Global Dynamics of a SEIR Model with a Varying Total Population Size and Vaccination

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Abstract

A SEIR model with vaccination, recruitment and natural death, as well as disease-caused death is studied. A threshold $R_0$ is identified which determines the global dynamics of the system and the outcome of the disease; if $R_0 \leq 1$, the infected individuals of the population disappears so the disease dies out, while if $R_0 > 1$, under a suitable restriction on the parameters, the infected individuals persist and a unique endemic equilibrium state is shown to be globally asymptotically stable in the interior of the feasible region.

Keywords: Epidemic models; Endemic equilibrium; Latent period; Global asymptotical stability; Compound matrices

1 Introduction

Studies of epidemic models that incorporate disease caused death and varying total population have become one of the important areas in the mathematical theory of epidemiology and they have largely been inspired by the works of Anderson and May [1,2]. Most of the research literature on these types of models assume that the disease incubation is negligible so that, once infected, each susceptible individual (in the class S) instantaneously becomes infectious (in the class I) and later recovers (in the class R) with a permanent or temporary acquired immunity. A compartmental model based on these assumptions is customarily called a SIR or SIRS model. Many diseases, however, incubate inside the hosts for a period of time before the hosts become infectious. Models

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that are more general than the SIR or SIRS types need to be studied to investi-
igate the role of incubation in disease transmission. Using a compartment
approach, one may assume that a susceptible individual first goes through a
latent period (and is said to become exposed or in the class E) after infection
before becoming infectious. The resulting models are of SEIR or SEIRS type,
respectively, depending on whether the acquired immunity is permanent or
otherwise.

Hethcote and Tudor [3] studied endemic infectious disease models for which
infection conferred permanent immunity with no disease-related mortality but
with vaccination. The infectious period had a general distribution. SEIR
models were considered by Hoppensteadt and Waltman [4,5,6,7]. These mod-
els needed an accumulated exposure to infection before infectiousness occurred.
Liu, Hethcote and Levin [8] considered a general SEIRS model with a nonlin-
ear incidence rate $\lambda I^pS^q$ and showed that these models exhibit a much wider
range of dynamical behavior than those with bilinear incidence rates $\lambda IS$. For
these models, there may exist multiple attractive basins in phase space; thus,
whether or not the disease will die out will depend not only on the param-
ters but also on the initial conditions. Gao and Hethcote [9] discussed disease
transmission models with density-dependent demographics. They considered
SIS and SIRS models with a standard incidence $\lambda SI/N$, where $N$ the total
number of individuals. Greenhalgh and Das [10] considered an SIR model
with vaccination at birth and a transmission term $\beta(N)SI/N$. For the case
$\beta(N) = \beta$, they proved global stability results related to the results of Busen-
berg and Van den Driessche [11]. Classical epidemic models assume a trans-
mission term of the form $\beta SI$. This implies that the contact rate for a single
individual is $\beta N$, linearly proportional to the number of individuals in the
population. An alternative assumption is to take a transmission term $\beta SI/N$
, which is nearer to models discussed by Anderson [12] for AIDS. This implies
that the contact rate for a single individual is $\beta$, a constant, which is more
suitable for sexually transmitted diseases. Greenhalgh [13] considered some
SEIRS epidemiological models with vaccination and temporary immunity. He
assumed that the average duration of immunity exceeded the infectious period
and proved that there was a threshold parameter $R_0$ which determined the
dynamics of system. Li, Graef, Wang, Karsai, [14] studied a SEIR model for
the transmission of an infectious disease that spreads in a population through
direct contact of the hosts. The force of infection is of proportionate mixing

We can see that the above mentioned SEIR disease models either did not
involve vaccination or assumed that the total population is constant. In the
present paper, we study a SEIR model with a varying total population size and
vaccination, which is more interesting and more realistic in a sense. We assume
that a constant recruitment $A$ belongs to the susceptible and the infectious
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individuals suffer a disease-caused mortality, the rate for disease-caused death is a constant \( \mu \). Thus the total population size may vary in time. An infectious disease spreads in a population through direct contact, a transmission term \( \beta SI \) is bilinear incidence rate. After the fraction \( pS \) of the susceptible is vaccinated, they pass into the recovered and this vaccination is a permanent immunity. Research on this epidemic model is scare in the literature. Our proof uses a theoretical approach developed in [16].

