(t_5) = I^*$. Because I(t) is oscillatory about I^* , there exists $t_6 = \inf_{t \geq t_5} \{I(t) > I^*\}$. Then $I(t) \leq I^*$ for $t \in [t_5, t_6)$ and $I(t_6) = I^*$. In the same way, we can obtain a time sequence $t_4 \leq t_5 < t_6 < \dots < t_{2k} < t_{2k+1} < \dots$, such that

- (i) $I(t_i) = I^*$ for $i = 5, 6, \cdots$;
- (ii) $I(t) > I^*$ for $t \in (t_{2k}, t_{2k+1}), k = 2, 3, \cdots$;
- (iii) $I(t) < I^*$ for $t \in (t_{2k+1}, t_{2k+2}), k = 2, 3, \cdots$

We claim that there must be $\bar{T}=\sup\{t_{2k+2}-t_{2k+1},\ k\in Z_+,\ k\geq 2\}$. Otherwise, there exists a subsequence $\{T_j=t_{2k_j+2}-t_{2k_j+1},\ j\in Z_+\}$, such that $\lim_{j\to\infty}T_j=+\infty$. Using similar arguments as those in Lemma 3, $\lim_{j\to\infty}I(t_{2k_j+2})=\infty$. That is a contradiction with $I(t_{2k_j+2})=I^*$.

For any interval $[t_{2k+1}, t_{2k+2}]$, where k is sufficiently large such that $S(t) \geq u_{A,\mu_1+\frac{A\beta}{\mu_1+A\alpha}}^* - \varepsilon := v_1$ (referred to Lemma 2, $\varepsilon > 0$ sufficiently small) for $t \in [t_{2k+1}, t_{2k+2}]$. Let $\gamma = t_{2k+2} - t_{2k+1}$. Then $\gamma \leq \bar{T}$. Define $v_2 = \min_{1 \leq i \leq 2l-1} \{p_i\}$, where $l = \lceil \bar{T}/\tau \rceil$ ($\lceil x \rceil$ is the minimum integer being greater than or equal to x), and p_i ($1 \leq i \leq 2l-1$) are defined in (16), (17) and (18). We hope to show that $I(t) \geq v_2$ for all t sufficiently large.

Since I(t) is uniformly continuous, there is an $0 < \omega < \tau$ (independent of the choice of t_{2k+1}) such that $I(t) > I^*/2$ for $t_{2k+1} \le t \le t_{2k+1} + \omega$. If $\gamma \le \omega$, there is nothing to prove. Let us consider the case that $\omega < \gamma \le \tau$. For $t_{2k+1} + \omega < t \le t_{2k+1} + \gamma$, we have

$$I(t) \geq \beta v_1 \int_{t-\tau}^{t} \frac{I(u)}{1+\alpha I(u)} e^{-\mu_2(t-u)} du$$

$$\geq \beta v_1 \int_{t_{2k+1}}^{t_{2k+1}+\omega} \frac{I(u)}{1+\alpha I(u)} e^{-\mu_2(t-u)} du > \frac{\beta v_1 I^* e^{-\mu_2 \tau} \omega}{2+\alpha I^*} := p_0. \quad (16)$$

Define

$$p_1 = \min\{I^*/2, p_0\}. \tag{17}$$

We can get $I(t) \geq p_1$ for $t \in [t_{2k+1}, t_{2k+1} + \gamma]$. If $\gamma > \tau$, by the same reason, $I(t) \geq p_1$ for $t \in [t_{2k+1}, t_{2k+1} + \tau]$. For $t \in (t_{2k+1} + \tau, t_{2k+1} + 3\tau/2]$, we have

$$I(t) \ge \beta v_1 \int_{t_{2k+1}+\tau/2}^{t_{2k+1}+\tau} \frac{p_1}{1+\alpha p_1} e^{-\mu_2(t-u)} du \ge \frac{\beta p_1 v_1 e^{-\mu_2 \tau} \tau}{2(1+\alpha p_1)} := p_2.$$

For $t \in (t_{2k+1} + 3\tau/2, t_{2k+1} + 2\tau]$, we have

$$I(t) \ge \beta v_1 \int_{t_{2k+1}+\tau}^{t_{2k+1}+3\tau/2} \frac{p_2}{1+\alpha p_2} e^{-\mu_2(t-u)} du \ge \frac{\beta p_2 v_1 e^{-\mu_2 \tau} \tau}{2(1+\alpha p_2)} := p_3.$$

