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Research Article

Successive Vaccination and Difference in Immunity of a Delay SIR Model with a General Incidence Rate

Yongzhen Pei,[1] Li Changguo,[2] Qianyong Wu,[1] and Yunfei Lv[3]

[1] *School of Computer Science and Software Engineering, Tianjin Polytechnic University, Tianjin 300387, China*
[2] *Department of Basic Science, Military Transportation University, Tianjin 300161, China*
[3] *School of Science, Tianjin Polytechnic University, Tianjin 300387, China*

Correspondence should be addressed to Yongzhen Pei; yongzhenpei@163.com

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A delay SIR epidemic model with difference in immunity and successive vaccination is proposed to understand their effects on the disease spread. From theorems, it is obtained that the basic reproduction number governs the dynamic behavior of the system. The existence and stability of the possible equilibria are examined in terms of a certain threshold condition about the basic reproduction number. By use of new computational techniques for delay differential equations, we prove that the system is permanent. Our results indicate that the recovery rate and the vaccination rate are two factors for the dynamic behavior of the system. Numerical simulations are carried out to investigate the influence of the key parameters on the spread of the disease, to support the analytical conclusion, and to illustrate possible behavioral scenarios of the model.

1. Introduction

The current threat of some new type diseases has raised our awareness that curbing the spread of some emerging and reemerging human diseases is of public health importance such as H1N1. This emerging disease, which was first reported in Mexico, spread very quickly, due to the travel of infected persons by airplanes, trains, and buses to some other regions. It continued to spread around the world and caused about 5000 deaths. In recent years, many mathematical models have been developed for the transmission dynamics of infectious diseases such as SARS, HIV/AIDS, measles, and smallpox ([1–6], to name a few, and the references therein). These models have provided understanding of the underlying mechanisms which influence the spread of diseases and suggested some control strategies. Moreover, to our knowledge, the first effective control strategy for the elimination of infectious diseases is obtaining immunity. It has been reported that "People's immunity to A/H1N1 flu virus is greater than previously thought after access vaccines. The WHO is working to give more nations access vaccines to fight the H1N1 flu pandemic."

Besides these above studies, many authors formulated and analyzed SIR epidemic models for the control of diseases [7–20]. In particular, some authors have studied the effects of vaccination on the spread of diseases [7–11]; others have studied the effects of treatment on the spread of diseases [12–15]. Gao et al. have proposed an epidemic model with density-dependent birth pulses and seasonal prevention [16]. Recently, some works have investigated permanent and temporary immunity [17–20]. However, in these SIR models, an unrealistic assumption is that all the rest of infected individuals acquire immunity besides death. Measles encephalitis in adults in [21, 22] shows that there is difference in immunity of infected individuals. That is, some infected individuals can acquire immunity after recovery, but some do not acquire immunity and can be infected once more. At the same time, vaccination is an important strategy for the elimination of infectious diseases [7–11].

Vaccinations have many types; impulsive vaccination and successive vaccination are two main policies. Successive vaccination is that people have been vaccinated at birth to protect themselves from disease; the studies can be found in

[23, 24]. Makinde in [23] studied a SIR model for the transmission dynamics of a childhood disease in the presence of a preventive vaccine and analyzed the vaccination reproductive number for disease control and eradication qualitatively. Impulsive vaccination (only at fixed time sequence we execute effectively the vaccination for the disease) is an important and effective strategy for the elimination of infectious diseases and has been studied in the literature. For example, see [10, 17, 25–27]. In above-mentioned papers, authors almost considered the vaccination of susceptible population. But, in fact, under a certain situation, the vaccine treatment also should be considered for the newborns of the susceptible, the exposed, and the removed. We find that there are few studies on the aspect of the vaccination of newborns. In this case, successive vaccination seems more reasonable than impulsive vaccination. Therefore, in this paper, a SIR model with difference in immunity and successive vaccination is considered.

As far as disease transmission is concerned, the incidence rate, defined as the rate of new infection, plays a very important role in modelling infectious diseases. Bilinear incidence rate βSI in [28, 29] and standard incidence rates $\lambda SI/N$ in [30, 31] have often been used in epidemic models. However, it is unreasonable to consider the bilinear incidence rate (based on the law of mass action) as the number of susceptibles is large, owing to the number of susceptibles with which every infective contact within a certain time is limited. Standard incidence rate may be a good approximation if the number of available partners is large enough but it is not possible to make more contacts when the population N is small. Combine the two previous approaches by assuming that if the number of available partners N is low, the number of actual per capita partners is proportional to N, whereas if the number of available partners is large, there is a saturation effect which makes the number of actual partners constant. Considering this case, a saturation incidence rate of type $f(I)S$ with $f(I) = kI/(1 + \alpha I)$ is being proposed in [32]. More general incidence rate used in the literature is the one for which $f(I) = kI^l/(1 + \alpha I^h)$ [33, 34], where I^l measures the infection force of the disease and $f(I) = 1/(1 + \alpha I^h)$ measures the inhibitory effect caused by behavioral changes. Note that if $f(I)$ is decreasing when I is large, this may be interpreted as the fact that susceptibles tend to reduce their social contacts if the perceived number of infectives increases over a psychologically significant value. The above saturation incidence rate depends also on the size of the infectives I termed as infectives-dependent. Particular examples of susceptibles-dependent incidence rate are $f(S) = kS/(1+\alpha S)$ [35]. Very general incidence rates which are not linear in S are also used in Derrick and van den Driessche [36] ($f(S, I, N) = I\Phi(S, I, N)$, where $N = S + I$), Korobeinikov and Maini [37] ($f(S, I) = h_1(I)h_2(S)$), and Moghadas and Alexander [38] ($f(S, I) = \beta(1 + g(I, v))IS$).

Nie et al. [19] and Ji et al. [39] respectively considered a delay SIR epidemic model with nonlinear incidence rate and density-dependent birth and death rates. Motivated by the main idea described in [6, 39], in this paper, we consider a delay SIR model with difference in immunity and successive vaccination and an abstract incidence rate. The main difference between our study and those described in [6, 39] is the difference in immunity and successive vaccination and an abstract incidence rate. An abstract incidence rate of type $f(I)S$ is employed to model the spread of the disease which is propagated through the infective individuals, under a few biologically feasible assumptions upon $f(I)$.