2 Model formulation

A population of size \( N(t) \) is partitioned into subclasses of individuals who are susceptible, exposed (infected but not yet infectious), infectious and recovered, with sizes denoted by \( S(t) \), \( E(t) \), \( I(t) \) and \( R(t) \), respectively. The sum \( E(t) + I(t) \) is the total infected population. It is assumed that all immigrant individuals are susceptible and vertical transmission can be neglected. The recovered individuals are assumed to acquire permanent immunity, and vaccination programs for the disease is only applied to the susceptible.

Then the SEIR model with vaccination is derived based on the above basic assumptions and described by the following system of differential equations:

\[
\begin{align*}
S'(t) &= A - \beta S(t)I(t) - (d + p)S(t) \\
E'(t) &= \beta S(t)I(t) - (d + \epsilon)E(t) \\
I'(t) &= \epsilon E(t) - (d + \alpha + \mu)I(t) \\
R'(t) &= \alpha I(t) + pS(t) - dR(t)
\end{align*}
\]

(2.1)

The parameter \( d > 0 \) is the rate for natural death, \( p > 0 \) is the rate for vaccination of susceptible, \( \mu > 0 \) is the rate for disease-caused death, \( \alpha > 0 \) is the rate for recovery and \( \epsilon > 0 \) is the rate at which the exposed individuals become infective so that \( 1/\epsilon \) is the mean latent period. There is no transfer from the \( R \) class back to the \( S \) class. The force of infection is \( \beta I(t) \), where \( \beta > 0 \) is the effective per capita contact rate of infective individuals and the incidence rate is \( \beta S(t)I(t) \). In the limit when \( \epsilon \to \infty \), or equivalently, when the mean latent period \( 1/\epsilon \to 0 \), the SEIR model becomes a SIR model.

The total population size \( N(t) \) can be determined by \( N(t) = S(t) + E(t) + I(t) + R(t) \), and is a solution of the differential equation

\[
\frac{dN(t)}{dt} = A - dN(t) - \mu I(t)
\]

(2.2)

Obviously, \( \limsup_{t \to \infty} N(t) \leq \frac{A}{d} \).

Note that the variable \( R(t) \) dose not appear in the first three equations of
This allows us to attack (2.1) by studying the following subsystem

\[
\begin{align*}
S'(t) &= A - \beta S(t)I(t) - (d + p)S(t) \\
E'(t) &= \beta S(t)I(t) - (d + \epsilon)E(t) \\
I'(t) &= \epsilon E(t) - (d + \alpha + \mu)I(t)
\end{align*}
\]

(2.3)

Therefore, in the rest of the paper, we will study (2.3) in the closed set

\[D = \{(S(t), E(t), I(t)) \in R^3_+ | S(t) + E(t) + I(t) \leq \frac{A}{d}\}\]

where \(R^3_+\) denotes the non-negative cone of \(R^3\) including its lower dimensional faces. It can be verified that \(D\) is positively invariant with respect to (2.3). \(\partial D\) denote the boundary of \(D\), \(\text{Int} D\) is the interior of \(D\).

3 Disease-free equilibrium

Let

\[R_0 = \frac{\epsilon \beta A}{(d + p)\omega \delta}\]

(3.1)

where \(\omega = d + \epsilon, \ \delta = d + \alpha + \mu\).

The point \(P_0 = (\frac{A}{d+p}, 0, 0)\) is the disease-free equilibrium of (2.3), and it exists for all positive values of the parameters.

**Theorem 3.1.** The disease-free equilibrium \(P_0 = (\frac{A}{d+p}, 0, 0)\) of (2.3) is globally stable in \(D\) if \(R_0 \leq 1\); and it is unstable if \(R_0 > 1\), and the solutions of (2.3) starting sufficiently close to \(P_0\) in \(D\) move away from \(P_0\) except that those solutions starting on the invariant \(S\)-axis approach \(P_0\) along this axis.

**Proof.** By the Routh-Hurwitz criterion, we can prove that the disease-free equilibrium \(P_0\) of (2.3) is locally stable in \(D\) if \(R_0 < 1\).

