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ANALYSIS OF AN SIRS EPIDEMIC MODEL WITH NONLINEAR INCIDENCE AND VACCINATION

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Abstract. In this paper, we study an SIRS epidemic model with a nonlinear incidence rate and vaccination. We give the existence of positivity, and boundedness of the equilibrium of the model. We calculate the basic reproduction number of the proposed model by using the next generation matrix method. By constructing Lyapunov function, we show that the disease-free equilibrium is globally asymptotically stable when the basic reproduction number is less or equal than one and that the endemic equilibrium is globally asymptotically stable when the basic reproduction number is greater than one. Numerical simulations are performed to investigate the effect of vaccinate on model behavior.

Keywords: SIRS epidemic model; nonlinear incidence; vaccination; Lyapunov function; global stability.

2010 AMS Subject Classification: 34D23, 92D30, 93D05.

1. INTRODUCTION

Infectious diseases have been enormous burden to the human society. Every outbreak of infectious diseases, such as smallpox, plague, cholera, and dengue fever, have caused many

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human deaths. In 2016, the world experienced a massive outbreak of dengue fever. According to the World Health Organization (WHO), more than 2.38 million dengue fever cases were reported in the American region, of which 1.5 million were Brazilian [1]. In 1906, Hamer [2] constructed and analyzed a discrete time model to help understand the causes of repeated outbreaks of measles. This is one of the first applications of mathematical models to infectious diseases. In 1927, Kermack and McKendrick [3] constructed a famous SIR model, in which the population is divided into susceptible (S), infective (I) and recovery (R). Mathematical models have been widely applied to the study of the transmission and control of infectious diseases since then. The beginning of the western industrial revolution in the late 18th century caused a large amount of labor to gather in cities. High human density and high-contact lifestyles caused infectious diseases to occur frequently. People have an immediate need for prevention and control of infectious diseases. Early prevention plays a key role in reducing the incidence of infectious diseases. Immunization by vaccination allows susceptible individuals to gain immunity without premature morbidity. For example, in the case of smallpox, people take the sputum that exposes the individual to the infected person or the skin lesion of the smallpox patient. Now with the rapid growth of biotechnology, more and more efficient vaccines have been developed by scientists to help humans overcome various infectious diseases. Immunization of newborns by vaccination can greatly reduce the incidence of many infectious diseases.

The common incidence of infectious diseases includes bilinear incidence and standard incidence, but sometimes other incidence is used for a specific infectious disease. To better describe the transmission mechanisms and control decisions of infectious diseases, Capasso and Serio [4] proposed a saturated nonlinear incidence $Sf(I)$ to explain the spread of cholera, where $f(I)$ represents the infection force from the infective. Later, Liu et al. [5, 6] proposed a nonlinear incidence rate kS^pI^q, where k is the transmission rate, p and q are positive constants. Wang et al. [7] proposed a nonlinear incidence rate $\varphi(P,L)$, where P and L represent the number of potential smokers and smokers. Yuan et al. [8] considered a generalized saturating incidence rate $\frac{k(I)S}{1+\alpha(I)S}$, where $k(I)$ and $\alpha(I)$ are nonnegative functions. These nonlinear incidence rates are applicable to different mathematical models and the results obtained are more consistent with the actual data.

For some infectious diseases, such as jaundice, chickenpox and mumps, infected individuals will get permanent immunity and will not get the same infectious diseases after rehabilitation. For some other infectious diseases, such as cholera, whooping cough and influenza, susceptible individuals will temporarily obtain antibody response after being infected, but return to the susceptible compartment immediately after the patient recover, or after a short period of time. In this paper, we present an SIRS model with vaccination immunity, in which susceptible individuals can obtain short-term immunity after vaccination but return to the susceptible compartment after a short time period. We use the method of Lyapunov-LaSalle invariant principle to analyze its global stability.

In the second part of the paper, we introduce the model formulation in detail. In the third part, we analyze some basic properties of the model, and in the fourth part, we analyze the global stability of the model. In the fifth part, we perform numerical simulations to discuss how the vaccinate affect the dynamic behavior. Finally, we summarize the article briefly.