In view of above facts, we will formulate a mathematical model in Section 2. We provide the region of biologically feasible solutions in Section 3. Then, we study the existence and stability of the steady states in the next section, analyze the permanence result in Section 5, and give some numerical simulations in Section 6. Lastly, we end the paper with a brief discussion of our results in Section 6.

2. Model Formulation and Invariant Region

In this section, we will present a delay SIR epidemic model with a general nonlinear incidence rate. The total population $N(t)$ is divided into three subclasses, namely, the susceptibles $S(t)$, the infectives $I(t)$, and the recovered individuals $R(t)$. Based on the SIR model in [12, 39], we considered following system:

$$\begin{aligned}
\frac{dS}{dt} &= \left[b - \frac{arN}{K}\right] N - \beta e^{-d_1\tau} S f\left(I\left(t-\tau\right)\right) \\
&\quad - \left[d + \frac{(1-a)\, rN}{K}\right] S - \theta S + \mu_1 I, \\
\frac{dI}{dt} &= \beta e^{-d_1\tau} f\left(I\left(t-\tau\right)\right) S \\
&\quad - \left[d + \frac{(1-a)\, rN}{K}\right] I - \mu_1 I - e^{-d_1\omega}\delta I\left(t-\omega\right), \\
\frac{dR}{dt} &= e^{-d_1\omega}\delta I\left(t-\omega\right) - \left[d + \frac{(1-a)\, rN}{K}\right] R + \theta S, \\
N\left(0\right) &= S_0 > 0, \qquad I\left(0\right) = \varphi\left(\theta\right) \geq 0, \\
&\quad \forall \theta \in [-\overline{\tau}, 0], \; R\left(0\right) = R_0 \geq 0,
\end{aligned} \tag{1}$$

where $\overline{\tau} = \max\{\tau, \omega\}$ and $\varphi \in C([-\overline{\tau}, 0], R)$. We give the following useful assumptions.

(1) There are no disease induced deaths, and all the newborns are susceptible.

(2) $f(I)$ is the nonlinear incidence rate satisfying the following assumptions:

$$\begin{aligned}
&f\left(0\right) = 0, \qquad f'\left(I\right) > 0, \qquad f''\left(I\right) < 0, \\
&\lim_{t\to\infty} f\left(I\right) = c < +\infty.
\end{aligned} \tag{2}$$

(3) The force of infection at any time t is dominated by $\beta e^{-d_1\tau} S(t) f(I(t-\tau))$, where τ is incubation period and $0 < e^{-d_1\tau} \leq 1$ represents the survival probability of individuals in the population after time τ [20]. It is also assumed that $d_1 \leq d$ in $[-\tau, 0]$, where d is the death rate and d_1 is the death rate in the time interval $[-\tau, 0]$.

(4) The parameters a are a convex combination constant, $r = b - d > 0$ is the intrinsic growth rate (b is the birth rate), and $K > 0$ is the carrying capacity of the population. The term $(b - (arN(t)/K))$ has a density-dependent per capita birth rate and the term $(d + ((1 - a)rN(t)/K))$ has a density-dependent per capita death rate [39].

(5) For $0 < a < 1$, the birth and death rates are consistent with the limited resources associated with density dependence. The birth rate is density independent when $a = 0$ and the death rate is density independent when $a = 1$. Thus, the spread of the disease (animals such as rodents, etc.) is assumed to be governed by the following system of logistic equations with time delay.

(6) The total population is assumed to be large enough to be adequately described by a deterministic model and is divided into compartments based on the disease status [40].

(7) The successive vaccination rate θ is positive. The positive constant μ_1 is the recovery rate of the infectious individuals from compartment I to S. The parameters β are the effective per capita contact rate constant of infected individuals. The parameters δ are the recovery rate of infected individuals.

(8) Models are formulated as functional differential and/or integral equations when time delay is included [40]. Ours follows the former with the assumption that the I-equation satisfies a certain integral condition [41].

Models with multiple delays are not common, but few authors have in the past considered these-Beretta et al. [42]- to name but a few. Since $N(t) = S(t) + I(t) + R(t)$, thus the governing equation (1) can be rewritten as

$$\frac{dN}{dt} = r\left[1 - \frac{N}{K}\right]N,$$

$$\begin{aligned}\frac{dI}{dt} &= \beta e^{-d_1\tau}(N - I - R)\, f(I(t-\tau)) \\ &\quad - \left[d + \frac{(1-a)\, rN}{K}\right] I - \mu_1 I - e^{-d_1\omega}\delta I(t-\omega), \qquad (3)\\ \frac{dR}{dt} &= e^{-d_1\omega}\delta I(t-\omega) \\ &\quad - \left[d + \frac{(1-a)\, rN}{K}\right] R + \theta(N - I - R).\end{aligned}$$

Let $\overline{\tau} = \max(\omega, \tau)$. Then (3) satisfies the following initial conditions

$$\begin{aligned} N(0) = S_0 > 0, \qquad I(0) = \varphi(\theta) \geq 0, \\ \forall \theta \in [-\overline{\tau}, 0], \ R(0) = R_0 \geq 0. \end{aligned} \qquad (4)$$

In this paper, we will consider two different delays τ, ω which are important parameters on the dynamic behavior. So, the present study is continuation of the previous work $\tau = \omega$ by Naresh et al. [43].

Lemma 1. *All solutions of the model system* (3) *starting in* R^3_+ *are bounded and eventually enter the compact attracting set*

$$\Phi = \left\{(S, I, R) \in R^3_+ : S(t) + I(t) + R(t) = N(t) \leq K\right\}. \qquad (5)$$

Lemma 2. *Let the initial data be* $N(0) = S_0 > 0$, $I(0) = I_0(u) \geq 0$, *for all* $u \in [-\overline{\tau}, 0]$, *with* $I_0(0) > 0$, $R(0) = R_0 \geq 0$. *Then, the solution* $(S(t), I(t), R(t))$ *of the model remains positive for all time* $t > 0$.

