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PULSE VACCINATION STRATEGIES IN A INFECTIOUS DISEASE MODEL WITH A NONMONOTONE INCIDENCE RATE AND TWO DELAYS

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ABSTRACT. This paper deals with a delayed SEIRS epidemic model with pulse vaccination and crowded incidence rate. Moreover, the case of vertical and horizontal transmission is considered. By using the discrete dynamical system determined by the stroboscopic map, the exact infection-free periodic solution of the SEIRS model is obtained. Further, by employing the comparison arguments, we prove that under the condition that $R_* < 1$ the infection-free periodic solution is globally attractive, and that under the condition that $R^* > 1$ the disease is uniformly persistent, which means that after some period of time the disease will become endemic.

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1. Introduction

In developing countries, infectious diseases have continued to be the major causes of suffering and mortality. Moreover, epidemic disease agents adapt and evolve, then new epidemic diseases have emerged and some existing diseases have reemerged (see, for example, Levins et al.[1]). Newly identified diseases include Lyme disease (1975), Legionnaires disease (1976), toxic-shock syndrome (1978), hepatitis C (1989), hepatitis E (1990), hantavirus (1993), severe acute respiratory syndrome(2004). The human immunodeficiency virus (HIV), which is the etiological agent for acquired immunodeficiency syndrome (AIDS), emerged in 1981 and has become an important sexually transmitted disease throughout the world.

Recently, many monographs have given us exciting insights of the emergence and detection of new diseases (see, for example, Preston[2]; Oldstone[3]; Garrett[4]). Mathematical models have become important tools in analyzing the

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spread and control of epidemic diseases (see, for example, Hethcote[5]) to avoid an economic loss.

Beginning in 1926 Kermack and McKendrick published papers about epidemic models and obtained the epidemic threshold result that the density of susceptibles must exceed a critical value in order for an epidemic outbreak to occur. Mathematical epidemiology has grown rapidly starting in the middle of the 20th century, so that a tremendous variety of models have now been formulated, mathematically analyzed, and applied to infectious diseases(see, for example, V. Capasso and G. Serio[6]; Anderson and May[7]; Busenberg et al.[8],[9]; Castillo-Chavez et al.[10]; Esteva and Vargas[11]; Feng et al.[12]; Hyman and Li[13] ,[21]; Chen et al.[14],[15],[20]; Shulgin[16]; Hethcoto[19]; Li et al.[23] and the references cited therein).

In classic epidemic dynamics, we usually divided total population into following groups:

- Susceptible (class S): Can contract the disease;
- Infected (class I): Has the disease and can infect others;
- Removed (class R) : Former infectives who are no longer infectious.

A SIRS model which has birth rate and death rate can be written as

$$\begin{cases} \dot{S}(t) = -IH(I,S) - dS + \mathbf{v}R + B, \\ \dot{I}(t) = IH(I,S) - (d+\gamma)I, \\ \dot{R}(t) = \gamma I - (d+\mathbf{v})R. \end{cases} \tag{1}$$

Here d is common natural death rate of three groups S, I *and* R; B denotes the recruitment rate of the population; γ is the rate of removal at which the infective individuals go into the removed class; $\mathbf{v}$ is the probability of which individuals in the removed class loss their immunity and go into the susceptible class; $IH(I,S)$ is the linear or nonlinear incidence rate concerning S and I.

The development of vaccines against infectious diseases has been a boon to human being. Pulse vaccination strategies in contrast to continuous vaccination are cost effective as they help in disease eradication at relatively low values of vaccination. Pulse vaccination strategy (PVS), whose theoretical study was started by Z.Agur and joint authors(see, for example, Agur[33]), consists of periodical repetitions of impulsive vaccinations in a population with multi age-cohort. Every vaccination time a constant fraction m of susceptible people (see, for example, Sabin[34]) is vaccinated and it was shown theoretically that pulse vaccination in which child aged one to seven years are immunized once every five years, may be enough to prevent the epidemics. PVS allows to reach the eradication of a disease with some practical advantages, as discussed in Shulgin[16]; Sabin et al.[34]; DeQuadors et al.[35]; Ransay et al.[36]; Gao et al.[37]. Examples of good successful applications of this policy include the global eradication of smallpox was announced by the WHA in May 1980 and the vaccination campaign against measles in UK in 1994(see, for example, Ramsay[36]). In 1995, Nokes and Swinton [38] have pointed out that pulse vaccination is gaining prominence as a strategy for the elimination of childhood viral infections such as measles

and polio.

On the other hand, it is well known that in an epidemiological model the waiting times in the compartments must be specified. Possible compartments are the susceptible compartment S, the latent compartment E (in which individuals are infected, but not yet infectious), the infectious compartment I, and recovered compartment R (in which individuals have permanent or temporary immunity). Here it is assumed that the latent period and, respectively, infectious period are constants for all individuals.

