SIR-SVS epidemic models with continuous and impulsive vaccination strategies

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ABSTRACT

Vaccination is important for the control of some infectious diseases. This paper considers two SIR-SVS epidemic models with vaccination, where it is assumed that the vaccination for the newborns is continuous in the two models, and that the vaccination for the susceptible individuals is continuous and impulsive, respectively. The basic reproduction numbers of two models, determining whether the disease dies out or persists eventually, are all obtained. For the model with continuous vaccination for the susceptibles, the global stability is proved by using the Lyapunov function. Especially for the endemic equilibrium, to prove the negative definiteness of the derivative of the Lyapunov function for all the feasible values of parameters, it is expressed in three different forms for all the feasible values of parameters. For the model with pulse vaccination for the susceptibles, the global stability of the disease-free periodic solution is proved by the comparison theorem of impulsive differential equations. At last, the effect of vaccination strategies on the control of the disease transmission is discussed, and two types of vaccination strategies for the susceptible individuals are also compared.

1. Introduction

Infectious diseases have tremendous influence on human life. Millions of people died of various infectious diseases every year. To prevent and control the spread of infection, vaccination is one of the common effective strategies. In recent years, some epidemiological models have incorporated vaccination strategy into mathematical models to investigate the effect of vaccination on control of disease transmission. With respect to the description of vaccination strategy, there are often two types: continuous vaccination strategy (CVS) and pulse vaccination strategy (PVS). CVS implies that the vaccination is done instantaneously and that the rate coefficient of vaccination for individuals in a population is constant (Li and Ma, 2004; Li et al., 2006; Kribs-Zaleta and Velasco-Hernandez, 2000; Buonomo et al., 2008). PVS implies that vaccination is done with periodical repetition at some times, and that the fraction of the vaccinated individuals is a constant at each vaccination (Gao et al., 2006; Zhang and Teng, 2008; Zhang et al., 2010; Pei et al., 2008). Both strategies belong to herd immunity, the main difference between them is the continuity of variation of size for the vaccinated individuals. For CVS, this variation is always continuous; for PVS, the size of vaccinated individuals between two successive vaccinations is constant due to the absence of vaccination within these time intervals, and the size of vaccinated individuals at the vaccination times has a jump because vaccination occurs only at these times.

For mathematical models with vaccination, it is often assumed that the vaccine is completely effective in preventing the spread of infection, that is, the vaccinated individuals cannot be infected (Pei et al., 2008; Li and Ma, 2004; Capasso, 1993). In fact, there are often different types of vaccines for a disease to use, and any type of vaccine is not always completely effective, then a few of vaccinated individuals may still be susceptible to infection, but their susceptibility may be weaker than that of unvaccinated susceptible individuals, that is, they may have partial immunity because they have been vaccinated. This case was also considered in recent years in some references (Li et al., 2006; Kribs-Zaleta and Velasco-Hernandez, 2000; Yang et al., 2010; Shim, 2006; Liu et al., 2008a). For most of communicable diseases, the vaccinated individuals are susceptible for the disease before vaccinating the vaccine, and both types of vaccination strategies can be realized for the susceptibles. Currently, the newborns can also be vaccinated to prevent the spread of some diseases (such as, tuberculosis and hepatitis B). However, few mathematical models consider vaccination for the newborns. Since the birth of the newborns does not occur at the regular times, then the vaccination for the newborns should be described in CVS.

Motivated by the above consideration, we here formulate two SIR epidemic models with vaccination for both the newborns and susceptibles, where the types of vaccination for the susceptibles...
are CVS and PVS, respectively, and the types for the newborns are all CVS. It is assumed that the vaccinated individuals have the partial immunity and that the immunity of the vaccinated individuals is temporary.

Let \(S(t)\), \(I(t)\), and \(R(t)\) denote the numbers of the susceptible, infectious, and recovery individuals at time \(t\), respectively, \(\mu\) is the per capita natural death rate, \(\muA\) is the birth rate of newborns, \(\beta\) is the transmission coefficient of the disease, \(\gamma\) is the recovery rate coefficient of the infected individuals, \(a\) is the per capita disease-induced rate, the classical SIR model

\[
\begin{align*}
S' &= \muA - \muS - \beta SI , \\
I' &= \beta SI - (\mu + \gamma + a) I , \\
R' &= \gamma I - \mu R
\end{align*}
\] (1)

has been discussed in Ma and Li (2009) and Brauer and Castillo-Chavez (2001). Its basic reproduction number is \(R_0 = \frac{\betaA}{(\mu + \gamma + a)}\), and the disease free equilibrium is globally stable in the feasible region as \(R_0 \leq 1\), and the endemic equilibrium is globally stable as \(R_0 > 1\). Based on model (1), incorporating vaccination for the newborns and the susceptibles into model (1) yields the following two models:

\[
\begin{align*}
S' &= \mu(1-q)A - \muS - \beta SI + eV , \\
V' &= \muqA - \beta a VI - (\mu + e) V , \\
I' &= \beta S + \beta a VI - (\mu + \gamma + a) I , \\
R' &= \gamma I - \mu R
\end{align*}
\] (2)

and

\[
\begin{align*}
S' &= \mu(1-q)A - \muS - \beta SI + eV , \\
V' &= \muqA - \beta e VI - (\mu + e) V , \\
I' &= \beta S + \beta e VI - (\mu + \gamma + a) I , \\
R' &= \gamma I - \mu R
\end{align*}
\] (3)

In the above two models, \(V = V(t)\) denotes the numbers of the vaccinated individuals at time \(t\), the vaccination strategies for the newborns are all assumed to be continuous, \(q(0 < q < 1)\) denotes the fraction of the vaccinated newborns, and \(1 - q\) denotes that of the unvaccinated newborns. For the susceptible individuals in models (2) and (3), two different types of vaccination strategies are considered, respectively. In model (2), the vaccination strategy is continuous, the per-capita vaccination rate is \(p \geq 0\); in model (3), it is impulsive, the fraction of vaccination is \(p(0 \leq p \leq 1)\), the period of vaccination is \(T\), and the vaccination is done at time \(t = nT, n \in \mathbb{N}\). For the vaccinated individuals, let \(\epsilon\) denote the per capita rate coefficient at which the immunity wears off, which implies that the vaccinated individuals have the temporary immunity, and the factor \(\sigma(0 < \sigma < 1)\) reflects the effect of vaccine reducing the infection rate, the corresponding transmission coefficient for the vaccinated individuals is \(\beta\), where \(0 < \sigma < 1\) means that the vaccine is not completely effective, and that the vaccinated individuals have only partial immunity.