Lemma 3 (see [44]). *For the characteristic equation in the form* $p(\lambda) + q(\lambda)e^{-r\lambda} = 0$, *where* p *and* q *are polynomials with real coefficients and* $r > 0$ *is the delay, suppose*

(a) $p(\lambda) \neq 0$, $R(\lambda) > 0$;

(b) $|q(iy)| < |p(iy)|$; $0 \leq y < \infty$;

(c) $\lim_{|\lambda| \to \infty, R(\lambda) \geq 0} |q(\lambda)/p(\lambda)| = 0$.

Then $R(\lambda) < 0$ *for every root* λ *and all* $r > 0$.

3. Equilibrium and Stability Analysis

In this section, we focus on the existence and local stability of equilibria. Let the right-hand side of equalities in model (3) be zero. Then, there are two equilibria; namely,

(i) $E_0 = (K, 0, p)$, $p = K\theta/[d + (1-a)r + \theta])$, disease-free equilibrium;

(ii) $E^* = (N^*, I^*, R^*)$, endemic equilibrium,

where the values of N^*, I^*, and R^* are given in Section 3.2.

3.1. Community Matrix. Firstly, after computing the Jacobian or community matrix of model (3) at point (N, I, R), the characteristic equation is given by

$$\begin{vmatrix} r - \frac{2r}{K}N - \lambda & 0 & 0 \\ \beta e^{-d_1\tau} f(I) - \frac{(1-a)\, r}{K} I & me^{-(d_1+\lambda)\tau} - \delta e^{-(d_1+\lambda)\omega} - n - \lambda & \beta e^{-d_1\tau} f(I) \\ \theta - \frac{(1-a)\, r}{K} R & \delta e^{-(d_1+\lambda)\omega} - \theta & -\left[d + \frac{(1-a)\, r}{K} N + \theta\right] - \lambda \end{vmatrix} = 0, \qquad (6)$$

where $m = \beta(N - I - R)f'(I)$, $n = \beta e^{-d_1\tau} f(I) + d + ((1 - a)r/K)N + \mu_1$.

Now, we analyze the equilibria stability of system (3). Computing the Jacobian of system (3) evaluated at E_0, one gets the following matrix

$$J(E_0) = \begin{pmatrix} -r - \lambda & 0 & 0 \\ 0 & a_{22} - \lambda & 0 \\ \dfrac{(d+\theta)\theta}{d + (1-a)r + \theta} & \delta e^{-(d_1+\lambda)\tau} - \theta & -[d + (1-a)r + \theta] - \lambda \end{pmatrix}, \tag{7}$$

where

$$a_{22} = \beta(K - p) f'(0) e^{-(d_1+\lambda)\tau} - \delta e^{-(d_1+\lambda)\omega} - [d + (1-a)r + \mu_1]. \tag{8}$$

Denote

$$A = \beta(K - p) f'(0), \qquad C = d + (1 - a)r + \mu_1; \tag{9}$$

then

$$a_{22} = Ae^{-(d_1+\lambda)\tau} - \delta e^{-(d_1+\lambda)\omega} - C. \tag{10}$$

Denote

$$h(\lambda) = Ae^{-(d_1+\lambda)\tau} - \delta e^{-(d_1+\lambda)\omega} - C - \lambda. \tag{11}$$

The eigenvalues of the system (3) about the steady state E_0 are $\lambda_1 = -r$, $h(\lambda) = 0$ and $\lambda_3 = -[d + (1 - a)r + \theta]$. All the parameters of the model are assumed to be nonnegative. Therefore, λ_1 and λ_3 are negative. Next, we discuss the roots of $h(\lambda) = 0$ in five cases.

Case 1. For $\tau = \omega \neq 0$, from the second equation of the system (3), we can get the following.

Proposition 4. *For $\tau = \omega > 0$, $R(\lambda) < 0$ for every root λ of $h(\lambda) = 0$ when*

$$(A - \delta) e^{-d_1\tau} < C. \tag{12}$$

Proof. From the above analysis, λ_2 satisfies the following characteristic equation:

$$g(\lambda) = (A - \delta) e^{-(d_1+\lambda)\tau} - C - \lambda = 0. \tag{13}$$

(1) Clearly, $\lambda = 0$ is not a root of $g(\lambda) = 0$.

(2) From the fact that $g(0) < 0$, $g'(\lambda) < 0$ for $\lambda > 0$, it is obtained that $g(\lambda) = 0$ has no positive real root.

(3) It is sufficient to show that $g(\lambda) = 0$ does not admit a purely imaginary root. In fact, if $\lambda = iv$ $(v > 0)$ is a root of $(g(\lambda) = 0)$, then by separating the real part, one gets

$$(A - \delta) e^{-d_1\tau} \cos(v\tau) = C. \tag{14}$$

Together with the condition of Proposition 4, we have

$$\cos(v\tau) > 1. \tag{15}$$

This is impossible.

(4) It is easy to show that $g(\lambda) = 0$ has no imaginary root whose real part is positive. Otherwise, there is an imaginary root $\lambda = u + iv$ with $u > 0$. Without any loss of generality, we consider $v > 0$. Then, we take the real and imaginary parts of $g(\lambda) = 0$; namely,

$$(A - \delta) e^{-(d_1+u)\tau} \cos(v\tau) = C + u. \tag{16}$$

Combined with (9), we have

$$C > (A - \delta) e^{-d_1\tau} > (A - \delta) e^{-(d_1+u)\tau} \cos(v\tau) = C + u. \tag{17}$$

This is a contradiction which implies that all eigenvalues roots of $g(\lambda)$ have negative real parts. Therefore, the disease-free equilibrium of the system (3) is locally asymptotically stable when (9) holds. The proof is completed. □

Case 2. For $\tau \neq 0, \omega = 0$, by the same way as in Case 1, one gets the following.

Proposition 5. *For all $\tau \neq 0$, $\omega = 0$, $R(\lambda) < 0$ for every root λ of $h(\lambda) = 0$ when*

$$Ae^{-d_1\tau} - \delta < C. \tag{18}$$

Case 3. For $\omega \neq 0$, $\tau = 0$, one gets the following.

Proposition 6. *For $\omega \neq 0$, $\tau = 0$, $R(\lambda) < 0$ for every root λ of $h(\lambda) = 0$ when*

$$\delta e^{-d_1\omega} < |A - C|. \tag{19}$$

Proof. By the fact that $h(\lambda) = 0$ is equivalent to $p(\lambda) + q(\lambda)e^{-\lambda\omega} = 0$ with $p(\lambda) = A - C - \lambda$, $q(\lambda) = -\delta e^{-d_1\omega}$.