In recent years, many authors have studied epidemiological models with non-linear incidence rates. The most common non-linear incidence rate takes the form $\beta I^p S^q (\beta, p, q > 0)$, especially $\beta IS (p = 1, q = 1)$ is known as the bilinear incidence rate. Epidemiological models with this kind of incidence rate have been studied by Liu et al. [24, 25] and later by Hethcote et al. [17], Hethcote and Van den Driessche [18], Derrick and Van den Driessche [26, 27] and many others. After studying the cholera epidemic spread in Bari in 1973, Capasso and Serio [6] introduced a saturated incidence rate g(I)S into epidemic models, where g(I) tends to a saturation level when I gets large, i.e.,

$$g(I) = \frac{kIS}{1 + \alpha I},$$

in which kI measures the infect force of the disease and $\frac{1}{1+\alpha I}$ measures the inhibition effect from the crowding effect of the infective individuals. This incidence rate seems more reasonable than the bilinear incidence rate because it includes the crowding effect of the infective individuals and prevents the unboundedness of the contact rate by choosing suitable parameters. In 2003, Ruan and Wang [28] have studied the epidemic model with the incidence rate of the form

$$\frac{kI^2 S}{1 + \alpha I^2}.$$

Obviously, these incidence rates are special cases of the following incidence rate which is given by

$$\frac{kI^l S}{1 + \alpha I^h}$$

which is proposed by Liu et al.[24]. Also, Xiao and Ruan [31] proposed a non-monotone incidence rate

$$\frac{kIS}{1 + \alpha I^2}$$

which model the phenomenon that when the SARS emerges, people has little knowledge about the disease such that the infection probability are increasing. However, at the late stage of the SARS outbreak, psychological effects on the general public (see Leung et al. [30]), aggressive measures and policies, such as border screening, mask wearing, quarantine, isolation, etc. have been proved to be very effective (Gumel et al. [29]) in reducing the infective rate , even when the number of infective individuals were getting relatively larger. Similarly, for susceptible individuals their psychological effects also play an important role in

reducing the infective rate. For example, due to be afraid of catching a disease, many susceptible individuals would like to stay at home rather than in public places. Hence, the probabilities of effective contacts between susceptible individuals and infective individuals are reduced even when the number of susceptible individuals was getting relatively larger. To model this phenomenon, we propose a incidence rate of the complete form

$$f(S,I) = p(S)I = \frac{\beta SI}{1 + aS + bS^2} (\beta, a, b > 0).$$

So far as we know, people are seldom study ordinary differential equations concerning mathematical epidemiology models with this type incidence rate because qualitative analysis in these kind of differential systems is very complex. Also, it is noted that the newborns of the infectious may already be infected with the disease at birth such as hepatitis, phthisis, etc. This is called vertical transmission. On the other hand, some diseases may be spread from one individual to another via horizontal contacting transmission. Some epidemic models with vertical transmission were studied by many authors. However, few literatures deal with the analysis of disease with the crowded incidence rate, pulse vaccination, vertical and horizontal transmission.

In this work, motivated by the above-mentioned surveys, we consider the impact of pulse vaccination, latent period, infectious period, a crowded form for the incidence rate, vertical and horizontal transmission. The novel aspect of our paper is that we analyze the spread of epidemic diseases which are mainly influenced by the effects of psychological factor of susceptible individuals, and are connected with the latent period, infectious period, vertical and horizontal transmission. Also, using suitable mathematical tools, we focus on the control of epidemic diseases via pulse vaccination. The organization of this paper is as follows. We, in Section 2, formulate our model. To prove our main results, in Section 3, we give some preliminary results. In Section 4, main results and proofs of main results are given. Finally, in Section 5, we give out the conclusion of this paper and also point out some future research directions.

2. Model formulation

Since the natural birth rate and death rate are the same (denoted by μ below) and the disease is assumed not to inflict death on the infected host, so following the classical assumptions, we divide a population of constant size $N \equiv 1$ into a number of classes of epidemiological significance. For the SEIRS model, these classes are the susceptibles, the exposed populations(those who are already infected but are not yet infectious), the infectives and recovered (or removed) with populations denoted, respectively, by S, E, I and R, such that $S+E+I+R = 1$.