Model (2) consists of four ordinary differential equations. Analysis of global stability of equilibria for model (2) is mathematically an important issue, and has clear meanings epidemiologically. The Lyapunov function is a powerful tool to the analysis of stability for autonomous differential system. For the given Lyapunov function, how to prove the negative definiteness (or semidefiniteness) of its derivative is a key problem. In recent years, to prove the negative definiteness (or semidefiniteness) of the derivative, the relation between the arithmetic mean and the geometric mean is often used. To do this, it is often needed to arrange the derivative of the Lyapunov function in a suitable form. As far as we knew, this form is unique for the given Lyapunov function in the previous papers (Liu et al., 2008a, 2008b; Guo and Li, 2006). However, the case for model (2) is not so. That is, for the given Lyapunov function, the corresponding derivative needs to be arranged in the different forms for the different values of parameters to prove its negative definiteness (or semidefiniteness).

Model (3) is a system of impulsive differential equations. For it, we find the basic reproduction number determining whether the disease persists or dies out eventually by means of Floquet theory and comparison principle of impulsive differential equations, and compare the effects of two vaccination strategies for controlling the spread of the disease.

The organization of this paper is as follows: In the next section, we investigate the model with continuous vaccination for the susceptible individuals, its global stability is proved by constructing a Lyapunov function. In Section 3, we consider the model with impulsive vaccination for the susceptible individuals, the persistence and extinction of the disease are analyzed by using Floquet theory and comparison principle of impulsive differential equations. The basic reproduction numbers under two vaccination strategies are compared in Section 4. Finally, the effects of vaccination strategy on the control of disease spread are discussed.

2. The model with continuous vaccination for the susceptible individuals

Since the equation for \(R\) in model (2) are decoupled from other equations, then the dynamical behavior of model (2) is determined by the following system:

\[
\begin{align*}
S' &= \mu(1-q)A - (\mu + p)S - \beta SI + eV , \\
V' &= \muqA - \beta e VI - (\mu + e) V , \\
I' &= \beta S + \beta e VI - (\mu + \gamma + a) I , \\
R' &= \gamma I - \mu R
\end{align*}
\] (4)

For model (4), we have

\((S + V + I)' = [\mu(A - (S + V + I)] - (\gamma + a)I \leq \mu[A - (S + V + I)]\)

then it follows that \(\lim sup_{t \to \infty} (S + V + I) \leq A\). Therefore, the set \(\Omega = \{(S, V, I) \in \mathbb{R}^3_+ : S + V + I \leq A\}\) is positively invariant for system (4).

Direct calculation shows that (4) always has the disease free equilibrium \((S_0, V_0, 0)\), where

\[
S_0 = \left[\frac{\epsilon + \mu(1-q)A}{\mu + p + \epsilon}\right], \quad V_0 = \left(\frac{p + q\mu A}{\mu + p + \epsilon}\right).
\]

By the method of the next generation matrix proposed by van den Driessche and Watmough (2002), the basic reproduction number of (4) is given by

\[
R_{0c} = \frac{\beta(S_0 + \sigma V_0)}{\mu + \gamma + a} = \frac{\beta[\epsilon + \mu(1-q)] + \sigma(\mu + q\mu)}{(\mu + \gamma + a)(\mu + p + \epsilon)}.
\]

which represents the average number of new infections produced by one infected individuals during his infectious period when the population is at the disease free state.

The endemic equilibrium of (4), \(E^*(S^*, V^*, I^*)\), is determined by

\[
\begin{align*}
\mu + p + \beta 0S - eV &= \mu(1-q)A, \\
-pS + (\mu + e + \beta a) V &= \mu q A, \\
\beta(S + e V) &= \mu + \gamma + a.
\end{align*}
\] (5)
From the first two equations in (5), we can obtain
\[
\begin{align*}
S &= \frac{\mu A[e+(1-q)(\mu+\beta A \sigma)]}{(\mu+p+\beta \lambda)(\mu+\beta A \sigma)-p \epsilon} \\
V &= \frac{\mu A[q+p(\mu+\beta A \sigma)]}{(\mu+p+\beta \lambda)(\mu+\beta A \sigma)-p \epsilon}.
\end{align*}
\] (6)
Substituting them into the third equation in (5) yields
\[
H(I) = \frac{[e+(1-q)(\mu+\beta A \sigma)+\sigma p+q(\mu+\beta A \sigma)]}{(\mu+p+\beta \lambda)(\mu+\beta A \sigma)-p \epsilon} - \frac{\mu+\gamma+\zeta}{\beta \mu A} = 0.
\] (7)
Straightforward calculation shows
\[
H(I) = -\frac{[\beta^2(\mu^2+2 \beta \sigma A)]}{\beta A(\mu+\beta A \sigma)-p \epsilon} < 0,
\]
where
\[
c_0 = [e+(1-q)\mu]+\sigma(p+q \mu),
\]
\[
c_1 = (\mu+p)(p+q \mu)\sigma^2 + [2p(e+(1-q)\mu)+q \mu(\sigma+\mu+\epsilon)[e+(1-q)\mu].
\] then function \(H(I)\) is decreasing for \(I > 0\).

At \(I = A\),
\[
H(A) = \frac{\mu+\gamma+\zeta}{\beta \mu A}.
\]
where
\[
N_1 = [e+(1-q)(\mu+\beta A \sigma)+\sigma p+q(\mu+\beta A)],
\]
\[
N_2 = (\mu+p+\beta A)(\mu+\beta A \sigma)-p \epsilon.
\]
Since \(N_1 < \mu+\beta A \sigma+\beta A \sigma A \sigma \) and \(N_2 > \beta A(\mu+\epsilon+\sigma p+\beta A \sigma A \sigma \), then
\[
H(A) < \frac{1}{\beta A} - \frac{\mu+\gamma+\zeta}{\beta \mu A} < 0.
\]
Thus, \(H(I) < 0\) for \(I > A\).

On the other hand,
\[
H(0) = \frac{\mu+\gamma+\zeta}{\beta \mu A} \quad \text{(Reoc-1)}.
\]
Hence, when \(R_{ec} \leq 1\), (7) has no positive root; when \(R_{ec} > 1\), (7) has a unique positive root \(I^* \in (0,A)\). Furthermore, the components of the endemic equilibrium \(E^*, S^*\) and \(V^*\) are determined by (6). Therefore, with respect to the existence of equilibria of (4), we have

**Theorem 1.** System (4) always has the disease free equilibrium \(E_0(S_0,V_0)\); when \(R_{ec} > 1\), besides \(E_0\) system (4) also has a unique endemic equilibrium \(E^*(S^*,V^*,I^*)\), where
\[
S^* = \frac{[e+(1-q)\mu]}{\mu+p+\epsilon}, \quad V^* = \frac{[p+\mu]A}{\mu+p+\epsilon}, \quad I^* = \frac{[\mu A[e+(1-q)(\mu+\beta A \sigma)]]}{[\mu+p+\beta \lambda](\mu+\beta A \sigma)-p \epsilon}.
\]

With respect to the global stability of equilibria of (4), we have

**Theorem 2.** For system (4), the disease free equilibrium \(E_0\) is globally stable on the set \(\Omega\) when \(R_{ec} \leq 1\); the endemic equilibrium \(E^*\) is globally stable in the interior of the set \(\Omega\) when \(R_{ec} > 1\).