Consider the function \(L(t) = \epsilon E(t) + \omega I(t)\). Its derivative along the system (2.3) is \(L'(t) = \epsilon \beta S(t)I(t) - \omega \delta I(t) = (\epsilon \beta S(t) - \omega \delta)I(t) = \frac{\epsilon \beta}{R_0}(R_0 S(t) - \frac{A}{d+p})I(t)\).

From the first equation of (2.3), we can see that \(\limsup t \rightarrow \infty S(t) \leq \frac{A}{d+p}\).

If \(S(t) \leq \frac{A}{d+p}\), then \(L'(t) = \frac{\epsilon \beta}{R_0}(R_0 S(t) - \frac{A}{d+p})I(t) \leq 0, (R_0 \leq 1)\). Noting that the maximum invariant set in \(\{(S(t), E(t), I(t)) \in D | L'(t) = 0\}\) is the singleton \(\{P_0\}\), the global stability of \(P_0\) when \(R_0 \leq 1\) follows from LaSalle’s Invariance Principle([17],pp.296-297).

If \(S(t) > \frac{A}{d+p}\) for all \(t > 0\), then \(S'(t) \leq A - (d + p)S(t) < 0\), thus, \(S(t) \rightarrow \frac{A}{d+p}, (t \rightarrow \infty)\). We can obtain the limit systems from systems (2.3) as follows

\[
\begin{align*}
E'(t) &= \frac{\beta A}{d+p}I(t) - (\epsilon + d)E(t) \\
I'(t) &= \epsilon E(t) - (\alpha + d + \gamma)I(t)
\end{align*}
\]

(3.2)
The derivative of the function $L(t)$ along the solution of (3.2) is

$$L'(t) = \left[ \frac{\epsilon \beta A}{d + p} - (\epsilon + d)(\alpha + d + \gamma) \right] I(t) = (\epsilon + d)(\alpha + d + \gamma)(R_0 - 1)I(t) \leq 0, \ (R_0 \leq 1)$$

Thus, the maximum invariant set in $\{(S(t), E(t), I(t)) \in D | L'(t) = 0, S(t) = \frac{A}{d + p}, 0 \}$ is the singleton $\{( \frac{A}{d + p}, 0, 0) \}$. Therefore, when $S(t) > \frac{A}{d + p}$ and $R_0 \leq 1$, $(S(t), E(t), I(t)) \to (\frac{A}{d + p}, 0, 0)$, as $t \to \infty$.

From the above discussions, we can see that the disease-free equilibrium $P_0 = (\frac{A}{d + p}, 0, 0)$ of (2.3) is globally stable in $D$ if $R_0 \leq 1$.

If $R_0 > 1$, then $L'(t) > 0$ for $S(t)$ sufficiently close to $\frac{A}{d + p}$ except for $E(t) = I(t) = 0$. Solutions starting sufficiently close to $P_0$ leave a neighborhood of $P_0$ except those on the $S-$axis, thus the disease-free equilibrium $P_0$ is unstable. However, for solutions starting on the $S-$axis, the first equation of (2.3) reduces to $S'(t) = A - (d + p)S(t)$ and thus $S(t) \to \frac{A}{d + p}$, as $t \to \infty$.

## 4 Local stability of the endemic equilibrium

We say the disease is endemic if the infectious part of the population persists above a certain positive level for sufficiently large time. The endemcity of disease can be well captured and analyzed through the notion of uniform persistence([18]). The disease is endemic if (2.3) is uniformly persistent. In this case, both the infective and the latent individuals persist above a certain positive level.

**Lemma 4.1.** System (2.3) is uniformly persistent in $\text{IntD}$ if and only if $R_0 > 1$.

**Proof.** The necessity of $R_0 > 1$ follows from Theorem 3.1 and the fact that the asymptotic stability of $P_0$ precludes any kind of persistence. The sufficiency of the condition $R_0 > 1$ follows from a uniform persistence result, see Theorem 4.3 in [19]. Since the maximal invariant set on the boundary $\partial D$ is the singleton $\{P_0\}$ and is isolated for $R_0 > 1$, the set of stability of $\{P_0\}$ is contained in the $S-$axis and $\partial D$. Thus the hypothesis $(H)$ of Theorem 4.3 in [19] holds for (2.3). The condition for uniform persistence of Theorem 4.3 in [19] is equivalent to $P_0$ being unstable. Therefore, when $R_0 > 1$, the system (2.3) is uniformly persistence in $\text{IntD}$.