The delayed SEIRS epidemic model with pulse vaccination and crowded incidence rate is constructed as follows

$$\left\{ \begin{array}{l} \dot{S}(t) = \mu(1 - S(t)) - f(S(t), I(t)) + \gamma R(t) - (1-p)\mu I, \\ \dot{E}(t) = f(S(t), I(t)) - e^{-\omega\mu} f(S(t-\omega), I(t-\omega)) - \mu E, \end{array} \right\}$$

$$\left\{\begin{array}{l} \left.\begin{array}{l} \dot{I}(t) = e^{-\omega\mu} f(S(t-\omega), I(t-\omega)) - e^{-(\omega+\tau)\mu} f(S(t-\omega-\tau), \\ \qquad I(t-\omega-\tau)) - p\mu I, \\ \dot{R}(t) = e^{-(\omega+\tau)\mu} f(S(t-\omega-\tau), I(t-\omega-\tau)) - \gamma R(t) \\ \qquad -\mu R(t), \end{array}\right\} t \neq nT, \\ \left.\begin{array}{l} S(t^+) = (1-\theta)S(t), \\ E(t^+) = E(t), \\ I(t^+) = I(t), \\ R(t^+) = R(t) + \theta S(t), \end{array}\right\} \qquad t = nT, \end{array}\right. \tag{2}$$

in which

- S, E, I, R, $f(S,I)$ and μ are as in above. Also, $n \in Z_+ = \{0, 1, 2 \cdots\}$.
- θ is the proportion of those vaccinated successfully(with $0 < \theta < 1$).
- The delay ω is the latent period of the disease, and another delay τ is the length of the infection period. The term $e^{-(\omega+\tau)\mu} f(S(t-\omega-\tau), I(t-\omega-\tau))$ reflects the fact that an individual has recovered from infection and still are alive after infection period τ. The coefficient γ is the loss of immunity rate.
- T is the time between two consecutive pulse vaccinations. Without significant modification to the successive analysis, T could be also a rational or also irrational number.
- The term $p\mu(0 < p < 1)$ represents the number of newborns of infectious who transfer to the susceptible class, and $(1-p)\mu$ denotes the number of newborns of infectious who are infected vertically.

Obviously, we can get the following system which is equivalent to system (2)

$$\left\{\begin{array}{l} \left.\begin{array}{l} \dot{S}(t) = (\mu+\gamma)(1-S(t)) - f(S(t), I(t)) - \gamma(I(t)+E(t)) \\ \qquad -(1-p)\mu I, \\ \dot{E}(t) = f(S(t), I(t)) - e^{-\omega\mu} f(S(t-\omega), I(t-\omega)) - \mu E \\ \dot{I}(t) = e^{-\omega\mu} f(S(t-\omega), I(t-\omega)) - e^{-(\omega+\tau)\mu} f(S(t-\omega-\tau), \\ \qquad I(t-\omega-\tau)) - p\mu I, \end{array}\right\} t \neq nT, \\ \left.\begin{array}{l} S(t^+) = (1-\theta)S(t), \\ E(t^+) = E(t), \\ I(t^+) = I(t), \end{array}\right\} \qquad t = nT. \end{array}\right. \tag{3}$$

Considering ecological significance, we always assume that the initial value $\Phi(t) = (\phi_1, \phi_2, \phi_3)$ for system (3) satisfies

$$(\phi_1, \phi_2, \phi_3) \in C([-(\omega+\tau), 0], R^3_+), \ \phi_i(0) > 0 (i = 1, 2, 3),$$

where $R^3_+ = \{(S, E, I) | S \geq 0, E \geq 0, I \geq 0\}$. The meaningful domain of system (3) is

$$\Omega = \{(S, E, I) \in R^3_+ | 0 \leq S, E, I \leq 1, \ S + E + I \leq 1\},$$

and it is easy to prove that Ω is a positive invariant set.

In this paper, we shall study the dynamics of the "disease-free" state for system (3). Also, persistence (or permanence) is an important property of dynamical systems and of the systems in ecology, epidemics, etc. On the one hand

it is an important concept in itself addressing the long-term survival of some or all components of a system (see, for example, Hutson, Schmitt [42]; Waltman [43]), for reviews and references of the development until the early nineties, and Zhao [44] for an update. Hence, our another central focus is on the persistence of system (3) implies that the disease will be endemic.

3. Preliminaries

In this section, we shall introduce some definitions and state some preliminary lemmas which will be useful for establishing our main results.

Lemma 1. *Consider the following impulsive system*

$$\begin{cases} y'(t) = a - by(t), & t \neq nT, \\ y(t^+) = (1-\lambda)y(t), & t{=}nT, \end{cases} \tag{4}$$

in which a, $b > 0$, $0 < \lambda < 1$. *Then system (4) has a unique T-periodic solution given by*

$$y^*(t) = \frac{a}{b}\Big(1 - \frac{\lambda e^{-b(t-nT)}}{1-(1-\lambda)e^{-bT}}\Big),\ t \in \big(nT, (n+1)T\big],$$

which is globally asymptotically stable.