**Proof.** We first prove the global stability of the disease free equilibrium \(E_0\) when \(R_{ec} \leq 1\).

Since \(S_0\) and \(V_0\) satisfy the following equations:
\[
\begin{align*}
\frac{dS}{dt} &= (\mu+p)S-V = \mu(1-q)A, \\
\frac{dV}{dt} &= -pS+(\mu+\epsilon)V = \mu q A
\end{align*}
\]
then system (4) can be rewritten as
\[
\begin{align*}
S &= -(\mu+p)(S-S_0)-\epsilon(V-V_0)-\beta(S-S_0)I-\beta S V, \\
V &= p(S-S_0)-(\mu+\epsilon)(V-V_0)-\beta S V-I, \\
I &= [\beta(S_S_0)+\sigma V_0]-(\mu+\gamma+\zeta)I+\beta(S-S_0)I+\beta \sigma V (V-V_0)I.
\end{align*}
\] (8)
Define the Lyapunov function
\[
L_1 = \frac{(S-S_0)^2}{2S_0} + \frac{(V-V_0)^2}{2V_0} + I,
\]
then the derivative of \(L_1\) with respect to \(t\) along the solution of (8) is given by
\[
L_1' = \frac{(\mu+p)(S-S_0)^2}{S_0} + \frac{(S-S_0)(V-V_0)^2}{V_0} + \frac{(\mu+\epsilon)(V-V_0)^2}{V_0} - \frac{(S-S_0)(V-V_0)^2}{V_0} - \frac{(\mu+\epsilon)(V-V_0)^2}{V_0} + \frac{(\mu+p)+\beta \sigma I}{V_0} (S-S_0)(V-V_0) - \frac{(\mu+p)+\beta \sigma I}{V_0} (S-S_0)(V-V_0) - \frac{(\mu+p)+\beta \sigma I}{V_0} (S-S_0)(V-V_0)
\]
\[
\leq G(S,I) + [\beta(S+S_0)(V-V_0) -(\mu+\gamma+\zeta)I],
\]
where
\[
G(S,I) = \frac{\mu+p}{S_0} (S-S_0)^2 + \frac{(\mu+p)}{V_0} (S-S_0)(V-V_0) - \frac{(\mu+p)+\beta \sigma I}{V_0} (S-S_0)(V-V_0)^2.
\]
Using the expressions of \(S_0\) and \(V_0\) yields
\[
\left(\frac{\mu+p}{S_0} + \frac{p}{V_0}\right)^2 - \frac{4(\mu+p)(\mu+\epsilon)}{S_0 V_0}
\]
\[
= \left[\frac{(\mu+p)}{V_0}\right]^2 \left[(\mu+p)(\mu+\epsilon)(1-q)^2 - 4(\mu+\epsilon)(\mu+\mu q) \frac{\mu+p}{S_0 V_0} \right].
\]
Since \(\frac{p}{\mu+\mu q} < 1\) and \(\frac{\mu+p}{\mu+\mu q} > 1\) for \(0 < q < 1\),
then
\[
\left(\frac{\mu+p}{S_0} + \frac{p}{V_0}\right)^2 - \frac{4(\mu+p)(\mu+\epsilon)}{S_0 V_0} < \frac{1}{S_0 V_0} \left[(\mu+p)(\mu+\epsilon)(1-q)^2 - 4(\mu+\epsilon)(\mu+\mu q)\right]
\]
\[
< \frac{1}{S_0 V_0} \left[(\mu+p)(\mu+\epsilon)(1-q)^2 - 4(\mu+\epsilon)(\mu+\mu q)\right] < 0.
\]
Therefore, function \(G(S,I)\) is negative definite with respect to \(S=S_0\) and \(V=V_0\). Notice that \(\beta(S+S_0)-(\mu+\gamma+\zeta)I = (\mu+\gamma+\zeta)(R_{ec}-1)\), then \(I^* \leq 0\) for \(R_{ec} \leq 1\). It follows from LaSalle’s Invaraiable Principle (LaSalle, 1976) that \(E_0\) is globally stable on the set \(\Omega\) when \(R_{ec} \leq 1\).

Second, we prove the global stability of the endemic equilibrium \(E^*\).

Since \(S^*, V^*\), and \(I^*\) satisfy the following equations:
\[
\begin{align*}
\mu+p &= \frac{\mu(1-q)A}{S} - \beta I^* \quad S^*, \\
\mu+\epsilon &= \frac{\mu q A}{V} - \beta A \sigma I, \\
\beta(S+S_0) &= \mu+\gamma+\zeta
\end{align*}
\]
then system (4) can be rewritten as
\[
\begin{align*}
S &= S \left[\mu(1-q)A \left(\frac{1}{S} - \frac{1}{S^*}\right) - \beta (I-I^*) + \left(V - V^*\right)\right], \\
V &= V \left[\mu q A \left(\frac{1}{V} - \frac{1}{V^*}\right) + p \left(S - S^*\right) - \beta \sigma (I-I^*)\right], \\
I &= \beta \sigma (S-S^*) + \sigma (V-V^*).
\end{align*}
\] (9)
Let \( x = S/S^* \), \( y = V/V^* \), and \( z = I/I^* \), then (9) becomes
\[
\begin{align*}
\dot{x} &= x \left[ \frac{(1-q)A}{S} - \frac{my}{S}y \right] - \beta S^* (z-1) + \frac{C_3}{y} \left( V - \frac{S}{y} \right) - \beta \sigma t^* (z-1), \\
\dot{y} &= y \left[ \frac{muA}{V} - \frac{S}{y} - \frac{C_3}{y} \right] + \frac{pS}{y} - \beta \sigma t^* (z-1), \\
\dot{z} &= -\beta (S^*(x-1) + \sigma V^* (y-1)).
\end{align*}
\]

(10)

Define the Lyapunov function
\[
L_2 = (S-1-aV^*) + (V-(1-bV)) + (z-1-\lambda z)
\]
then the derivative of \( L_2 \) with respect to \( t \) along solution of (10) is given by
\[
\begin{align*}
\dot{L}_2 &= 2\mu A + \alpha V^* - \mu S - (\mu + \beta \sigma t^*) S^* - \frac{C_3}{y} \left( V - \frac{S}{y} \right) - \frac{C_3}{y} \left( V - \frac{S}{y} \right) = F(x, y),
\end{align*}
\]
where \( \mu_1 A + eV^* - pS^* = (\mu + \beta \sigma t^*) S^* \) and \( \mu_2 A + pS^* - eV^* = (\mu + \beta \sigma t^*) V^* \) are used.

To show that function \( F(x, y) \) is negative definite for all the feasible values of parameters, we divide the feasible region of parameters into three parts: \( pS^* < eV^* \), \( pS^* = eV^* \), and \( pS^* > eV^* \), and rearrange function \( F(x, y) \) by using Eqs. (5).