2. MODEL FORMULATION

Li et al. [9] considered an SIRS mathematical model with a nonlinear incidence. The recovered person receives a brief immunization and then returns to the susceptible. Their model does not take into account the vaccination, which plays a critical role in the control of the disease [10]. Motivated by these, we consider the following mathematical model to study the effect of immunization on the infectious disease dynamics.

$$\begin{aligned} \frac{dS(t)}{dt} &= \lambda(1-p) - \mu S - Sf(I) + \gamma_1 I + \delta R, \\ \frac{dI(t)}{dt} &= Sf(I) - (\mu + \gamma_1 + \gamma_2 + \alpha)I, \\ \frac{dR(t)}{dt} &= p\lambda + \gamma_2 I - (\mu + \delta)R, \end{aligned} \tag{1}$$

with the initial conditions

$$S(0) = S_0 > 0,\ I(0) = I_0 \geq 0,\ R(0) = R_0 \geq 0. \tag{2}$$

In model (1), $S(t)$, $I(t)$, $R(t)$ represent the susceptible, infective and recovery individuals, respectively. Here $Sf(I)$ is a nonlinear infection incidence rate. The recruitment rate of susceptible individuals is λ. We assume that all recruitment are susceptible, of which a fraction p is vaccinated where $0 \leq p \leq 1$. μ is the natural mortality rate, γ_1 denotes the reversion rate from infected individuals to susceptible individuals, γ_2 denotes the transition rate from infected individuals to recovered individuals. α is the disease-induced death rate. δ is the immunity loss rate. Here we assume that λ, μ are positive while other parameters are nonnegative.

In our study, we need some hypotheses about f according to [11]. f is a real locally Lipschitz function on $\mathbb{R}_+ = [0, +\infty)$ satisfying

(A_1). $f(0) = 0$ and $f(I) > 0$ for $I > 0$;

(A_2). $\frac{f(I)}{I}$ is a continuous monotonic nonincreasing function for $I > 0$, and $\lim_{I \to 0^+} \frac{f(I)}{I}$ exists, denoted by β with $\beta > 0$.

According to the hypotheses about f, we can get that $f(I) \leq \beta I$.

Obviously, every solution of model (1) with the initial condition (2) keeps nonnegative for all $t > 0$. Next, we discuss the boundedness of model (1).

After adding the three equations of model (1), we get

$$\begin{aligned} \frac{d(S+I+R)}{dt} &= \lambda(1-p) - \mu S - Sf(I) + \gamma_1 I + \delta R + Sf(I) \\ &\quad -(\mu + \gamma_1 + \gamma_2 + \alpha)I + p\lambda + \gamma_2 I - (\mu + \delta)R \\ &= \lambda - \mu S - \mu I - \mu R - \alpha I \\ &\leq \lambda - \mu(S + I + R). \end{aligned}$$

According to the comparison principle, we have

$$\limsup_{t \to +\infty} (S(t) + I(t) + R(t)) \leq \frac{\lambda}{\mu}.$$

So we can define a bounded set $\Omega = \{(S(t),\, I(t),\, R(t)) \in \mathbb{R}^3_+ :\, S + R + I \leq \frac{\lambda}{\mu}\}$, which indicates that Ω is positive invariant with respect to model (1).

3. PRELIMINARIES

3.1. The basic reproduction number and existence of equilibrium. By straightforward calculation, we can obtain the disease-free equilibrium $E_0 = (S_0,\, I_0,\, V_0) = (\frac{\lambda(\mu(1-p)+\delta)}{\mu(\mu+\delta)},\, 0,\, \frac{p\lambda}{\mu+\delta})$. Using the method of Diekmann [12] and van den Driessche [13], we can derive the basic reproduction number

$$\mathscr{R}_0 = \rho(\mathbb{F}\mathbb{V}^{-1}) = \frac{\lambda\beta(\mu(1-p)+\delta)}{\mu(\mu+\delta)(\mu+\gamma_1+\gamma_2+\alpha)},$$