Proof. Integrating and solving the first equation of system (4) between pulses, we deduce

$$y(t) = \frac{a}{b} - (\frac{a}{b} - y(nT^+))e^{-b(t-nT)},\ t \in \big(nT, (n+1)T\big],$$

where $y(nT^+)$ be the initial value at time nT. Using the second equation of system (4), we deduce the stroboscopic map such that

$$y\big((n+1)T^+\big) = (1-\lambda)\Big(\frac{a}{b} - (\frac{a}{b} - y(nT^+))e^{-bT}\Big) \doteq f\big(y(nT^+)\big), \tag{5}$$

where $f(y) = (1-\lambda)\Big(\frac{a}{b} - (\frac{a}{b} - y)e^{-bT}\Big)$. It easy to see that (5) has a unique positive equilibrium $\widehat{y} = \frac{a}{b}\frac{(1-\lambda)(1-e^{-bT})}{1-(1-\lambda)e^{-bT}}$ that satisfies $y < f(y) < \widehat{y}$ if $0 < y < \widehat{y}$; $\widehat{y} < f(y) < y$ if $y > \widehat{y}$. Hence, we are easy to note that $\widehat{y}$ is globally asymptotically stable. It implies that the corresponding periodic solution of system (4)

$$y^*(t) = \frac{a}{b}\Big(1 - \frac{\lambda e^{-b(t-nT)}}{1-(1-\lambda)e^{-bT}}\Big),\ t \in \big(nT, (n+1)T\big],$$

which is globally asymptotically stable. This completes the proof. □

Lemma 2. [32] *If* $m : [t_0, \infty) \to [0, \infty)$ *is continuous such that*

$$\dot{m}(t) = -\rho m(t) + \varrho[\sup_{t-\tau_0 \le s \le t} m(s)] \text{ for } t \ge t_0 \text{ and } \tau_0 > 0,$$

and if $\rho > \varrho > 0$, *then there exist positive numbers* ι *and* κ *such that*

$$m(t) < \iota e^{-\kappa t} \text{ for } t > t_0.$$

Lemma 3. *Consider the following differential inequality*

$$\dot{\tilde{v}}(t) \geq (\leq)\hat{b} - \hat{a}\tilde{v}(t),$$

where $\hat{a}$, $\hat{b} > 0$, $\tilde{v}(t_0') > 0$. *One has*

$$\tilde{v}(t) \geq (\leq)\frac{\hat{b}}{\hat{a}}\{1 + (\frac{\hat{a}\tilde{v}(t_0)}{\hat{b}} - 1)\exp(-\hat{a}(t - t_0))\} \text{ for } t \geq t_0'.$$

Uniform persistence (or permanence) is an important property of dynamical systems and of the ecosystems. It is actually a concept which is important in itself, addressing the long-term survival of some or all components of a system.

Definition 1. System (3) is said to be uniformly persistent if there is a constant $m > 0$(independent of initial value) and a finite time T_0 such that for all solutions $(S(t), E(t), I(t))$ with all initial values $S(0^+) > 0$, $E(0^+) > 0$, $I(0^+) > 0$, $S(t) \geq M$, $E(t) \geq M$, $I(t) \geq M$ hold for all $t \geq T_0$. Here T_0 may depend on the initial values $S(0^+)$, $E(0^+)$ and $I(0^+)$.

Definition 2. System (3) is said to be permanent if system (3) is uniformly persistent and there is a constant $M > 0$(independent of initial value) and a finite time T_0 such that for all solutions $(S(t), E(t), I(t))$ with all initial values $S(0^+) > 0$, $E(0^+) > 0$, $I(0^+) > 0$, $S(t) \leq M$, $E(t) \leq M$, $I(t) \leq M$ hold for all $t \geq T_0$. Here T_0 may depend on the initial values $S(0^+)$, $E(0^+)$ and $I(0^+)$.

4. Main results and proofs

4.1 Main results Our main results are the following:

Theorem 1. *If* $R_* < 1$, *then infection-free periodic solution* $(S^*(t), 0, 0)$ *of system (3) is globally attractive, where*

$$R_* = \frac{\beta}{\mu e^{\omega\mu}}(1 - \frac{\theta e^{-(\mu+\gamma)T}}{1 - (1-\theta)e^{-(\mu+\gamma)T}}).$$

Theorem 2. *System (3) is permanent provided*

$$R^* = \frac{\beta e^{-\omega\mu}(1 - e^{-\tau\mu})}{p(1 + a + b)(\mu + \gamma)}\Big(1 - \frac{\theta e^{-(\mu+\gamma)T}}{1 - (1-\theta)e^{-(\mu+\gamma)T}}\Big) > 1$$

holds.

Corollary 1. *If* $\beta \leq \mu e^{\omega\mu}$, *then the periodic infection-free solution* $(S^*(t), 0, 0)$ *of system (3) is globally attractive.*

Corollary 2. *If* $\beta > \mu e^{\omega\mu}$, *then the periodic infection-free solution* $(S^*(t), 0, 0)$ *of system (3) is globally attractive provided that*

$$T < T_* \doteq \frac{1}{\mu + \gamma}\ln\frac{\beta - (1-\theta)\mu e^{\mu\omega}}{\beta - \mu e^{\mu\omega}}$$

holds or

$$\theta > \theta^* \doteq (\frac{\beta}{\mu e^{\omega\mu}} - 1)(e^{(\mu+\gamma)T} - 1)$$

holds.