When \( pS^* < eV^* \), function \( F(x, y) \) can be expressed as
\[
F_1(x, y) = \mu (1-q) A \left( 2 - \frac{x}{y} - \frac{1}{y} \right) + \mu (\beta + \sigma) t^* V^* \left( 2 - \frac{y}{x} - \frac{1}{y} \right) + pS^* \left( 2 - \frac{y}{x} - \frac{1}{y} \right) + \left( eV^* - pS^* \right) \left( 3 - \frac{1}{x} - \frac{y}{x} \right).
\]

When \( pS^* = eV^* \), function \( F(x, y) \) can be expressed as
\[
F_2(x, y) = \mu (1-q) A \left( 2 - \frac{x}{y} - \frac{1}{y} \right) + \mu (\beta + \sigma) t^* V^* \left( 2 - \frac{y}{x} - \frac{1}{y} \right) + pS^* \left( 2 - \frac{y}{x} - \frac{1}{y} \right) + \left( eV^* - pS^* \right) \left( 3 - \frac{1}{x} - \frac{y}{x} \right).
\]

When \( pS^* > eV^* \), function \( F(x, y) \) can be expressed as
\[
F_3(x, y) = (\mu + \beta \sigma t^*) S^* \left( 2 - \frac{x}{y} - \frac{1}{y} \right) + \mu (\beta + \sigma) V^* \left( 2 - \frac{y}{x} - \frac{1}{y} \right) + eV^* \left( 2 - \frac{y}{x} - \frac{1}{y} \right) + \left( eV^* - pS^* \right) \left( 3 - \frac{1}{x} - \frac{y}{x} \right).
\]

By the property that the arithmetic mean is not less than the geometric mean, all functions \( F_i(x, y) (i = 1, 2, 3) \) are negative definite with respect to \( x = 1 - y = 1 \). It is easy to see that the maximum invariant set of (10) on the set \( \{(x, y, z) \in \mathbb{R}^3_+ : L_2 = 0 \} \) is the singleton \( \{(1, 1, 1)\} \), then it follows from LaSalle’s Invariant Principle (LaSalle, 1976) that \( E^* \) is globally stable in the interior of the set \( \Omega \). This completes the proof of Theorem 2. \( \square \)

3. The model with impulsive vaccination for the susceptible individuals

Since the equations for the variable \( R \) in model (3) are both independent of other equations, then the dynamical behavior of (3) is determined by the following system:
\[
\begin{align*}
S' &= S \left( (1-q)A - \mu S^* - \beta S I + \epsilon V \right), \\
V' &= V \left( \mu A - \beta S V + (\mu + \epsilon) V \right), \\
I' &= I \left( \beta S V - \mu I + \gamma S \right), \\
S(t^n) &= S(t^n) - pS(t^n), \\
V(t^n) &= V(t^n) + pS(t^n), \\
I(t^n) &= I(t^n).
\end{align*}
\]

(11)

For model (3), we have
\[
(S + V + I') = \mu (A - (S + V + I) - (\gamma + \epsilon) I) \leq \mu (A - (S + V + I))
\]
then it follows that \( \lim_{t \to \infty} (S + V + I) \leq A \). Therefore, the set \( \Omega = \{(S, V, I) \in \mathbb{R}^3_+ : S + V + I \leq A \} \) is positively invariant for system (11).

We first introduce the following lemma, which is useful for the inference later.

**Lemma 1.** Consider the impulsive differential equations:
\[
\begin{align*}
S' &= (1-q)A - \mu S + \epsilon V, \\
V' &= \mu A - (\mu + \epsilon) V, \\
I' &= \beta S V - (\mu + \gamma + \epsilon) I, \\
S(t^n) &= (1-p)S(t^n), \\
V(t^n) &= V(t^n) + pS(t^n).
\end{align*}
\]

(12)

Then (12) has a unique positive \( T \)-periodic solution \( \tilde{z}(t) = (S(t), V(t), I(t)) \) which is globally asymptotically stable, where
\[
\begin{align*}
S_p(t) &= \mathbf{A}(\mu+\epsilon) - \left( \frac{V_p A}{\mu+\epsilon} \right) e^{-(\mu+\epsilon)t}, \\
V_p(t) &= \mathbf{A}(\mu+\epsilon) - \left( \frac{V_p A}{\mu+\epsilon} \right) e^{-(\mu+\epsilon)t}, \\
S_{p_0} &= \frac{(1-p)(1-q)A}{(1-p)(1-q)A} + \frac{(1-p)(1-q)A}{(1-p)(1-q)A} e^{-(\mu+\epsilon)t}, \\
V_{p_0} &= \frac{A(\mu+\epsilon) + q(1-p)(1-q)}{A(\mu+\epsilon) + q(1-p)(1-q)} e^{-(\mu+\epsilon)t},
\end{align*}
\]

(13)

and
\[
\begin{align*}
S_p &= \frac{(1-p)(1-q)(1-q)A}{(1-p)(1-q)(1-q)A} - \left( \frac{V_p A}{\mu+\epsilon} \right) e^{-(\mu+\epsilon)t}, \\
V_p &= \frac{(1-p)(1-q)(1-q)A}{(1-p)(1-q)(1-q)A} e^{-(\mu+\epsilon)t},
\end{align*}
\]

(14)

Lemma 1 is proved in Appendix A. Corresponding to the positive \( T \)-periodic solution of (12), \( \tilde{z}(t) \), (11) has the disease free \( T \)-periodic solution \( \bar{z}(t) = (S(t), V(t), 0) \).

In the following, we will investigate the local stability and global attractivity of the disease free \( T \)-periodic solution \( \bar{z}(t) \) of (11), respectively.

The local stability of the disease free \( T \)-periodic solution \( \bar{z}(t) \) of system (11) may be determined by considering the behavior of small-amplitude perturbations of the solution. Defining \( (S(t), V(t), I(t)) = (S_p(t) + \delta S(t), V_p(t) + \delta V(t), I(t)), \) where \( x, y, z \) are small perturbations, then the linearizing system of (11) at the disease free periodic solution \( \bar{z}(t) \) is given by
\[
\begin{pmatrix}
\dot{x} \\
\dot{y} \\
\dot{z}
\end{pmatrix}
= 
\begin{pmatrix}
-\mu & e & -\beta S_p(t) \\
0 & -(\mu+\epsilon) & -\beta V_p(t) \\
0 & 0 & \beta [S_p(t) + \sigma V_p(t)] - (\mu + \gamma + \epsilon)
\end{pmatrix}
\begin{pmatrix}
x \\
y \\
z
\end{pmatrix}
\]

(14)