Thus, when $R_0 > 1$, Lemma 4.1 and the boundedness of $D$ implies the system (2.3) has a compact absorbing set $K$ in $\text{IntD}$.

The coordinates of an equilibrium $P^* = (S^*, E^*, I^*)$ in the interior of $D$ satisfy

$$\begin{align*}
\beta S(t)I(t) - \omega E(t) &= 0 \\
\epsilon E(t) - \delta I(t) &= 0 \\
A - \beta S(t)I(t) - (d + p)S(t) &= 0
\end{align*}$$

(4.1)
Theorem 4.1. If $R_0 > 1$, then the unique endemic equilibrium $P^*$ of (2.3) is locally asymptotically stable in IntD.

Proof. We will use the method of Routh-Hurwitz to show the local asymptotic stability of the equilibrium $P^*$. The Jacobian matrix of (2.3) at a point $P = (S(t), E(t), I(t))$ is

$$
J(P) = \begin{bmatrix}
-\beta I(t) - (d + p) & 0 & -\beta S(t) \\
\beta I(t) & -\omega & \beta S(t) \\
0 & \epsilon & -\delta 
\end{bmatrix}
$$

(4.2)

We prove that the matrix $J(P^*)$ is stable, namely, all its eigenvalues have negative real parts. Using the Jacobian matrix $J(P^*)$, we find that the characteristic equation is

$$
\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0
$$

where $a_1 = \omega + \delta + \frac{\epsilon \beta A}{\omega \delta} > 0$, $a_2 = (\omega + \delta) \frac{\epsilon \beta A}{\omega \delta} > 0$.

If $R_0 > 1$, then $a_3 = \omega \delta (d + p) (R_0 - 1) > 0$ and

$$
a_1 a_2 - a_3 = (\omega + \delta + \frac{\epsilon \beta A}{\omega \delta})(\omega + \delta) \frac{\epsilon \beta A}{\omega \delta} - \omega \delta (d + p) (R_0 - 1) = (d + p) R_0 (\omega + \delta)^2 + (d + p) (\omega + \delta) R_0 - \omega \delta + \omega \delta (d + p) = (d + p) R_0 [\omega^2 + \delta^2 + (d + p) (\omega + \delta) R_0] + \omega \delta (d + p) > 0
$$

Thus, the endemic equilibrium $P^*$ is locally asymptotically stable in IntD.

5 Global stability of the endemic equilibrium

The main aim of this section is to prove the endemic equilibrium $P^*$ is globally asymptotically stable in IntD for $R_0 > 1$ by using the approach in [16]. Once this result is proved, together with Theorem 3.1, we can claim that the basic reproduction number $R_0$ is a sharp threshold parameter and the global dynamical behaviors of the system (2.3) are completely determined. To show the global stability of the endemic equilibrium $P^*$, we need introduce some notions.

Let $x(t, 0, x_0)$ be the solution of the differential system

$$
\begin{aligned}
&x' = f(x) \\
x(0) = x_0
\end{aligned}
$$

(5.1)

Let $|\cdot|$ denote a vector norm in $\mathbb{R}^N$ as well as the matrix norm which is induces for $N \times N$ matrices. The Lozinski\u0103 measure $\mu(E)$ of a $N \times N$ matrix $E$ with respect to the norm $|\cdot|$ is defined as $\mu(E) = \lim_{h \to 0^+} \frac{|I + hE|^{-1}}{h}$, where $I$ is the unit matrix[20].
Let $q_2 = \limsup_{t \to \infty} \frac{1}{t} \int_0^t \mu(B(x(s, x_0)))ds$, where $B = A_f A^{-1} + A \frac{\partial f}{\partial x}[A]^{-1}$, $K$ is a compact absorbing set in IntD and $x \mapsto A(x)$ is a \(\left( \begin{array}{c} n \\ 2 \end{array} \right) \times \left( \begin{array}{c} n \\ 2 \end{array} \right)\) matrix-valued function. $A_f$ is the matrix obtained by replacing each entry $a_{ij}$ in $A$ by its directional derivative in the direction of $\frac{\partial f}{\partial x}$ with respect to (5.1), and $\frac{\partial f}{\partial x}[A]$ is the second additive compound matrix [21] of Jacobian matrix $\frac{\partial f}{\partial x}$.