Corollary 3. *If $\varpi\beta > \mu e^{\omega\mu}$, then the disease is permanent provided that*

$$T > T^* \doteq \frac{1}{\mu+\gamma} \ln \frac{\varpi\beta - (1-\theta)\mu e^{\mu\omega}}{\varpi\beta - \mu e^{\mu\omega}}$$

holds or

$$\theta < \theta_* \doteq (\frac{\varpi\beta}{\mu e^{\omega\mu}} - 1)(e^{(\mu+\gamma)T} - 1)$$

holds, in which $\varpi \doteq (1 - e^{-\tau\mu})\frac{\mu}{p(1+a+b)(\mu+\gamma)} < 1$.

4.2 Proof of Theorem 1

We first determine the existence of the infection-free solution of system (3), in which infectious individuals are entirely absent from the population permanently, i.e. $I(t) \equiv 0$ for $t \geq 0$. Under this condition, the growth of susceptible individuals and exposed individuals satisfy the following impulsive system without delays

$$\begin{cases} \left.\begin{aligned} \dot{S}(t) &= (\mu+\gamma)(1-S(t)) - \gamma E(t), \\ \dot{E}(t) &= -\mu E, \end{aligned}\right\} & t \neq nT, \\ \left.\begin{aligned} S(t^+) &= (1-\theta)S(t), \\ E(t^+) &= E(t), \end{aligned}\right\} & t = nT, \end{cases}$$

Obviously, it follows from the second and fourth equations of the above system that $\lim_{t\to\infty} E(t) = 0$. Hence we have the following limit system

$$\begin{cases} \dot{S}(t) = (\mu+\gamma)(1-S(t)), & t \neq nT, \\ S(t^+) = (1-\theta)S(t), & \text{t=nT.} \end{cases} \tag{6}$$

By Lemma 1, it is easy to know that system (6) has a unique T-periodic solution given by

$$S^*(t) = 1 - \frac{\theta e^{-(\mu+\gamma)(t-nT)}}{1-(1-\theta)e^{-(\mu+\gamma)T}}, \ t \in \big(nT, (n+1)T\big],$$

which is globally asymptotically stable.
Since $R_* < 1$, we can choose a $\epsilon > 0$ sufficiently small such that

$$e^{-\omega\mu}\beta\left(\frac{1-e^{-(\mu+\gamma)T}}{1-(1-\theta)e^{-(\mu+\gamma)T}} + \epsilon\right) < \mu. \tag{7}$$

From the first equation of system (3), we get $\dot{S} < (\mu+\gamma)(1-S)$. We consider the comparison equation

$$\begin{cases} \dot{x}(t) = (\mu+\gamma)(1-x(t)), & t \neq nT, \\ x(nT^+) = (1-\theta)x(nT), & t = nT. \end{cases}$$

It follows from Lemma 1 that the above system has a unique globally asymptotically stable $T-$periodic solution

$$x^*(t) = S^*(t) = 1 - \frac{\theta}{1-(1-\theta)e^{-(\mu+\gamma)T}} e^{-(\mu+\gamma)(t-nT)}$$
$$for\ t \in (nT, (n+1)T].$$

According to the impulsive comparison theorem (see, for example, Bainov et al. [40],[41]), for the given ϵ, there exists a $n_1 \in Z_+$ such that

$$\begin{gathered} S(t) < x^*(t) + \epsilon = S^*(t) + \epsilon \leq \frac{1-e^{-(\mu+\gamma)T}}{1-(1-\theta)e^{-(\mu+\gamma)T}} + \epsilon \doteq \overline{S} \\ for\ t \in (nT, (n+1)T],\ n > n_1. \end{gathered} \tag{8}$$

From the third equation of system (3), we get

$$\dot{I}(t) < e^{-\omega\mu}\beta\overline{S}I(t-\omega) - \mu I(t)\ for\ t > nT + \omega,\ n > n_1,$$

then by Lemma 2 and the comparison theorem, it follows that $\lim_{t\to\infty} I(t) = 0$, i.e. for any sufficiently small $\epsilon_1 > 0$, there exists an integer $n_2 > n_1$ such that $I(t) < \epsilon_1$ for all $t > n_2T$. Further, we are easy to get $\lim_{t\to\infty} E(t) = 0$, i.e. for any sufficiently small $\epsilon_2 > 0$, there exists an integer $n_3 > n_2$ such that $E(t) < \epsilon_2$ for all $t > n_3T$. From the first equation of system (3), we obtain $\dot{S} > (\mu + \gamma(1 - \epsilon_1 - \epsilon_2)) - (\mu + \gamma + \beta\epsilon_1)S$. Consider the following impulsive auxiliary system