(i) Suppose $\lambda = u + iv$ $(u > 0)$. Then, $p(\lambda) = A - C - u - iv \neq 0$.

(ii) By $|q(iv)| = \delta e^{-d_1\omega}$, $|p(iv)| = |A - C - iv| = \sqrt{(A - c)^2 + v^2}$, together with the condition of Proposition 6, we know that $|q(iv)| < |p(iv)|$.

(iii) Suppose $\lambda = u + iv$, $(u > 0)$. Then,

$$\lim_{u^2+v^2 \to +\infty} \left| \frac{q(\lambda)}{p(\lambda)} \right| = \delta e^{-d_1 \omega},$$
$$\lim_{u^2+v^2 \to +\infty} \frac{1}{\sqrt{(A - C - u)^2 + v^2}} = 0. \tag{20}$$

Then, using Lemma 3, we have $R(\lambda) < 0$ for all ω. □

Case 4. For $\omega > \tau > 0$, let $\varepsilon = \omega - \tau$. Then, $\omega = \tau + \varepsilon$. For fixed τ,

$$h(\lambda) = Ae^{-(d_1+\lambda)\tau} - \delta e^{-(d_1+\lambda)(\tau+\varepsilon)} - C - \lambda. \tag{21}$$

Let $\lambda = u + iv$ $(u > 0)$. Then, we take the real and imaginary parts of $h(\lambda) = 0$; namely,

$$\begin{aligned} &Ae^{-(d_1+u)\tau} \cos(v\tau) - \delta e^{-(d_1+u)(\tau+\varepsilon)} \cos(v(\tau+\varepsilon)) \\ &\quad = C + u, \\ &-Ae^{-(d_1+u)\tau} \sin(v\tau) - \delta e^{-(d_1+u)(\tau+\varepsilon)} \sin(v(\tau+\varepsilon)) = v. \end{aligned} \tag{22}$$

Sum of squares of the above equalities is

$$\begin{aligned} &A^2 e^{-2(d_1+u)\tau} + \delta^2 e^{-2(d_1+u)(\tau+\varepsilon)} \\ &\quad - 2A\delta e^{-(d_1+u)(2\tau+\varepsilon)} \cos(v\varepsilon) - (C+u)^2 - v^2 = 0. \end{aligned} \tag{23}$$

Then, we have

$$\left.\frac{\partial u}{\partial \varepsilon}\right|_{\varepsilon=0} = \frac{\delta(u + d_1)(A - \delta)}{(A-\delta)^2 \tau + (C+u)e^{2\tau(u+\delta)}}. \tag{24}$$

Obviously, $\partial u/\partial \varepsilon|_{\varepsilon=0} < 0$ when $A - \delta < 0$. Combined with $(A-\delta)e^{-d_1\tau} < Ae^{-d_1\tau} - \delta e^{-d_1\omega}$, we have the following.

Proposition 7. *For $\omega > \tau > 0$, $R(\lambda) < 0$ for every root λ of $h(\lambda) = 0$ when*

$$Ae^{-d_1\tau} - \delta e^{-d_1\omega} < C, \quad A - \delta < 0, \quad 0 < \omega - \tau \ll 1. \tag{25}$$

Case 5. For $0 < \tau < \omega$, in the same way as in Case 4, we have the following.

Proposition 8. *For $0 < \tau < \omega$, $R(\lambda) < 0$ for every root λ of $h(\lambda) = 0$ when*

$$Ae^{-d_1\tau} - \delta e^{-d_1\omega} < C, \quad A - \delta > 0, \quad 0 < \tau - \omega \ll 1. \tag{26}$$

From what has been discussed above, we get the following.

Theorem 9. *The disease-free equilibrium of the system* (3) *is locally asymptotically stable if one of the following conditions holds.*

(a) $\tau = \omega \neq 0$, $(A-\delta)e^{-d_1\tau} < C$.
(b) $\tau \neq 0$, $\omega = 0$, $Ae^{-d_1\tau} - \delta < C$.
(c) $\tau = 0$, $\omega \neq 0$, $\delta e^{-d_1\omega} < |A - C|$.
(d) $Ae^{-d_1\tau} - \delta e^{-d_1\omega} < C$, $A - \delta < 0$, $0 < \omega - \tau \ll 1$.
(e) $Ae^{-d_1\tau} - \delta e^{-d_1\omega} < C$, $A - \delta > 0$, $0 < \tau - \omega \ll 1$.

3.2. Existence of Endemic Equilibrium. Thus, by Theorem 9, we may define the basic reproduction number as

$$R_0 = \frac{Ae^{-d_1\tau} - \delta e^{-d_1\omega}}{C}. \tag{27}$$

This threshold R_0 defines the average number of secondary infections generated by a typical infectious individual in a completely susceptible population in a steady demographic state.

In Theorem 9, we have already shown that the system (3) has an infection-free steady state which is locally asymptotically stable under condition $R_0 < 1$. The disease-free equilibrium is unstable when $R_0 > 1$, and the system (3) has a nontrivial endemic equilibrium $E^* = (N^*, I^*, R^*)$ when $R_0 > 1$. From (3),

$$\begin{aligned} N^* &= K > 0, \\ R^* &= \frac{\delta e^{-d_1\omega} - \theta}{d + (1-a)r + \theta} I^* \\ &\quad + \frac{K\theta}{d + (1-a)r + \theta} \doteq qI^* + p, \end{aligned} \tag{28}$$

where $q = (e^{-d_1\omega}\delta - \theta)/(d + (1-a)r + \theta)$. Substituting these values of N^* and R^* in the second equation of (3), we get the following equation for I:

$$G(I) = \beta e^{-d_1\tau}(K - (1+q)I - p) f(I) - \left[C + \delta e^{-d_1\omega}\right] I. \tag{29}$$

Obviously, $I = 0$ is one of the roots of (29) as $f(0) = 0$. Therefore, to exclude that root, choose

$$H(I) = \beta e^{-d_1\tau}(K - (1+q)I - p)\frac{f(I)}{I} - \left[C + \delta e^{-d_1\omega}\right]. \tag{30}$$