The fundamental matrix of (14) is
\[
\Phi(t) = e^{\int_0^t [\beta S_p(t) + \sigma V_p(t)] - (\mu + \gamma + \epsilon) dt},
\]
where
\[
\begin{align*}
\phi_1(t) &= e^{\int_0^t [\beta S_p(t) + \sigma V_p(t)] - (\mu + \gamma + \epsilon) dt}, \\
\phi_2(t) &= e^{\int_0^t [\beta S_p(t) + \sigma V_p(t)] - (\mu + \gamma + \epsilon) dt}.
\end{align*}
\]

(14)
The resetting conditions of the last three equations in (11) can be rewritten in the following form:

\[
\begin{pmatrix}
\chi(nT^{+}) \\
y(nT^{+}) \\
z(nT^{+})
\end{pmatrix} =
\begin{pmatrix}
1-p & 0 & 0 \\
p & 1 & 0 \\
0 & 0 & 1
\end{pmatrix}
\begin{pmatrix}
\chi(nT) \\
y(nT) \\
z(nT)
\end{pmatrix}.
\]

Then the local stability of the disease free \( T \)-periodic solution \( \xi(t) \) is determined by the eigenvalues of the matrix

\[
M = \begin{pmatrix}
1-p & 0 & 0 \\
p & 1 & 0 \\
0 & 0 & 1
\end{pmatrix} \phi(T)
= \begin{pmatrix}
(1-p)e^{-\mu T} & (1-p)e^{-\mu T} & (1-p)\phi_1(T) \\
pe^{-\mu T} & e^{-\mu T}[p+(1-p)e^{-\mu T}] & p\phi_1(T)+\phi_2(T) \\
0 & 0 & \phi(T)
\end{pmatrix}.
\]

The disease free \( T \)-periodic solution \( \xi(t) \) is locally asymptotically stable when all eigenvalues of matrix \( M \) have absolute values less than one, and unstable when one of all the eigenvalues of matrix \( M \) have absolute values greater than one (Roberts and Kao, 1998).

For matrix

\[
M = \begin{pmatrix}
(1-p)e^{-\mu T} & (1-p)e^{-\mu T} & (1-p)\phi_1(T) \\
pe^{-\mu T} & e^{-\mu T}[p+(1-p)e^{-\mu T}] & p\phi_1(T)+\phi_2(T) \\
0 & 0 & \phi(T)
\end{pmatrix},
\]

\[
\det(M) = (1-p)e^{-(\mu+\gamma+\delta)T} < 1,
\]
and

\[
\text{tr}(M) = 1 - \det(M) = (1-e^{-\mu T})[1-(1-p)e^{-(\mu+\gamma+\delta)T}] < 0
\]

is true, \( \text{tr}(M) < 1 \) and \( \det(M) < 2 \), then both eigenvalues of matrix \( M \) have absolute values less than one (Elaydi, 2005). Therefore, the local stability of \( \xi(t) \) is determined by the other eigenvalue of matrix \( M \), \( \phi(T) \). That is, \( \xi(t) \) is locally asymptotically stable for \( \phi(T) < 1 \), and unstable for \( \phi(T) > 1 \).

Notice that

\[
\phi(T) < 1 \iff \frac{\beta}{(\mu+\gamma+\delta)T} \int_0^T [\psi(p(t)+\sigma V_p(t))] dt < 1.
\]

Denote

\[
R_{op} = \frac{\beta}{\mu+\gamma+\delta} \int_0^T \psi(p(t)+\sigma V_p(t)) dt,
\]

that is,

\[
R_{op} = \frac{\beta A}{(\mu+\gamma+\delta)T},
\]

where

\[
A = [(1-q)-(\mu+\epsilon+\sigma\mu)] + \frac{(1-\sigma)([1-q]-\mu+\epsilon+\sigma\mu)e^{-(\mu+\gamma+\delta)T}-1}{(\mu+\epsilon)(1-(1-p)e^{-(\mu+\gamma+\delta)T})}.
\]

Since

\[
\frac{1}{T} \int_0^T S_p(t) dt = \lim_{n \to \infty} \frac{1}{T} \int_0^T S_p(t) dt
\]

and

\[
\frac{1}{T} \int_0^T V_p(t) dt = \lim_{n \to \infty} \frac{1}{T} \int_0^T V_p(t) dt
\]

are the average levels of susceptible and vaccinated individuals in the absence of infection, respectively, then \( R_{op} \) evaluates the average new infections produced by one infected individual during his infectious period when the population is composed of susceptible and vaccinated individuals. Thus the disease free \( T \)-periodic solution \( \xi(t) \) is locally asymptotically stable if \( R_{op} < 1 \), and unstable if \( R_{op} > 1 \).

In the following, we will prove the global attractivity of the disease free \( T \)-periodic solution \( \xi(t) \) when \( R_{op} < 1 \). To do this, we need the following lemma (Lakshmikantham et al., 1989):

**Lemma 2.** Suppose that

(i) the sequence \( \{t_k\} \) satisfies \( 0 < t_0 < t_1 < t_2 < \ldots \), with \( \lim_{k \to \infty} t_k = \infty \);

(ii) \( m \in PC([R_+, R^2], m(t)) \) is left continuous at \( t = t_k \), \( k = 1, 2, \ldots, g \in C[R_+ \times R^2, R^2], g(t, u) \) is quasimonotone nondecreasing in \( u \) for each \( t \), for \( k = 1, 2, \ldots, \psi_k(u) \in C[R^2, R^2] \) and \( \psi_k(u) \) is non-decreasing in \( u \) and

\[
\begin{align*}
D_m(t) & \leq g(t, m(t)), \\
(\text{if} \ t \neq t_k,
\end{align*}
\]

\[
(\text{if} \ t \neq t_k,
\end{align*}
\]

\[
\psi_k(u(t)) \leq \psi_k(u(t_k)),
\]

(iii) \( r(t) \) is the maximal solution of the impulsive differential system

\[
\begin{align*}
\dot{u} &= g(t, u), \\
(\text{if} \ t \neq t_k,
\end{align*}
\]

\[
(\text{if} \ t \neq t_k,
\end{align*}
\]

\[
\psi_k(u(t_k)) \leq \psi_k(u(t_k)),
\]

existing on \( [t_0, \infty) \). Then \( m(t_o) \leq u_0 \) implies that \( m(t) \leq r(t) \) from \( t \geq t_0 \).

From (11), we have

\[
\begin{align*}
S(t) & = (1-p)S(t_0) + (1-q)A - \mu S(t) + \epsilon = 0, \\
V(t) & = (1-q)V(t_0) + (1-q)A - (\mu+\epsilon) + \sigma\mu = 0, \\
\psi_k(V(t)) & = \psi_k(V(t_k)),
\end{align*}
\]

Considering (12) as the comparison impulsive differential equations, then it follows from Lemma 1 that, for the arbitrary positive number \( \delta \), there exists an integer \( n_1 > 0 \) such that

\[
S(t) < S(t_0) + \delta, \quad V(t) < V(t_0) + \delta
\]

for all \( nT < t \leq (n+1)T \) and \( n \geq n_1 \), where \( S(t), V(t) \) is the solution of system (12) with the initial value \( S(0^+) = S^0 \geq 0, V(0^+) = V^0 \geq 0 \).