$$\begin{cases} \dot{z}(t) = (\mu + \gamma(1 - \epsilon_1 - \epsilon_2)) - (\mu + \gamma + \beta\epsilon_1)z(t), & t \neq nT, \\ z(t^+) = (1-\theta)z(t), & \text{t=nT}, \end{cases} \tag{9}$$

in which $n > n_3$. Then by Lemma 1 system (9) has a unique T-periodic solution given by

$$z^*(t) = \frac{\mu + \gamma(1 - \epsilon_1 - \epsilon_2)}{\mu + \gamma + \beta\epsilon_1}\Big(1 - \frac{\theta e^{-(\mu+\gamma+\beta\epsilon_1)(t-nT)}}{1-(1-\theta)e^{-(\mu+\gamma+\beta\epsilon_1)T}}\Big),\ t \in \big(nT, (n+1)T\big],$$

which is globally asymptotically stable. It follows from the impulsive comparison theorem that

$$S(t) > z^*(t) - \epsilon\ for\ t > n_3T.$$

Hence

$$z^*(t) - \epsilon < S(t) < S^*(t) + \epsilon\ for\ t > n_3T.$$

Let $\epsilon_1, \epsilon_2 \to 0$, we get $z^*(t) \to S^*(t)$. Since ϵ is sufficiently small, the periodic infection-free solution $(S^*(t), 0, 0)$ of system (3) is globally attractive.

4.3 Proof of Theorem 2

Let $(S(t), E(t), I(t))$ be any solution with initial values of system (3). Hence, it is obvious that $S(t) \leq 1$, $E(t) \leq 1$ and $I(t) \leq 1$ for all $t > 0$, we shall determine that there exist positive constants m_S, m_E, m_I and $\widetilde{t_0}$ (t_0 is sufficiently large) such that $S(t) \geq m_S$, $E(t) \geq m_E$ and $I(t) \geq m_I$ for all $t > \widetilde{t_0}$.

Firstly, from the first equation of system (3), we derive

$$\dot{S} > p\mu - (\mu + \gamma + \beta)S,$$

then considering the following impulsive auxiliary system

$$\begin{cases} \dot{v}(t) = p\mu - (\mu + \gamma + \beta)v(t), & t \neq nT, \\ v(t^+) = (1-\theta)v(t), & \text{t=nT}. \end{cases} \tag{10}$$

By Lemma 1 system (10) has a unique T-periodic solution given by

$$v^*(t) = \frac{p\mu}{\mu+\gamma+\beta}\Big(1 - \frac{\theta e^{-(\mu+\gamma+\beta)(t-nT)}}{1-(1-\theta)e^{-(\mu+\gamma+\beta)T}}\Big),\ t \in \big(nT,(n+1)T\big],$$

which is globally asymptotically stable. We note from the impulsive comparison theorem and the global asymptotical stability of $v^*(t)$ that for any sufficiently small $\varepsilon > 0$, there exists a $t_0 > 0$ such that

$$S(t) > \frac{p\mu}{\mu+\gamma+\beta}\Big(1 - \frac{\theta e^{-(\mu+\gamma+\beta)T}}{1-(1-\theta)e^{-(\mu+\gamma+\beta)T}}\Big) - \varepsilon \doteq m_S \ \textit{for}\ t \geq t_0.$$

Secondly, for the above t_0, we are ready to show that there exists a $m_I > 0$ such that $I(t) > m_I$ for $t \geq t_0$. Since $R^* > 1$, we can choose sufficiently small m_I', $\varepsilon' > 0$ such that

$$\mu - (\gamma + (1-p)\mu)m_I' > 0 \ \textit{and}\ \frac{\beta e^{-\omega\mu}(1-e^{-\tau\mu})}{1+a+b}\overline{\overline{S}} - p\mu > 0, \tag{11}$$

where

$$\overline{\overline{S}} = \frac{p\mu - \gamma m_I'}{\mu+\gamma+\beta m_I'}\Big(1 - \frac{\theta e^{-(\mu+\gamma+\beta m_I')T}}{1-(1-\theta)e^{-(\mu+\gamma+\beta m_I')T}}\Big) - \varepsilon'.$$

It is claimed that for any $\widetilde{t}_0 > 0$, it is impossible that $I(t) < m_I'$ for all $t > \widetilde{t}_0$. Otherwise, there exists a $\widetilde{t}_0 > 0$ such that $I(t) < m_I'$ for all $t \geq \widetilde{t}_0$. According to the first equation of system (3), we derive

$$\dot{S} > (\mu - (\gamma + (1-p)\mu)m_I') - (\mu+\gamma+\beta m_I')S,$$

then considering the following impulsive auxiliary system

$$\begin{cases} \dot{\widehat{v}}(t) = (\mu - (\gamma+(1-p)\mu)m_I') - (\mu+\gamma+\beta m_I')\widehat{v}(t), & t \neq nT, \\ \widehat{v}(t^+) = (1-\theta)\widehat{v}(t), & \text{t=nT.} \end{cases} \tag{12}$$