It can easily be seen that the function $H(I)$ is negative for large positive I; that is,

$$H(K) = -\beta e^{-d_1\tau}(Kq + p)\frac{f(k)}{K} - \left[C + \delta e^{-d_1\omega}\right] < 0. \tag{31}$$

Next, we determine the sign of its derivative

$$\begin{aligned} H'(I) &= \beta e^{-d_1\tau}(K - p)\frac{f'(I)I - f(I)}{I^2} \\ &\quad - \beta e^{-d_1\tau}(1+q) f'(I). \end{aligned} \tag{32}$$

It can easily be seen that $K > p$. In addition, from the properties of the function $f(I)$, in particular from $f(0) = 0$ and $f''(0) < 0$, it follows that $f(I) - f'(I)I > 0$, and consequently $H'(I) < 0$ for all $I > 0$. Therefore, for a positive root of $H(I) = 0$ to exist, $H(I)$ has to satisfy $H(0) > 0$; that is,

$$\begin{aligned} H(0) &= Ae^{-d_1\tau} - \delta e^{-d_1\omega} - C \\ &= \left(\frac{Ae^{-d_1\tau} - e^{-d_1\omega}\delta}{C} - 1\right) C \\ &= (R_0 - 1)C. \end{aligned} \tag{33}$$

Hence, one needs the requirement that $R_0 > 1$ to ensure the existence of the endemic equilibrium. From the above analysis, we have the following theorem.

Theorem 10. *The system* (3) *has a nontrivial endemic equilibrium* $E^* = (N^*, I^*, R^*)$ *when* $R_0 > 1$.

3.3. Local Stability of the Endemic Equilibrium. In this section, we analyze the local stability of the endemic equilibrium E^* for $\tau = \omega$. Its characteristic equation is given by

$$\begin{vmatrix} -r-\lambda & 0 & 0 \\ \beta e^{-d_1\tau} f(I^*) - \dfrac{(1-a)rI^*}{K} & me^{-(d_1+\lambda)\tau} - n - \lambda & \beta e^{-d_1\tau} f(I^*) \\ \theta - \dfrac{(1-a)rR^*}{K} & \delta e^{-(d_1+\lambda)\tau} - \theta & -[d+(1-a)r+\theta] - \lambda \end{vmatrix} = 0, \tag{34}$$

where $m = \beta(K - I^* - R^*)f'(I^*) - \delta$, $n = \beta e^{-d_1\tau} f(I^*) + d + (1-a)r + \mu_1$.

The Jacobin matrix leads to the characteristic equation

$$(\lambda + r)\left[\lambda^2 + m_1\lambda + m_0 + (n_1\lambda + n_0)e^{-\lambda\tau}\right] = 0, \tag{35}$$

where

$$\begin{aligned} m_1 &= \beta e^{-d_1\tau} f(I^*) + 2d + 2(1-a)r + \theta + \mu_1 > 0, \\ m_0 &= \left[\beta e^{-d_1\tau} f(I^*) + d + (1-a)r + \mu_1\right] \\ &\quad \times (d + (1-a)r + \theta) + (d + (1-a)r + \mu_1)\theta > 0, \\ n_1 &= -\left[\beta f'(I^*)(K - I^* - R^*) - \delta\right] e^{-d_1\tau}, \\ n_0 &= \left[\beta f'(I^*)(K - I^* - R^*) - \delta\right][d + (1-a)r + \theta] \\ &\quad \times e^{-d_1\tau} + \delta f(I^*) e^{-2d_1\tau}. \end{aligned} \tag{36}$$

Since all the model parameters are assumed to be nonnegative, it follows that one eigenvalue is negative; that is, $\lambda_1 = -r$. Thus, the stability of E^* depends on the roots of the quasi-polynomial

$$\lambda^2 + m_1\lambda + m_0 + (n_1\lambda + n_0)e^{-\lambda\tau} = 0. \tag{37}$$

We note that $m_1 > 0$ and $m_0 > 0$, whereas n_1 and n_0 may be positive or negative. For $\tau = 0$, we state the following results that follow directly from (39). The endemic steady state is locally asymptotically stable if the following conditions hold:

$$\begin{aligned} &\beta f(I^*) + 2d + 2(1-a)r + \theta + \mu_1 + \delta\beta f'(I^*) \\ &\quad > \beta(K - I^* - R^*) f'(I^*), \end{aligned} \tag{H_1}$$

$$\begin{aligned} &\left[\beta f(I^*) + d + (1-a)r + \mu_1 + \delta - \beta(K - I^* - R^*) f'(I^*)\right] \\ &\quad \times [d + (1-a)r + \theta] + f(I^*)\delta > 0. \end{aligned} \tag{H_2}$$

The main purpose of this paper is to study the stability behavior of E^* in the case $\tau \neq 0$. Obviously, $i\eta$ ($\eta > 0$) is the root of (29) if and only if η satisfies

$$-\eta^2 + m_1 i\eta + m_0 = -(n_1 i\eta + n_0)(\cos\eta\tau - i\sin\eta\tau). \tag{38}$$

Separating the real and imaginary parts, we have

$$-\eta^2 + m_0 = -n_0\cos\eta\tau - n_1\eta\sin\eta\tau, \tag{39}$$

$$m_1\eta = -n_1\eta\cos\eta\tau + n_0\sin\eta\tau. \tag{40}$$

Eliminating τ by squaring and adding (39) and (40), we obtain a polynomial in η as

$$\eta^4 + \left(m_1^2 - n_1^2 - 2m_0\right)\eta^2 + m_0^2 - n_0^2 = 0. \tag{41}$$

Suppose that the conditions

$$m_1^2 > n_1^2 + 2m_0, \quad m_0^2 > n_0^2 \tag{H_3}$$

hold for all $\tau \geq 0$. Then, the infected steady state of the system (3) is locally asymptotically stable.

Theorem 11. *For* $\tau = \omega$, *if* $R_0 > 1$, *then the endemic equilibrium of the system* (3) *is locally asymptotically stable, when conditions* (H_1)–(H_3) *hold.*

Corollary 12. *For* $\tau = \omega$, *if* $\mu_1 < \mu_1^*$ *or* $\theta < \theta^*$, *then the endemic equilibrium of the system* (3) *is locally asymptotically stable, when conditions* (H_1)–(H_3) *hold.*

4. Permanence

In this section, we investigate a permanence result [5]. The following is our main result of this paper. We will give the following result by using some techniques given in [8, 11]. The proof of the permanence with nonlinear incidence is a daunting task. Consequently, for simplicity and mathematical convenience, let us choose a linear incidence rate $f(I) = I$. The result holds with the nonlinear incidence, as shown numerically, but the algebraic proof is long and tedious, and the conditions to impose on some of the parameters may be very restrictive. Now, let us firstly give the following theorem.