**Lemma 5.1.** For the system (5.1), assume that

- (H1) IntD is simple connected;
- (H2) there is a compact absorbing set $K \subset \text{IntD}$;
- (H3) $\bar{x}$ is the only equilibrium of (2.3) in IntD. If $q_2 < 0$, then the global stability of $\bar{x}$ with respect to IntD is implied by its local stability.

Now, we will prove the global stability of the endemic equilibrium $P^*$.

**Theorem 5.1.** Suppose that $R_0 > 1$. Then the unique positive equilibrium $P^*$ of (2.3) is globally asymptotically stable in IntD for $p \leq \epsilon$.

**Proof.** The proof of the theorem consists of choosing a suitable vector norm $| \cdot |$ in $\mathbb{R}^3$ and a $3 \times 3$ matrix-valued function $A(x)$ so that the quantity $q_2$ is negative. We set $A$ as the following diagonal matrix:

\[
A(S(t), E(t), I(t)) = \text{diag}(1, \frac{E(t)}{I(t)}, \frac{E(t)}{I(t)})
\]

Then $A$ is $C^1$ and nonsingular in IntD. Let $f$ denote the vector field of (2.3).

Then

\[
A_f A^{-1} = \text{diag}(0, \frac{E'(t)}{E(t)} - \frac{I'(t)}{I(t)}, \frac{E'(t)}{E(t)} - \frac{I'(t)}{I(t)})
\]

\[
AJ^{[2]}A^{-1} = \begin{bmatrix}
-\beta I(t) - 2d - p - \epsilon & \beta \frac{S(t)I(t)}{E(t)} & \beta \frac{S(t)I(t)}{E(t)} \\
\epsilon \frac{E(t)}{I(t)} & -\beta I(t) - 2d - p - \mu - \alpha & 0 \\
0 & \beta I(t) & -2d - \epsilon - \mu - \alpha
\end{bmatrix}
\]

where $J^{[2]}$ is the second additive compound matrix of the Jacobian matrix $J$ of (2.3). Therefore, the matrix $A_f A^{-1} + AJ^{[2]}A^{-1}$ can be written in the following block form:

\[
B = \begin{bmatrix}
B_{11} & B_{12} \\
B_{21} & B_{22}
\end{bmatrix}
\]

With

\[
B_{11} = -\beta I(t) - 2d - p - \epsilon, \quad B_{12} = (\beta \frac{S(t)I(t)}{E(t)}, \frac{S(t)I(t)}{E(t)}), \quad B_{21} = (\epsilon \frac{E(t)}{I(t)}, 0)^T
\]

\[
B_{22} = \begin{bmatrix}
\frac{E'(t)}{E(t)} - \frac{I'(t)}{I(t)} - (\beta I(t) + 2d + p + \mu + \alpha) & 0 \\
\beta I(t) & \frac{E'(t)}{E(t)} - \frac{I'(t)}{I(t)} - (2d + \epsilon + \mu + \alpha)
\end{bmatrix}
\]
The vector norm $| \cdot |$ in $\mathbb{R}^3$ is chosen as

$$ |(u, v, w)| = \sup\{|u|, |v| + |w|\}. $$

The Lozinskii measure $\mu(B)$ with respect to the norm $| \cdot |$ can be calculated as follow as (see [22]):

$$ \mu(B) \leq \sup\{g_1, g_2\} \tag{5.2} $$

where

$$ g_1 = B_{11} + |B_{12}| = -\beta I(t) - 2d - p - \epsilon + \beta \frac{S(t)I(t)}{E(t)} $$

$$ g_2 = |B_{21}| + \mu_1(B_{22}) \leq \epsilon \frac{E(t)}{I(t)} + \frac{E'(t)}{I(t)} - \frac{r(t)}{I(t)} - (2d + p + \mu + \alpha), $$

if $p \leq \epsilon$. \tag{5.3}

Note that $\mu_1(B_{22})$ is the Lozinskii measure of the $2 \times 2$ matrix $B_{22}$ with respect to the $l_1$ norm in $\mathbb{R}^2$, $|B_{12}|$ and $|B_{21}|$ are the operator norms of $B_{12}$ and $B_{21}$ when they are regarded as mappings from $\mathbb{R}^2$ to $\mathbb{R}$, and $\mathbb{R}^2$ is endowed with the $l_1$ norm. Also note that since $B_{11}$ is a scalar, its Lozinskii measure with respect to any vector norm in $\mathbb{R}^1$ is equal to $B_{11}$. A solution $(S(t), E(t), I(t))$ to (2.3) with $(S(0), E(0), I(0))$ in the basin of attraction of the absorbing set $K$ exists for all $t > 0$. From the equations in (2.3), we find

$$ \frac{\beta S(t)I(t)}{E(t)} = \frac{E'(t)}{E(t)} + (d + \epsilon) \tag{5.4} $$