It follows from Lemma 1 that system (12) has a unique T-periodic solution given by

$$\widehat{v}^*(t) = \frac{\mu - (\gamma+(1-p)\mu)m_I'}{\mu+\gamma+\beta m_I'}\Big(1 - \frac{\theta e^{-(\mu+\gamma+\beta m_I')(t-nT)}}{1-(1-\theta)e^{-(\mu+\gamma+\beta m_I')T}}\Big),\ t \in \big(nT,(n+1)T\big],$$

which is globally asymptotically stable. It is noted that from the impulsive comparison theorem and the global asymptotical stability of $\widehat{v}^*(t)$ that for any sufficiently small $\varepsilon' > 0$, there exists a $t_1 (> \widetilde{t}_0 + \tau + \omega)$ such that

$$S(t) > \frac{\mu - (\gamma+(1-p)\mu)m_I'}{\mu+\gamma+\beta m_I'}\Big(1 - \frac{\theta e^{-(\mu+\gamma+\beta m_I')T}}{1-(1-\theta)e^{-(\mu+\gamma+\beta m_I')T}}\Big) - \varepsilon' \doteq \overline{\overline{S}}\ \textit{for}\ t \geq t_1.$$

Next, we define a function $V(t)$ which is governed by

$$V(t) = I(t) + e^{-\omega\mu}(1-e^{-\tau\mu})\int_{t-\omega}^{t} f(S(\zeta),I(\zeta))d\zeta - e^{-(\omega+\tau)\mu}\int_{t-\omega-\tau}^{t-\omega} f(S(\zeta),I(\zeta))d\zeta.$$

After a little calculation, the derivative of $V(t)$ along the solution of system (3) is

$$\begin{aligned}\dot{V}(t) &= e^{-\omega\mu}(1-e^{-\tau\mu})f(S(t),I(t)) - p\mu I(t)\\ &> (\frac{\beta e^{-\omega\mu}(1-e^{-\tau\mu})\overline{\overline{S}}}{1+a+b} - p\mu)I(t) \ for\ t \geq t_1.\end{aligned}$$

Setting $I_L = \min_{t\in[t_1,t_1+\tau+\omega]} I(t)$, we will show that $I(t) \geq I_L$ for all $t \geq t_1$. Suppose the contrary. Then there is a $\widetilde{T}_0 \geq 0$ such that $I(t) \geq I_L$ for all $t_1 \leq t \leq t_1+\tau+\omega+\widetilde{T}_0$, and $I(t_1+\tau+\omega+\widetilde{T}_0) = I_L$. It follows from the third equation of system (2) and (11) that

$$\begin{aligned}I(t_1+\tau+\omega+\widetilde{T}_0) &\geq \int_{t_1+\widetilde{T}_0}^{t_1+\tau+\widetilde{T}_0} f(S(\xi),I(\xi))e^{-\mu(t-\xi)}d\xi\\ &> \frac{\beta e^{-\omega\tau}(1-e^{-\mu\tau})\overline{\overline{S}}}{(1+a+b)\mu}I_L > I_L,\end{aligned}$$

which leads to a contradiction. Thus, $I(t) > I_L$ for all $t \geq t_1$. As a result, we derive that

$$\dot{V}(t) > (\frac{\beta e^{-\omega\mu}(1-e^{-\tau\mu})\overline{\overline{S}}}{1+a+b} - \mu)I_L > 0 \ for\ t > t_2.$$

Hence $V(t) \to \infty$ as $t \to \infty$, which is contrary to $V(t) < 1+\omega\beta e^{-\omega\mu}(1-e^{-\tau\mu})$. Upon that, we accomplish that there exists a $t_1 > 0$ such that $I(t_1) \geq m_I'$.
By the claim, we are left to consider two cases. First, $I(t_1) \geq m_I'$ for all $t \geq t_1$ which indicates that our aim is obtained(here let $m_I = m_I'$). Second, $I(t)$ oscillates about m_I' for all large t. We hope to show that $I(t) \geq \frac{m_I'}{2}$ for all large t. Let $t^*(> t_1+\omega+\tau)$ and $\eta > 0$ satisfy

$$I(t^*) = I(t^*+\eta)$$

and

$$I(t) < m_I' \ for\ t \in (t^*, t^*+\eta).$$

Moreover, according to the ultimate boundedness of the positive solution in system (3) and $I(t)$ does not undergo the impulsive effect, it is easy to see that $I(t)$ is uniformly equicontinuous. So there exists a constant $T_1(0 < T_1 < \omega+\tau$, T_1 is independent of the choice of $t^*)$ such that $I(t) \geq \frac{m_I'}{2}$ for all $t \in [t^*, t^*+T_1]$.