Theorem 13. *If $R_0 > 1$ holds, then the system* (1) *with $\tau = \omega$ is permanent; that is, there are positive constants c_i $(i = 1, 2, 3)$ such that*

$$\begin{aligned} c_1 &< \lim_{t\to\infty} \inf S(t) \le \lim_{t\to\infty} \sup S(t) \le K, \\ c_2 &< \lim_{t\to\infty} \inf I(t) \le \lim_{t\to\infty} \sup I(t) \le K, \\ c_3 &< \lim_{t\to\infty} \inf R(t) \le \lim_{t\to\infty} \sup R(t) \le K \end{aligned} \tag{42}$$

hold for any solution of (1) *with $(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta))$ in the interior of Φ for all $\theta \in [-\tau, 0]$. In fact, c_i $(i = 1, 2, 3)$ can be chosen explicitly as*

$$\begin{aligned} c_1 &= \frac{(b-ar)K}{\beta K e^{-d_1\tau} + d + (1-a)r + \theta}, \\ c_2 &= I^* e^{-(d+(1-a)r+\mu_1+e^{-d_1\tau}\delta)}, \\ c_3 &= \frac{\delta e^{-d_1\tau} c_2 + \theta c_1}{d + (1-a)r}. \end{aligned} \tag{43}$$

Proof. Note that $0 < N(t) < K$ for all $t \ge 0$ and that $\lim_{t\to\infty} N(t) = K$. It is easy to see that $\lim_{t\to\infty} \inf S(t) \ge c_1$. In fact, let $\epsilon < K$ be arbitrary. Choose $T_1 > \tau$ so large that $N(t) > K - \epsilon$ for $t > T_1$. We have the following inequality:

$$\begin{aligned} \dot{S}(t) &> -\left[\beta K e^{-d_1\tau} + d + (1-a)r + \theta\right] S(t) \\ &\quad + (b-ar)(K-\epsilon), \end{aligned} \tag{44}$$

for all $t \ge T_1$, which implies that

$$\liminf_{t\to\infty} S(t) \ge \frac{(b-ar)(K-\epsilon)}{\beta K e^{-d_1\tau} + d + (1-a)r + \theta}. \tag{45}$$

Note that ϵ may be arbitrarily small so that $\lim_{t\to\infty} \inf S(t) \ge c_1$.

Next, we will show $\lim_{t\to\infty} \inf I(t) \ge c_2$. For any ξ : $0 < \xi < 1$, we see the inequality $S^* < [(b-ar)K + \mu_1 I^*]/(\beta e^{-d_1\tau}\xi I^* + d + (1-a)r + \theta)$. There exist sufficiently large $\rho \ge 1$ and sufficiently small ϵ such that $S^* < \{[(b-ar)(K-\epsilon) + \mu_1 I^*]/(\beta e^{-d_1\tau}\xi I^* + d + (1-a)r + \theta)\}(1 - e^{-(\beta e^{-d_1\tau}\xi I^* + d + (1-a)r + \theta)\rho\tau}) \equiv S^\Delta$. We show that $I(t_0) > qI^*$ for some $t_0 \ge \rho\tau$. In fact, if not, it follows from the first equation of (1) that, for all $t \ge \rho\tau + \tau \ge T_1 + \tau$,

$$\begin{aligned} \dot{S}(t) &\ge (b-ar)(K-\epsilon) + \mu_1 I^* \\ &\quad - \left[\beta e^{-d_1\tau}\xi I^* + d + (1-a)r + \theta\right] S(t). \end{aligned} \tag{46}$$

Hence, for $t \ge \rho\tau + \tau$,

$$\begin{aligned} S(t) &\ge e^{-(\beta e^{-d_1\tau}\xi I^* + d + (1-a)r + \theta)(t-\rho\tau-\tau)} \\ &\quad \times \left[S(\rho\tau+\tau) + (b-ar)(K-\epsilon) + \mu_1 I^*\right] \\ &\quad \times \int_{\rho\tau+\tau}^{t} e^{-(\beta e^{-d_1\tau}\xi I^* + d + (1-a)r + \theta)(t-\rho\tau-\tau)} d\theta \\ &> \frac{\left[(b-ar)(K-\epsilon) + \mu_1 I^*\right]}{\beta e^{-d_1\tau}\xi I^* + d + (1-a)r + \theta} \\ &\quad \times \left(1 - e^{-(\beta e^{-d_1\tau}\xi I^* + d + (1-a)r + \theta)(t-\rho\tau-\tau)}\right), \end{aligned} \tag{47}$$

which gives us, for $t \ge 2\rho\tau + \tau$,

$$S(t) > S^\Delta > S^*. \tag{48}$$

For $t \ge 0$, we define a positive differentiable function $V(t)$ as follows:

$$V(t) = I(t) + \frac{\left[\beta(K-p) - \delta\right] e^{-d_1\tau}}{R_0} \int_{t-\tau}^{t} I(s)\, ds. \tag{49}$$

We obtain the inequality, for $t \ge 2\rho\tau + \tau$,

$$\begin{aligned} \dot{V}(t) &= \left[\beta e^{-d_1\tau}\left(S(t) - S^*\right) I(t-\tau) + (1-a)r\right] \\ &\quad \times \left(1 - \frac{N(t)}{K}\right) I(t) \\ &> \beta e^{-d_1\tau}\left(S(t) - S^*\right) I(t-\tau) \\ &> \beta e^{-d_1\tau}\left(S^\Delta - S^*\right) I(t-\tau). \end{aligned} \tag{50}$$