Define

$$p_i = \frac{\beta p_{i-1} v_1 e^{-\mu_2 \tau} \tau}{2(1 + \alpha p_{i-1})}, \ i = 2, 3, \dots, 2l - 1.$$
 (18)

Then, proceeding the above approach, we derive that

$$I(t) \ge p_{2m-2}, \quad t \in (t_{2k+1} + (m-1)\tau, t_{2k+1} + (m-1/2)\tau];$$

$$I(t) \ge p_{2m-1}, \quad t \in (t_{2k+1} + (m-1/2)\tau, t_{2k+1} + m\tau], \ m = 2, 3, \dots, l.$$

Therefore, $I(t) \geq v_2$ for $t \in [t_{2k+1}, t_{2k+2}]$. Since this kind of interval $[t_{2k+1}, t_{2k+2}]$ is chosen in an arbitrary way (we only need t_{2k+1} to be large), we conclude that $I(t) \geq v_2$ for all t sufficiently large. Accordingly, $\lim_{t \to \infty} \inf I(t) \geq v_2$.

Denote $\Omega_0 = \{(S, I) : S \geq v_1, I \geq v_2, S + I \leq A/\mu_1\}$. We know that the set Ω_0 is a global attractor in Ω , and of course, every solution of system (2) with initial conditions (3) will eventually enter and remain in region Ω_0 . Therefore, system (2) is permanent. The proof is complete.

Denote

$$\begin{split} \theta_* &= \frac{(e^{\mu_1 T} - 1)[A\beta(1 - e^{-\mu_2 \tau}) - \mu_1 \mu_2]}{A\beta(1 - e^{-\mu_2 \tau})(e^{\mu_1 T} - 1) + \mu_1 \mu_2}, \\ T^* &= \frac{1}{\mu_1} \ln \left[1 + \frac{\theta \mu_1 \mu_2}{A\beta(1 - \theta)(1 - e^{-\mu_2 \tau}) - \mu_1 \mu_2} \right]. \end{split}$$

According to Theorem 2, we can easily obtain the following results.

Corollary 2. Let $A\beta(1-e^{-\mu_2\tau}) > \mu_1\mu_2$. Then system (2) is permanent provided that $\theta < \theta_*$. Assume that $A\beta(1-\theta)(1-e^{-\mu_2\tau}) > \mu_1\mu_2$. Then system (2) is permanent provided that $T > T^*$.

4. Numerical simulations and discussions

In system (2), set $A=2, \mu_1=1, \beta=1, \alpha=4, \mu_2=2, \mu_3=1.5, \tau=1, T=3, \theta=0.25$. Then $R^*=0.9871<1$. According to Theorem 1, we know that the disease will disappear (see Fig. 1). If we set $A=3, \mu_1=1, \beta=2, \alpha=4, \mu_2=2, \mu_3=1.5, \tau=1, T=3, \theta=0.5$, then $R_*=1.2639>1$. According to Theorem 2, the disease will be permanent (see Fig. 2). If we set $A=2, \mu_1=1, \beta=1.5, \alpha=4, \mu_2=2, \mu_3=1.5, \tau=1, T=3, \theta=0.25$, then $R^*=1.4806>1$ and $R_*=0.9602<1$. Computer observation shows that the disease is still permanent (see Fig. 3).

In this paper, we have studied the dynamical behavior of an SIR epidemic model with pulse vaccination, nonlinear incidence rate, and time delay describing infection period. Two thresholds R^* and R_* have been established. Theorem 1 implies that the infectious population will vanish and the disease will die out provided that $R^* < 1$. The epidemiological implication of Theorem 2 is that the infectious population will persist and the disease will become endemic provided that $R_* > 1$. Corollaries 1 and 2 show that $\theta > \theta^*$ or $T < T_*$ leads the disease to fading out, whereas $\theta < \theta_*$ or $T > T^*$ leads the disease to uniform persistence. Our results indicate that we can prevent the epidemic disease becoming endemic

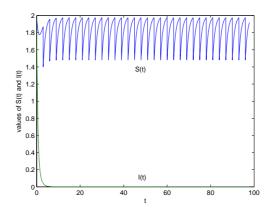


Fig. 1. This figure shows that the movement paths of S and I as functions of time t. $R^*=0.9871<1$. The disease dies out.