where

$$\mathbb{F} = \begin{pmatrix} \frac{\lambda\beta(\mu(1-p)+\delta)}{\mu(\mu+\delta)} & 0 \\ 0 & 0 \end{pmatrix}$$

and

$$\mathbb{V}^{-1} = \begin{pmatrix} \frac{1}{\mu+\gamma_1+\gamma_2+\alpha} & 0 \\ \frac{\gamma_2}{(\mu+\gamma_1+\gamma_2+\alpha)(\mu+\delta)} & \frac{1}{\mu+\delta} \end{pmatrix}.$$

$\mathscr{R}_0$ is usually referred to as the basic reproduction number, which can also be obtained by calculating the threshold for the existence of the endemic equilibrium point.

Next, we prove the existence and uniqueness of the endemic equilibrium $E^* = (S^*,\, I^*,\, R^*)$ of model (1). From model (1), we can get

$$\begin{aligned} S^* &= (\mu+\gamma_1+\gamma_2+\alpha)\frac{I^*}{f(I^*)}, \\ R^* &= \frac{p\lambda+\gamma_2 I^*}{\mu+\delta}, \\ h(I^*) &= \lambda - \mu S^* - \mu I^* - \mu R^* - \alpha I^*. \end{aligned} \tag{3}$$

After substituting S^* and R^* by their expressions in the third equation of (3). We obtain the following equation

$$h(I) = \lambda - \mu(\mu+\gamma_1+\gamma_2+\alpha)\frac{I}{f(I)} - \mu I - \mu\frac{p\lambda+\gamma_2 I}{\mu+\delta} - \alpha I.$$

Because $h(0^+) = \lambda - \frac{\mu(\mu+\gamma_1+\gamma_2+\alpha)}{\beta} - \frac{\mu p\lambda}{\mu+\delta} = \frac{\mu(\mu+\gamma_1+\gamma_2+\alpha)}{\beta}(\mathscr{R}_0 - 1) > 0$ when $\mathscr{R}_0 > 1$ and $h(\frac{\lambda}{\mu+\alpha}) = -\mu(\mu+\gamma_1+\gamma_2+\alpha)\frac{\lambda}{(\mu+\alpha)f(\frac{\lambda}{\mu+\alpha})} - \frac{\mu\lambda(p\mu+p\delta+\gamma_2)}{(\mu+\alpha)(\mu+\delta)} < 0$. Hence, there exists the positive zero for $h(I) = 0$. At the same time we can get that

$$h^{'}(I) = -\mu(\mu+\gamma_1+\gamma_2+\alpha)\frac{f(I) - If^{'}(I)}{f^2(I)} - \frac{\mu\gamma_2}{\mu+\delta} - \alpha - \mu.$$

Based on the assumption $(A2)$ about $f(I)$, we have $f(I)-If^{'}(I)>0$, which indicates $h^{'}(I)<0$. Hence the existence and uniqueness of the zero root of $h(I)=0$ is obtained.

4. STABILITY ANALYSIS

In order to simplify the calculation, we consider the following equivalent system of model (1).

$$\begin{aligned} \frac{dN}{dt} &= \lambda-\mu N-\alpha I, \\ \frac{dI}{dt} &= (N-I-R)f(I)-(\mu+\gamma_1+\gamma_2+\alpha)I, \\ \frac{dR}{dt} &= p\lambda+\gamma_2 I-(\mu+\delta)R, \end{aligned} \tag{4}$$

where $N(t)=S(t)+I(t)+R(t)$.

The disease-free equilibrium of model (4) is $\bar{E_0}=(\frac{\lambda}{\mu},\ 0,\ \frac{p\lambda}{\mu+\delta})$ and like model (1), there exists a unique endemic equilibrium $\bar{E}^*=(N^*,\ I^*,\ R^*)$ of model (4), where $N^*=\frac{\lambda-\alpha I^*}{\mu}$.

We first discuss the local stability of the disease-free equilibrium $\bar{E_0}$.