Let \( S(t), V(t), l(t) \) be a solution of system (11) with the initial condition \( S(0^+) = S^0 \geq 0, V(0^+) = V^0 \geq 0, l(0^+) = l^0 > 0 \), then it follows by Lemma 2 that

\[
S(t) \leq S(t) < S(t_0) + \delta, \quad V(t) \leq V(t) < V(t_0) + \delta
\]

for all \( nT < t \leq (n+1)T \) and \( n \geq n_1 \).

Furthermore, from the third equation in (11) we have

\[
l(t) = l_0\int_0^T [\psi_k(\dot{S}(t) + \sigma V(\tau)) - (\mu+\gamma+\delta) dt]
\]

\[
= l_0\int_0^T [\psi_k(\dot{S}(t) + \sigma V(\tau)) - (\mu+\gamma+\delta) dt]
\]

\[
\leq l_0\int_0^T [\psi_k(\dot{S}(t) + \sigma V(\tau)) - (\mu+\gamma+\delta) dt]
\]

for all \( t \geq nT \) and \( n \geq n_1 \).

When \( R_{op} < 1 \), that is, \( \beta \int_0^T [S_p(t)+\sigma V_p(t)] dt < (1+\gamma+\delta)T \), then the inequality

\[
\int_0^T [\beta S_p(t)+\sigma V_p(t)] dt < (\mu+\gamma+\delta)T < 0
\]

holds for the positive number \( \delta \) small enough.

From (16) it follows that

\[
\lim_{t \to \infty} \int_0^T \psi_k(\dot{S}(t) + \sigma V(\tau)) - (\mu+\gamma+\delta) dt = 0,
\]
then we have \( \lim_{t \to \infty} l(t) = 0 \). Thus, when \( R_{0p} < 1 \), the impulsive differential system

\[
\begin{align*}
S' &= \mu(1-q)A - \mu S - \beta S I(t) + eV, \\
V' &= \mu A - \mu V + \beta S(t) V(t) - (\mu + e) V,
\end{align*}
\]

\( t \neq nT, \)

\[
S(nT^+) = (1-p)S(nT), \\
V(nT^+) = V(nT) + pS(nT)
\]

has the following limiting system:

\[
\begin{align*}
S' &= \mu(1-q)A - \mu S + eV, \\
V' &= \mu A - \mu V + \beta S(t) V(t) - (\mu + e) V,
\end{align*}
\]

\( t \neq nT, \)

\[
S(nT^+) = (1-p)S(nT), \\
V(nT^+) = V(nT) + pS(nT).
\]

From Lemma 1, \( \lim_{t \to -\infty} S(t) = S_0(t) \) and \( \lim_{t \to -\infty} V(t) = V_0(t) \). Therefore, the disease free T-periodic solution \( \zeta(t) \) is globally attractive when \( R_{0p} < 1 \). Furthermore, the local stability of \( \zeta(t) \) implies that \( \zeta(t) \) is globally stable on \( \Omega \) when \( R_{0p} < 1 \).

On the other hand, when \( R_{0p} > 1 \), similar to the proof in Gao et al. (2006), Zhang and Teng (2008), Zhang et al. (2010, 2003) and Liu and Chen (2003), we may also prove the persistence of the disease by the following idea. First, \( l(t) \) cannot always be below some small level, otherwise, \( t_0 > 0 \) such that \( l(t) \) will keep increasing for \( t > t_0 \) and lead to contradiction that \( \lim_{t \to \infty} l(t) = \infty \). Second, the persistence constant can be found by evaluating the solutions with which \( l(t) \) does not return to be below the level again. The detailed proof is omitted.

Summarizing the results above, we have

**Theorem 3.** For (11), the disease free T-periodic solution \( \zeta(t) \) is globally asymptotically stable if \( R_{0p} < 1 \) and the disease is uniformly persistent if \( R_{0p} > 1 \), i.e., there exists a positive constant \( \eta \) such that any solution \( (S(t), V(t), l(t)) \) of system (11) with initial values \( S(0) \geq 0, V(0^+) \geq 0, l(0^+) > 0 \) satisfies \( \lim_{t \to \infty} S(t) \geq \eta \), where \( \eta \) is independent of the initial values.

### 4. Discussion

We have investigated two epidemic models with the continuous and impulsive vaccination strategies, respectively. The associated basic reproduction numbers were given respectively. In the following, we first compare the effects of two vaccination strategies on the control of the disease, and then discuss the effect of control measures on the spread of infection for the continuous and pulse vaccination strategies.

First, we will compare the effect of vaccination on the control of infection under two vaccination strategies by comparing the basic reproduction numbers under the continuous and impulsive vaccination strategies, since the two basic reproduction numbers determine dynamical behaviors of the corresponding models.

If the rate coefficient of continuous vaccination for the susceptible individuals is \( p \), then, in the absence of infection, input, and death, the number of susceptible individuals is governed by equation \( S' = -pS \), whose solution with the initial value \( S(t_0) \) at \( t = t_0 \) is \( S(t) = S(t_0)e^{-pt} \). Thus, the probability that a susceptible individual is vaccinated after \( T \) period time is \( 1 - e^{-pT} \).

When the continuous vaccination in the period \( nT, (n+1)T \) for the given susceptible individuals is done concentrically at time \( t = nT \), the model with the continuous vaccination will lead to the impulsive differential equations with vaccination probability \( 1 - e^{-pT} \). Therefore, the associated basic reproduction number can be obtained by replacing \( p \) in \( R_{0p} \) with \( 1 - e^{-pT} \), that is,

\[
R_{0p} = \frac{\beta A[(1-q)\mu + \epsilon + \sigma \mu q - (1-\sigma)(1-q)\mu + \epsilon]H(T, \mu + \epsilon)}{(\mu + \gamma + \sigma)(\mu + \epsilon)}.
\]

The basic reproduction number for the case of pulse vaccination is greater than the associated one for the concentrative vaccination, i.e., pulse vaccination.