Let $\underline{i} = \min_{\theta\in[-\tau,0]} I(2\rho\tau + 2\tau + \theta)$. Now, let us show that $I(t) \ge \underline{i}$ for all $t \ge 2\rho\tau + \tau$. In fact, if there exists $T_2 \ge 0$ such that $I(t) \ge \underline{i}$ for $2\rho\tau + \tau \le t \le 2\rho\tau + 2\tau + T_2$, $I(2\rho\tau + 2\tau + T_2) = \underline{i}$ and $\dot{I}(2\rho\tau + 2\tau + T_2) \le 0$. Direct calculation using the second equation of (1) and (48) gives

$$\begin{aligned} &\dot{I}(2\rho\tau + 2\tau + T_2) \\ &\quad > \left[\beta e^{-d_1\tau}\left(S(2\rho\tau + 2\tau + T_2)\right.\right. \\ &\qquad \left.\left. - \left(d + (1-a)r + \mu_1 + e^{-d_1\tau}\delta\right)\right]\underline{i} \\ &\quad > \left(d + (1-a)r + \mu_1 + e^{-d_1\tau}\delta\right)\left[\frac{S^\Delta}{S^*} - 1\right]\underline{i} > 0. \end{aligned} \tag{51}$$

This contradicts the definition of T_2. Thus, we have shown that $I(t) \ge \underline{i}$ for all $t \ge 2\rho\tau + \tau$. Hence, for all $t \ge 2\rho\tau + 2\tau$,

$$\dot{V}(t) > \beta e^{-d_1\tau}\left(S^\Delta - S^*\right)\underline{i}, \tag{52}$$

which implies that $V(t) \to +\infty$ as $t \to +\infty$. This contradicts the boundedness of $V(t)$. Consequently, $I(t_0) > \xi I^*$ for some $t_0 \ge \rho\tau$.

In the rest, we now need to consider two cases:

(i) $I(t) \ge \xi I^*$ for all large t;

(ii) $I(t)$ oscillates about ξI^* for all large t.

We now need to show that $I(t) \geq \xi c_2$ for large t. Obviously, it suffices to show that it holds only for case (ii). We suppose that for any large T there exists $t_1, t_2 > T$ such that $I(t_1) = I(t_2) = \xi I^*$ and $I(t) < \xi I^*$ for $t_1 < t < t_2$. If $t_2 - t_1 \leq \tau$, the second equation (1) gives us $\dot{I}(t) > -(d + (1-a)r + \mu_1)I(t)$, which implies that $I(t) > I(t_1)e^{-(d+(1-a)r+\mu_1)(t-t_1)}$ on (t_1, t_2). Thus, $I(t) > \xi c_2$. On the other hand, if $t_2 - t_1 > \tau$, applying the same manner gives $I(t) \geq \xi c_2$ on $[t_1, t_1 + \tau]$, and hence the remaining work is to show $I(t) \geq \xi c_2$ on $[t_1 + \tau, t_2]$. In fact, assuming that there exists $T_3 > 0$ such that $I(t) \geq \xi c_2$ on $[t_1, t_1 + \tau + T_3]$, $I(t_1 + \tau + T_3) = \xi c_2$, and $\dot{I}(t_1 + \tau + T_3) \leq 0$, it follows from (1) that

$$\begin{aligned} &\dot{I}\left(t_1 + \tau + T_3\right) \\ &\geq \left[\beta e^{-d_1\tau} S\left(t_1 + \tau + T_3\right)\right. \\ &\qquad \left. - \left(d + (1-a)\, r + \mu_1 + e^{-d_1\tau}\delta\right)\right] \xi c_2 \\ &> \left(d + (1-a)\, r + \mu_1 + e^{-d_1\tau}\delta\right)\left[\frac{S^{\Delta}}{S^*} - 1\right] \xi c_2 > 0. \end{aligned} \tag{53}$$

This contradicts the definition of T_3. Hence, $I(t) \geq \xi c_2$ on $[t_1, t_2]$. Consequently, $I(t) \geq \xi c_2$ for large t in the case (ii). Therefore, $\lim_{t\to\infty} \inf I(t) \geq \xi c_2$. Note that q may be so close to 1 that $\lim_{t\to\infty} \inf I(t) \geq c_2$.

Finally, let us show that $\lim_{t\to\infty} \inf R(t) \geq (\delta e^{-d_1\tau} c_2 + \theta c_1)/(d + (1-a)r)$. The third equation gives us

$$\begin{aligned} \dot{R}(t) &\geq \left[\delta e^{-d_1\tau} I + \theta S - [d + (1-a)\, r]\right] R \\ &\geq \left[\delta e^{-d_1\tau} \xi c_2 + \theta \xi c_1 - [d + (1-a)\, r]\right] R \end{aligned} \tag{54}$$

for large t. Hence, $\lim_{t\to\infty} \inf R(t) \geq (\delta e^{-d_1\tau} \xi c_2 + \theta \xi c_1)/(d + (1-a)r)$. In a similar manner, we could show $\lim_{t\to\infty} \inf R(t) \geq c_3$. This proves the theorem. □

Corollary 14. *If $\mu_1 < \mu_1^*$ and $\theta < \theta^*$, then the system* (1) *with $\tau = \omega$ is permanent.*

5. Numerical Analysis

Since it is important to visualize the dynamical behavior of the model, the model system (3) is integrated numerically with the help of MATLAB 7.0 using the following set of parameters.

(1) Let $r = 0.5$, $k = 8$, $d = 0.04$, $d_1 = 0.04$, $\beta = 1$, $a = 0.3$, $\delta = 0.2$, $\mu_1 = 0.8$, $\theta = 0.01$, and $\tau = 5$. It is easy to compute that $E_0 = (8, 0, 0.2)$ and $R_0 = 0.94 < 1$. In Figure 1, the infective population and recovered population, respectively, are plotted against the total population. We see from the figure that for any initial start the solution curves tend to the equilibrium E_0. Hence, we infer that the system (3) may be stable about the disease-free equilibrium point E_0, which satisfies Theorem 9.