Theorem 1. *If $\mathscr{R}_0<1$, then the disease-free equilibrium $\bar{E_0}$ of model* (4) *is locally asymptotically stable in* Ω.

Proof. From the model (4) we can get the following Jacobian matrix

$$J_{\bar{E_0}}=\begin{pmatrix} -\mu & -\alpha & 0 \\ 0 & (\frac{\lambda}{\mu}-\frac{p\lambda}{\mu+\delta})\beta-(\mu+\gamma_1+\gamma_2+\alpha) & 0 \\ 0 & \gamma_2 & -(\mu+\delta) \end{pmatrix}.$$

The characteristic equation of the above Jacobian matrix at $\bar{E_0}$ is

$$(X+\mu)(X+(\mu+\delta))[X-(\frac{\lambda}{\mu}-\frac{p\lambda}{\mu+\delta})\beta+(\mu+\gamma_1+\gamma_2+\alpha)]=0.$$

One of the eigenvalues is $(\frac{\lambda}{\mu}-\frac{p\lambda}{\mu+\delta})\beta-(\mu+\gamma_1+\gamma_2+\alpha)=(\mu+\gamma_1+\gamma_2+\alpha)(\mathscr{R}_0-1)$, which is negative when $\mathscr{R}_0<1$. We can easily get that the other two eigenvalues are negative. Thus the disease-free equilibrium $\bar{E_0}$ is locally asymptotically stable in Ω when $\mathscr{R}_0<1$.

Theorem 2. *If $\mathscr{R}_0 < 1$, then the disease-free equilibrium $\bar{E}_0$ of model* (4) *is globally asymptotically stable in* Ω.

Proof. We define a Lyapunov function

$$V_0(t) = \frac{1}{2\alpha}(N - \frac{\lambda}{\mu})^2 + \int_0^I \frac{U}{f(U)}dU + \frac{1}{2\gamma_2}(R - \frac{p\lambda}{\mu+\delta})^2.$$

The time derivative of V_0 along solutions of system (4) is

$$\begin{aligned}
\frac{dV_0}{dt} &= \frac{1}{\alpha}(N - \frac{\lambda}{\mu})\frac{dN}{dt} + \frac{I}{f(I)}\frac{dI}{dt} + \frac{1}{\gamma_2}(R - \frac{p\lambda}{\mu+\delta})\frac{dR}{dt} \\
&= \frac{1}{\alpha}(N - \frac{\lambda}{\mu})(\lambda - \mu N - \alpha I) + \frac{I}{f(I)}((N - I - R)f(I) - (\mu + \gamma_1 + \gamma_2 + \alpha)I) \\
&\quad + \frac{1}{\gamma_2}(R - \frac{p\lambda}{\mu+\delta})(p\lambda + \gamma_2 I - (\mu+\delta)R) \\
&= \frac{1}{\alpha}(N - \frac{\lambda}{\mu})(-\mu(N - \frac{\lambda}{\mu}) - \alpha I) + I(N - I - R) \\
&\quad - \frac{I^2}{f(I)}(\mu + \gamma_1 + \gamma_2 + \alpha) + \frac{1}{\gamma_2}(R - \frac{p\lambda}{\mu+\delta})(-(\mu+\delta)(R - \frac{p\lambda}{\mu+\delta}) + \gamma_2 I) \\
&= -\frac{1}{\alpha}\mu(N - \frac{\lambda}{\mu})^2 - I^2 - \frac{\mu+\delta}{\gamma_2}(R - \frac{p\lambda}{\mu+\delta})^2 + (\frac{\lambda}{\mu} - \frac{p\lambda}{\mu+\delta} - \frac{I}{f(I)}(\mu + \gamma_1 + \gamma_2 + \alpha))I \\
&\leq -\frac{1}{\alpha}\mu(N - \frac{\lambda}{\mu})^2 - I^2 - \frac{\mu+\delta}{\gamma_2}(R - \frac{p\lambda}{\mu+\delta})^2 + (\frac{\lambda}{\mu} - \frac{p\lambda}{\mu+\delta} - \frac{1}{\beta}(\mu + \gamma_1 + \gamma_2 + \alpha))I \\
&= -\frac{1}{\alpha}\mu(N - \frac{\lambda}{\mu})^2 - I^2 - \frac{\mu+\delta}{\gamma_2}(R - \frac{p\lambda}{\mu+\delta})^2 + \frac{1}{\beta}(\mu + \gamma_1 + \gamma_2 + \alpha)(\mathscr{R}_0 - 1)I.
\end{aligned}$$