On the other hand, for the case that the fraction of pulse vaccination with \( T \)-period for the susceptible individuals is \( p \), if the size of the given susceptible individuals were vaccinated impulsively at time \( t = nT \) is rearranged to the time period \( {nT + (1-n)T} \) in the form of continuous vaccination, then the corresponding rate coefficient of continuous vaccination is \( -ln(1-p)/T \). Thus, for the associated continuous vaccination model, the basic reproduction number can be obtained by replacing \( p \) in \( R_{0c} \) with \(-ln(1-p)/T \), that is,

\[
R_{0c} = \frac{\beta A H_{\epsilon}}{(\mu + \gamma + \sigma)(\mu + \epsilon)}.
\]

Where

\[
H_{\epsilon} = \frac{\epsilon + (1-q)\mu + \epsilon + \sigma \mu q - (1-\sigma)(1-q)\mu + \epsilon}{-ln(1-p)/(\mu + \epsilon)}.
\]

Again, the basic reproduction number of the model with pulse vaccination can be expressed by

\[
R_{0p} = \frac{\beta A H_{p}}{(\mu + \gamma + \sigma)(\mu + \epsilon)},
\]

Where

\[
H_{p} = \frac{\epsilon + (1-q)\mu + \epsilon + \sigma \mu q - (1-\sigma)(1-q)\mu + \epsilon}{-ln(1-p)/(\mu + \epsilon)}.
\]

Direct calculation can yields

\[
H_{p} - H_{\epsilon} = \frac{-ln(1-p)(\mu + \epsilon)}{\epsilon + (1-q)\mu + \epsilon + \sigma \mu q - (1-\sigma)(1-q)\mu + \epsilon}.
\]

Let \( x \) and \( y \) in Lemma 4 in Appendix B be \( p \) and \( (\mu + \epsilon)T \), respectively, then it follows from Lemma 4 that

\[
\epsilon + (1-q)\mu + \epsilon + \sigma \mu q - (1-\sigma)(1-q)\mu + \epsilon > 0.
\]

It implies that \( H_{p} > H_{\epsilon} \), that is, \( R_{0p} > R_{0c} \). Then the basic reproduction number for the case of pulse vaccination is greater than the associated one for the continuous vaccination.

The above discussion shows that CVS is more effective than PVS for the control of the disease when the numbers of vaccinated susceptible individuals during the same period of time are equal for two vaccination strategies.

Next, we discuss the effect of control measures on the spread of infection for the continuous and pulse vaccination strategies.

We know that, in the absence of vaccination, the basic reproduction number is \( R_{0} = \beta A/(\mu + \gamma + \sigma) \), then, when \( R_{0} \leq 1 \), the disease dies out eventually, and the vaccinated may not be
needed; when $R_0 > 1$, the disease persists in the absence of vaccination, and vaccination is necessary to control the spread of the disease. Two vaccination strategies can be rewritten as

$$R_{0}(p, q) = \frac{\rho + \mu + \sigma p + (1-\sigma)q}{\mu + p + \sigma}$$

and

$$R_{0p}(p, q, T) = \frac{R_0 A_2}{\mu + \sigma},$$

respectively, where

$$A_2 = \frac{[\rho + \mu - (1-\sigma)q + (1-\sigma)p(\rho + \mu - \frac{\sigma q}{1-(p+\sigma)e^{-\mu T}} - 1]}{T(\mu + \sigma)(1-(1-p)e^{-\mu T})}.$$

It is easy to see that $R_{0c} < R_0$ and $R_{0p} < R_0$. It shows that vaccinations can lead to the basic reproduction number for models (2) and (3) decline.

Direct calculation shows that

$$\frac{\partial R_{0c}}{\partial p} < 0, \quad \frac{\partial R_{0c}}{\partial q} < 0, \quad \frac{\partial R_{0c}}{\partial \sigma} > 0$$

and

$$\frac{\partial R_{0p}}{\partial p} < 0, \quad \frac{\partial R_{0p}}{\partial q} < 0, \quad \frac{\partial R_{0p}}{\partial \sigma} > 0.$$

The above inequalities illustrate that increasing the fractions of vaccination and improving the efficacy of vaccine can result in the associated basic reproduction number declining, and then are helpful to prevent the spread of infection. This result is also obvious for the realistic cases.

To consider the effect of the period of pulse vaccination on the basic reproduction number, $R_{0p}$, we rewrite it as follows:

$$R_{0p} = \frac{\beta A}{(\mu + \gamma + \sigma)(\mu + e)} \left\{ [q(1+\mu + e) + \sigma q A_2] + \frac{(1-\sigma)p q(1+\mu + e)}{(\mu + e)} H(T) \right\},$$

where

$$H(T) = \frac{e^{-\mu T} - 1}{T[1-(1-p)e^{-\mu T}]}.$$

Straightforward calculation shows

$$H(T) = -\frac{e^{-\mu T} - 1}{T[1-(1-p)e^{-\mu T}]} G(\mu + \sigma T),$$

where

$$G(x) = px + (1-e^x)(1-(1-p)e^{-x}), \quad x > 0.$$

Since $G'(x) = p - e^x + (1-p) - e^{-x}$ and $G'(x) = -e^x - (1-p)e^{-x}$. Then, $G'(x) < 0$ and $G(0) = 0$ imply that $G(x) < 0$ for $x > 0$. Furthermore, $G'(x) < 0$ and $G(0) = 0$ implies that $G(x) < 0$ for $x > 0$. Thus, $H(T) > 0$, that is, $\partial R_{0p}/\partial T > 0$. Therefore, for the fixed fraction of vaccination for the susceptible individuals, decreasing the vaccination period can result in the basic reproduction number declining, and is useful to control the spread of infection.

On the other hand, since $\lim_{q \to 1, p \to 1} R_{0c} = \sigma R_0$ and $\lim_{q \to 0, p \to 0} R_{0p} = \sigma R_0$, then, from the above inference we have $R_{0c} < \sigma R_0$ and $R_{0p} > \sigma R_0$. This implies that, for the case that $R_0 > 1$, any vaccination strategies cannot make the infection extinct if $1/R_0 < \sigma < 1$. This result shows that increasing the efficiency is more important than choosing the vaccination strategies.

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### Appendix A. Proof of Lemma 1

We can easily obtain the analytical solution of system (12) between pulses

$$S(t) = \frac{[1-q]e^{\mu T}}{1+q} [S(nT^+)] + V(nT^+) - A_0 e^{-\mu T} t$$

$$- V(nT^+) - \frac{q A}{\mu + e} e^{-\mu T} t, \quad nT < t \leq (n+1)T.$$  

$$V(t) = \frac{q A}{\mu + e} + V(nT^+) - \frac{q A}{\mu + e} e^{-\mu T} t.$$  

Furthermore, after each successive pulse, we can deduce the following stroboscopic map:

$$S(n+1) = (1-p) \left\{ \frac{[1-q]e^{\mu T}}{1+q} [S(nT^+) + V(nT^+) - A_0 e^{-\mu T} t] \right\}$$

$$- (1-p) \left\{ V(nT^+) - \frac{q A}{\mu + e} e^{-\mu T} t \right\}$$

$$V(n+1) = \frac{q A}{\mu + e} + p S(nT^+) e^{-\mu T} + \frac{p q A}{\mu + e} (1-e^{-\mu T})$$

$$+ \left\{ V(nT^+) - \frac{q A}{\mu + e} \right\} [(1-p)e^{-\mu T} + pe^{-\mu T}].$$

(A.1)