Relations (5.2) – (5.4) imply $\mu(B) \leq -(d + p) + \frac{E'(t)}{E(t)}$.

Since (2.3) is uniformly persistent when $R_0 > 1$, there exist $c > 0$ and $T > 0$ such that $t > T$ implies $E(t) \geq c$, and $\log \frac{E(t)}{E(0)}$ is bounded for all $(S(0), E(0), I(0)) \in K$.

Then, it is straightforward to calculate $q_2$ as follows:

$$ q_2 = \limsup_{t \to \infty} \sup_{x_0 \in K} \frac{1}{t} \int_0^t \mu(B(x(s, x_0)))ds $$

$$ \leq \limsup_{t \to \infty} \left( -(d + p) + \frac{1}{t} \log \frac{E(t)}{E(0)} \right) = -(d + p) < 0 $$

for all $(S(0), E(0), I(0)) \in K$. Thus, from lemma 5.1, we complete the proof of Theorem 5.1.

6 Conclusions and discussion

This paper has considered a SEIR model that incorporates vaccination, recruitment and natural death, as well as disease-caused death, so that the total population size may vary in time. A distinguishing feature of the SEIR model considered here is that there is a proportional vaccination.
The vaccination reproduction number $R_0$ defined by (3.1) of this SEIR model (2.3) is a sharp threshold parameter which completely determines the global dynamics of the system (2.3) and the outcome of the disease. If $R_0 \leq 1$, the disease-free equilibrium is globally stable so that the disease always dies out. If $R_0 > 1$, the disease-free equilibrium becomes unstable while the endemic equilibrium emerges as the unique positive equilibrium and it is globally stable in the interior of the feasible region $D$ under the restriction $p \leq \epsilon$. The technical condition $p \leq \epsilon$ used in theorem 5.1 is satisfied if the successful vaccination rate has a low level or the disease causes a short latent period. This condition includes both limiting cases when $\epsilon \to \infty$ and $p = 0$.

Note that when $p = 0$ in the SEIR model in this paper, the model (2.3) is written as follows:

\[
\begin{align*}
S'(t) &= A - \beta S(t)I(t) - dS(t) \\
E'(t) &= \beta S(t)I(t) - (d + \epsilon)E(t) \\
I'(t) &= \epsilon E(t) - (d + \alpha + \mu)I(t)
\end{align*}
\] (6.1)

The reproduction number of (6.1) is $R_{01} = \frac{\epsilon \beta A}{d(d+\epsilon)(d+\alpha+\mu)}$. If $R_{01} > 1$, then $(S^*_1, E^*_1, I^*_1)$ is the endemic equilibrium of (6.1), where $S^*_1 = \frac{(d+\epsilon)(d+\alpha+\mu)}{\epsilon \beta}$, $I^*_1 = \frac{\epsilon E^*}{\delta}$, $E^*_1 = \frac{\epsilon \beta A - d(d+\epsilon)(d+\alpha+\mu)}{\epsilon \beta(d+\epsilon)}$. But, $S^* = S^*_1$, $E^* = \frac{A \epsilon \beta - (d+p) \omega \delta}{\omega \epsilon \beta}$, $(p > 0)$, $I^* = \frac{\epsilon E^*}{\delta}$. Obviously, $E^*_1 > E^*$, then $I^*_1 > I^*$. Thus, although the threshold behavior and dynamic behavior of the model (2.3) in this paper are similar to those of the model (6.1), the vaccination reproduction number $R_0$ of (2.3) is less than the basic reproduction number $R_{01}$ of (6.1), and the infective level of the endemic equilibrium state is less than those of (6.1). All these demonstrate completely the effect of vaccination.

References


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