(2) Let $r = 0.5$, $k = 8$, $d = 0.04$, $d_1 = 0.04$, $\beta = 1$, $a = 0.3$, $\delta = 0.2$, $\mu_1 = 0.2$, $\theta = 0.02$, and $\tau = 5$. We get $E^* = (8, 2.23, 1.11)$ and this set of parameter values satisfies the local asymptotic stability conditions of E^*. It is

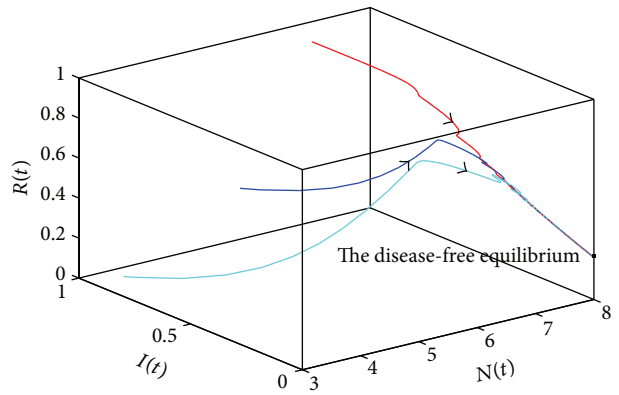

Figure 1: The disease-free equilibrium E_0 is locally asymptotically stable. Variation of infective population $I(t)$ and recovered population $R(t)$ with total population $N(t)$.

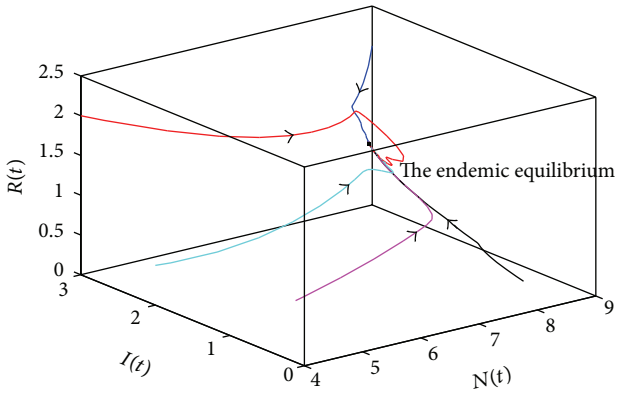

Figure 2: The endemic equilibrium E^* is locally asymptotically stable. Variation of infective population $I(t)$ and recovered population $R(t)$ with total population $N(t)$.

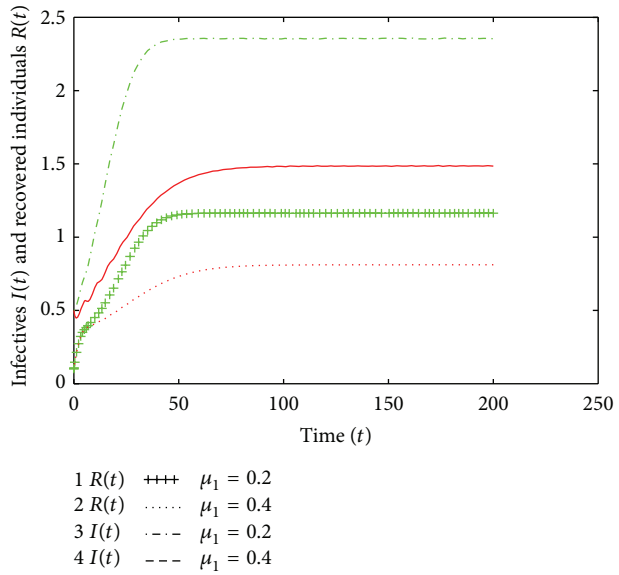

Figure 3: Variation of infective population $I(t)$ and recovered population $R(t)$ with time for different values of μ_1.

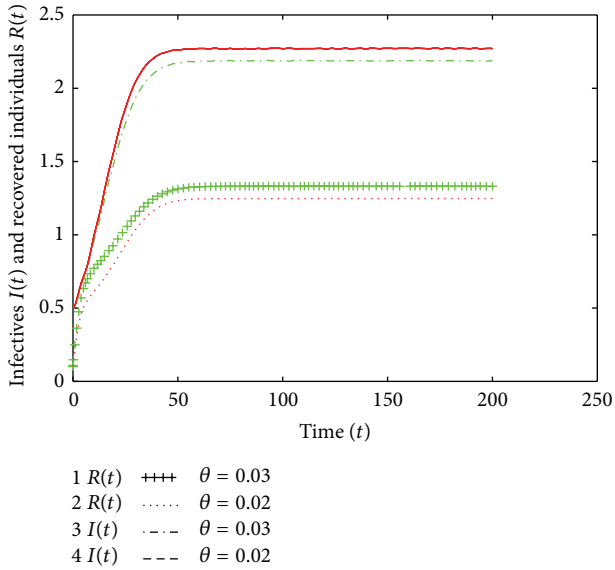

FIGURE 4: Variation of infective population $I(t)$ and recovered population $R(t)$ with time for different values of θ.

easy to verify that $R_0 = 1.83 > 1$ and all other conditions of Theorem 11 are satisfied. So, we can obtain from Figure 2 that the system (3) is stable at the endemic equilibrium point E^*.

(3) The results of numerical simulation are displayed graphically in Figures 3 and 4. In Figure 3, the variation of the infective population and recovered population is shown with time for different values of the removal rate constant from groups I to S, μ_1. It is found that both the infective population and the recovered population decrease as μ_1 increases. Figure 4 depicts the variation of infective population and recovered population, respectively, with time for the different successive vaccination rate, θ. As θ increases, the infective population decreases whereas the recovered population increases.

6. Discussion

In this paper, we will consider two different delays which are important parameters on the dynamic behavior. So, the present study is continuation of the previous work by [43]. Furthermore, from biological epidemic point of view, we investigate successive vaccination and difference in immunity in our system. From mathematical point of view, we study the stability of disease-free equilibrium and the existence of endemic equilibrium for different delay and consider the permanence of the system in the new paper.

In Theorems 9, 10, 11, and 13 corresponding to their corollaries, when the effect of the successive vaccination rate and the transfer rate from the infectious group to the susceptible group after treatment is strong, that is, $\theta > \theta^*$ and $\mu_1 > \mu_1^*$, the basic reproduction number R_0 being unity is a strict threshold for the control of the disease; the disease will be extinct or otherwise will tend to break out and persist. The other results are displayed graphically from our numerical simulation. We show the variation of the infective population and recovered population with time for different values of μ_1. It is found that both the infective population and the recovered population decrease as μ_1 increases. The infective population decreases whereas the recovered population increases as the successive vaccination rate increases, θ, respectively.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

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