Denote $x_n = S(nT^+)$, $y_n = V(nT^+)$, then (A.1) becomes the following difference equations:

$$x_{n+1} = (1-p) \left\{ \frac{[1-q]e^{\mu T}}{1+q} [x_n + y_n - A_0 e^{-\mu T} t] \right\}$$

$$- (1-p) \left\{ y_n - \frac{q A}{\mu + e} e^{-\mu T} t \right\},$$

$$y_{n+1} = \frac{q A}{\mu + e} + p x_n e^{-\mu T} + \frac{p q A}{\mu + e} (1-e^{-\mu T})$$

$$+ \left\{ y_n - \frac{q A}{\mu + e} \right\} [(1-p)e^{-\mu T} + pe^{-\mu T}].$$

(A.2)

Straightforward calculation shows that (A.2) has a unique positive fixed point $(x^*, y^*)$, where

$$x^* = \left(1-p\right)\frac{[1-q]e^{\mu T}}{1+q} [1-e^{-(1-p)e^{\mu T}}]$$

and

$$y^* = \frac{A [e^{\mu T} + q(1-p)e^{-(1-p)e^{\mu T}}]}{(1-p)e^{-(1-p)e^{\mu T}}}.$$

It implies that (12) has a unique positive $T$-periodic solution $\tilde{z}(t) = (\tilde{x}(t), \tilde{y}(t))$ with the initial value $S_0 = x^*$ and $V_0 = y^*$. Here, the expressions of $x^*$ and $y^*$ are the same as those of $S_0$ and $V_0$ defined in Lemma 1, respectively.

To prove the global stability of the $T$-periodic solution $\tilde{z}(t)$ of (12), it suffices to prove the global stability of the fixed point $(x^*, y^*)$ for (A.2).

Denote $u_n = x_n - x^*, w_n = y_n - y^*$, then (A.2) becomes

$$u_{n+1} = (1-p)e^{-\mu T} u_n + (1-p)e^{-(1-p)e^{\mu T}} w_n,$$

$$w_{n+1} = pe^{-\mu T} u_n + e^{-\mu T} [(1-p)e^{-(1-p)e^{\mu T}}] w_n,$$

(A.3)

which is a linear difference equations of $u_n$ and $w_n$. For its coefficient matrix

$$M = \left( \begin{array}{cc} (1-p)e^{-\mu T} & (1-p)e^{-(1-p)e^{\mu T}} \\ pe^{-\mu T} & e^{-\mu T} [(1-p)e^{-(1-p)e^{\mu T}}] \end{array} \right)$$

$$\det(M) = (1-p)e^{-(1-p)e^{\mu T}} < 1$$

and

$$\text{tr}(M) - 1 - \det(M) = -(1-e^{-\mu T}) [(1-(1-p)e^{-(1-p)e^{\mu T}}] < 0,$$
that is, $|\text{tr}(M)| < 1 + \text{det}(M) < 2$, then the origin of system (A.3) is locally asymptotically stable (Elaydi, 2005), and also globally stable. Therefore, the $T$-periodic solution $\bar{z}(t)$ of (12) is globally stable. This completes the proof of Lemma 1.

**Appendix B. Proof of two inequalities**

**Lemma 3.** The inequality

$$\frac{pT}{1-e^{-pT}} \cdot \frac{bT}{1-e^{-bT}} + \frac{(p+b)T}{1-e^{-(p+b)T}} > 0$$

holds for $p > 0$, $b > 0$, and $T > 0$.

**Proof.** Denote

$$f(x) = \frac{x}{1-e^{-x}} \quad \text{and} \quad g(x) = f(x) - f(pT + x)$$

then $\lim_{x \to 0} f(x) = 1 = f(0)$. Thus, we have

$$\frac{pT}{1-e^{-pT}} - \frac{bT}{1-e^{-bT}} + \frac{(p+b)T}{1-e^{-(p+b)T}} = g(0) - g(bT).$$

For function $f(x)$, we have

$$f'(x) = \frac{1 - (1 + x)e^{-x}}{(1-e^{-x})^2}, \quad f''(x) = \frac{-e^{-x}(1 + e^{-x})}{(1-e^{-x})^3} h(x),$$

where

$$h(x) = x - \frac{2(1-e^{-x})}{1-e^{-x}}.$$

Since

$$h''(x) = \left(\frac{1-e^{-x}}{1+e^{-x}}\right)^2 > 0$$

then it follows from $h(0) = 0$ that $h(x) > 0$, that is, $f''(x) > 0$. It implies that $f'(x) < f'(x + pT)$ for $x > 0$, that is, $g'(x) < 0$. Then $g(0) > g(bT)$. This completes the proof of Lemma 3. $\square$

**Lemma 4.** The inequality

$$\frac{x(1-e^x)}{\sigma^2 - 1 + x} - \frac{y \ln(1-x)}{y - \ln(1-x)} > 0$$

holds for $0 < x < 1$ and $y > 0$.

**Proof.** We first consider the following two functions:

$$g_1(x) = \frac{1}{x} + \frac{1}{\ln(1-x)} \quad \text{for} \quad 0 < x < 1,$$

and

$$g_2(y) = \frac{1}{y} + \frac{1}{1-e^y} \quad \text{for} \quad y > 0.$$

For function $g_1(x)$,

$$g'_1(x) = \frac{x}{\sqrt{1-x} \ln(1-x)} \left[ \frac{x}{\sqrt{1-x} \ln(1-x)} - \ln(1-x) \right],$$

where

$$h_1(x) = \frac{x}{\sqrt{1-x}} \ln(1-x).$$

Since

$$h'_1(x) = \frac{x(1-x)^{-3/2} \ln(1-x)}{2(1-x)^{3/2}} > 0$$

then $h'_1(0) = 0$ implies that $h_1(x) > 0$, that is, $g'_1(x) > 0$. Furthermore, $\lim_{x \to 0} g_1(x) = 1/2$ implies that $g_1(x) > 1/2$ for $0 < x < 1$.

For function $g_2(y)$,

$$g'_2(y) = \frac{h_2(y)}{y^2(e^y - 1)^2} \left( e^{2y} + e^y - 1 \right),$$

where

$$h_2(y) = ye^{y^2} - e^y + 1.$$  

By using the inequality that $e^x > 1 + u$ for $u > 0$,

$$h_2(y) = ye^{y^2} \left( 1 + \frac{b}{2} - e^{b/2} \right) < 0$$

then $h_2(0) = 0$ implies that $h_2(y) < 0$, that is, $g'_2(y) < 0$. Furthermore, $\lim_{y \to \infty} g_2(y) = 1/2$ implies that $g_2(y) < 1/2$ for $y > 0$.

It follows from the above discussion that $g_1(x) > 1/2 > g_2(y)$ for $0 < x < 1$ and $y > 0$, then

$$\frac{1}{x} + \frac{1}{\ln(1-x)} > \frac{1}{y} + \frac{1}{1-e^y}.$$

The last inequality is equivalent to the following one:

$$\frac{x(1-e^x)}{\sigma^2 - 1 + x} > \frac{y \ln(1-x)}{y - \ln(1-x)}$$

The proof of Lemma 4 is complete. $\square$

**References**