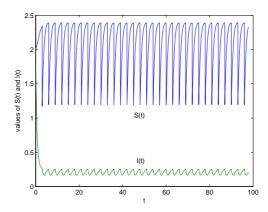


Fig. 2. This figure shows that the movement paths of S and I as functions of time t. $R_*=1.2639>1$. The disease is endemic.

by increasing the vaccination rate or shorting the period of pulsing. In addition, from Theorem 2, we see that if $(1-\theta)R^* > 1$, system (2) is permanent provided that $\tau > \frac{1}{\mu_2} \ln \left[1 + \frac{1}{(1-\theta)R^*-1} \right]$. That is, the disease will become endemic if the time delay τ is greater than a critical value.

We should mention here that we have only discussed two cases, i.e., $R^* < 1$ and $R_* > 1$, while for the condition that $R_* < 1 < R^*$, the dynamical behaviors of system (2) have not been studied theoretically. Computer observations suggest that the disease is uniformly persistent between R_* and R^* . Hence, we conjecture

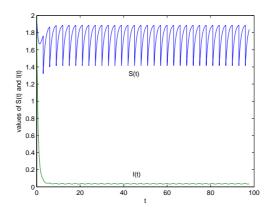


Fig. 3. This figure shows that the movement paths of S and I as functions of time t. $R^* = 1.4806 > 1$ and $R_* = 0.9602 < 1$. The disease is endemic.

that system (2) is permanent when $R^* > 1$, i.e., R^* is the threshold value whether the disease will go to extinction or not. We leave these for our future work.

Acknowledgements

This work was supported by the National Natural Science Foundation of P. R. China (10671209) and the Science Foundation of Shijiazhuang Mechanical Engineering College (JCB0803).

References

- Z. Agur, L. Cojocaru and G. Mazor, et al., Pulse mass measels vaccination across age cohorts, proc. Nat. Acad. Sci. USA 90 (1993) 698-702.
- V. Capasso and G. Serio, A generalization of Kermack-Mackendrick deterministic epidemic model, Math. Biosci. 42 (1978) 43-61.
- 3. A. D'Onofrio, Mixed pulse vaccination strategy in epidemic model with realistically distributed infectious and latent times, Appl. Math. Comput. 151 (2004) 181-187.
- A. D'Onofrio, Stability properties of pulse vaccination strategy in SEIR epidemic model, Math. Biosci. 179 (2002) 57-72.
- S. Gao, L. Chen and Z. Teng, Pulse vaccination of an SEIR epidemic model with time delay, Nonlinear Anal. RWA 9 (2008) 599-607.
- S. Gao, Z. Teng and D Xie, Analysis of a delayed SIR epidemic model with pulse vaccination, Chaos, Solitons, Fractals, 40 (2009), 1004-1011.
- H. Hethcote and P. Van Den Driessche, Two SIS epidemiological models for infectious disease. J. Math. Biol. 40 (2000) 2-26.
- 8. M. Kermark and A. Mckendrick, Contributions to the mathematical theory of epidemic model, Proc. Roy. Soc. A. 115 (1927) 700-721.
- Z. Lu, X. Chi and L. Chen, The effect of constant and pulse vaccination on SIR epidemic model with horizontal and vertical transmission, Math. Comput. Modell. 36 (2002) 1039-1057.

- L. Stone, B. Shulgin and Z. Agur, Theoretical examination of the pulse vaccination policy in the SIR epidemic model, Math. Comput. Modell. 31 (2000) 207-215.
- 11. G. Zeng and L. Chen, Complexity and asymptoical behavior of an SIRS epidemic model with proportional impulse vaccination, Adv. Complex Syst. 8 (2005) 419-431.

Yanke Du received her M.S. from Hebei Normal University. Since 2002 she has been at the Shijiazhuang Mechanical Engineering College, which named her a Lecturer in 2004 and now accepts her a Ph.D candidate. Her research interests focus on differential equations and mathematical biology.

Institute of Applied Mathematics, Shijiazhuang Mechanical Engineering College, Shijiazhuang, 050003, P. R. China

e-mail: dyk39@sohu.com

Rui Xu received his Ph.D at the University of Dundee under the direction of Mark Chaplain. Since 1987 he has been at Shijiazhuang Mechanical Engineering College which named him a full Professor in 2001. His research interests focus on differential equations and mathematical biology.

Institute of Applied Mathematics, Shijiazhuang Mechanical Engineering College, Shijiazhuang, 050003, P.R. China

e-mail: rxu88@yahoo.com.cn