In the following we shall discuss three possible cases in term of the size of T_1, η and $\omega+\tau$.

Case 1. When $\eta \leq T_1 < \omega+\tau$, it is obvious that $I(t) \geq \frac{m_I'}{2}$ for all $t \in [t^*, t^*+\eta]$.

Case 2. When $T_1 \leq \eta < \omega+\tau$, it is clear that

$$I(t) = \int_{t-\omega-\tau}^{t-\omega} f(S(\xi),I(\xi))e^{-\mu(t-\xi)}d\xi$$

$$\geq \int_{t^*}^{t^*+T_1} f(S(\xi), I(\xi)) e^{-\mu(t-\xi)} d\xi$$

$$> \frac{\beta \overline{\overline{S}}}{1+a+b} \frac{m_I'}{2} e^{-\mu(\omega+\tau)} T_1 \doteq m_I'' \tag{13}$$

for $t \in (t^* + T_1, t^* + \eta]$.

Case 3. When $T_1 < \omega + \tau < \eta$, we shall consider the following two subcase, respectively.

Subcase 3.1 If $t \in [t^*, t^* + \omega + \tau]$, it is easy to know that from (13), $I(t) \geq m_I''$ for all $t \in [t^*, t^* + \omega + \tau]$.

Subcase 3.2 If $t \in [t^* + \omega + \tau, t^* + \eta]$, we can affirm that $I(t) \geq m_I''$. Otherwise, suppose that there is a $\widehat{t} \in [t^* + \omega + \tau, t^* + \eta]$ such that $I(t) \geq m_I''$ for $t \in [\, t^* + \omega, \widehat{t}\,]$, and $I(\,\widehat{t}\,) = m_I''$. From the third equation of system (3) and (11), we obtain

$$I(\widehat{t}\,) = \int_{\widehat{t}-\omega-\tau}^{\widehat{t}-\omega} f(S(\xi), I(\xi)) e^{-\mu(\widehat{t}-\xi)} d\xi$$

$$> \frac{\beta e^{-\omega\mu}(1 - e^{-\tau\mu}) \overline{\overline{S}} m_I''}{(1+a+b)\mu} > m_I'',$$

which leads to a contradiction. Hence, the affirmation is obtained. Due to the randomicity of t^*, we can conclude that there exists $m_I \doteq \min\{\frac{m_I'}{2}, m_I''\} > 0$ such that $I(t) > m_I$ for $t \geq t_0$.

Next recalling the second equation of system (3), from $I(t) > m_I$ for $t \geq t_0 > 0$, we are easy to know that there exists a $\epsilon_0 > 0$ such that

$$\dot{E}(t) = f(S(t), I(t)) - e^{-\omega\mu} f(S(t-\omega), I(t-\omega)) - \mu E$$

$$> \epsilon_0 - \mu E, \tag{14}$$

then it follows from Lemma 3 that there exists a $\widetilde{t_0} > t_0$ $E(t) \geq \frac{\mu}{3\epsilon_0} \doteq m_E$ for $t > \widetilde{t_0}$.

Remark 1. *For the following SIRS model*

$$\left\{ \begin{array}{l} \left.\begin{array}{l} \dot{S}(t) = \mu(1 - S(t)) - f(S(t), I(t)) + \gamma R(t) - (1-p)\mu I, \\ \dot{I}(t) = f(S(t), I(t)) - e^{-\omega\mu} f(S(t-\omega), I(t-\omega)) \\ \qquad -p\mu I, \\ \dot{R}(t) = e^{-\omega\mu} f(S(t-\omega), I(t-\omega)) - \gamma R(t) - \mu R(t), \end{array}\right\} t \neq nT, \\ \left.\begin{array}{l} S(t^+) = (1-\theta) S(t), \\ I(t^+) = I(t), \\ R(t^+) = R(t) + \theta S(t), \end{array}\right\} \quad t = nT, \end{array} \right.$$

Using the similar analysis, we can get threshold parameters of the extinction of the disease and the uniform persistence of the disease. We omit arguments to avoid redundancy.

5. Discussion

In this paper we have proposed and analyzed a delayed epidemic model with pulse vaccination, vertical and horizontal transmission. The important results in the paper is that $R_* < 1$ implies that the periodic infection-free solution is globally attractive. From the mathematical viewpoint, it is noted that the global attractivity of the infection-free periodic solution is independent of the infection period τ. It is also noted that the disease will be permanent if $R^* > 1$, in which R^* is dependent on all coefficients in system (3). These seem to be reasonable from a biological point of view. But it should be noted that for the threshold parameter between the extinction of the disease and the uniform persistence of the disease, the dynamical behaviors of system (3) have not been studied. These issues would be left as our future consideration.

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