It is easy to have that $\frac{dV_0}{dt} < 0$ when $\mathscr{R}_0 < 1$. The largest invariant set of model (4) on the set in which $\frac{dV_0}{dt} = 0$ is the singleton $\{\bar{E}_0\} = \{(\frac{\lambda}{\mu}, 0, \frac{p\lambda}{\mu+\delta})\}$. It follows from the LaSalle's invariance principle (see [14, Theorem 5.3.1] or [15, Theorem 3.4.7]) that the disease-free equilibrium $\bar{E}_0$ is globally asymptotically stable on the feasible region Ω. □

Theorem 3. *If $\mathscr{R}_0 > 1$, then the endemic equilibrium $\bar{E}^*$ of model* (4) *is locally asymptotically stable in* Ω.

Proof. From model (4) we can get the following Jacobian matrix

$$J_{\bar{E}^*} = \begin{pmatrix} -\mu & -\alpha & 0 \\ f(I^*) & -B & -f(I^*) \\ 0 & \gamma_2 & -(\mu+\delta) \end{pmatrix}.$$

The characteristic equation of the above Jacobian matrix at $\bar{E}^*$ is

$$X^3 + CX^2 + DX + E = 0, \tag{5}$$

where,

$$B = f(I^*) - (N^* - I^* - R^*)f^{'}(I^*) + (\mu + \gamma_1 + \gamma_2 + \alpha),$$

$$C = 2\mu + \delta + B,$$

$$D = B(\mu+\delta) + \gamma_2 f(I^*) + B\mu + \mu(\mu+\delta) + \alpha f(I^*),$$

$$E = \mu(B(\mu+\delta) + \gamma_2 f(I^*)) + \alpha f(I^*)(\mu+\delta).$$

Plugging $\bar{E}^*$ into the second equation of model (4), we obtain

$$(\mu + \gamma_1 + \gamma_2 + \alpha) = (N^* - I^* - R^*)\frac{f(I^*)}{I^*}.$$

From the hypotheses about f, it is easy to know that $f^{'}(I^*) \leq \frac{f(I^*)}{I^*}$. Hence, we get

$$\begin{aligned} B &= f(I^*) - (N^* - I^* - R^*)f^{'}(I^*) + (\mu + \gamma_1 + \gamma_2 + \alpha) \\ &= f(I^*) + (N^* - I^* - R^*)(\frac{f(I^*)}{I^*} - f^{'}(I^*)) > 0. \end{aligned}$$

Thus, it is easily seen that $C, D, E > 0$ when $\mathscr{R}_0 > 1$. Further

$$\begin{aligned} CD - E = {} & \alpha B f(I^*) + (\mu + \delta + B)(B(\mu+\delta) + \gamma_2 f(I^*) + B\mu + \mu(\mu+\delta)) \\ & + \mu(B\mu + \mu(\mu+\delta) + \alpha f(I^*)) > 0. \end{aligned}$$

From Routh-Hurwitz criteria, it follows that all roots of (5) have negative real part if $\mathscr{R}_0 > 1$. Hence, the endemic equilibrium $\bar{E}^*$ of (4) is locally asymptotically stable in Ω.

Next, we prove the global stability of the endemic equilibrium $\bar{E}^*$. Model (4) has a unique positive equilibrium $\bar{E}^* = (N^*,\ I^*,\ R^*)$. Substituting the endemic equilibrium $\bar{E}^*$ into model

(4), we get

$$\begin{aligned} \lambda - \mu N^* - \alpha I^* &= 0, \\ (N^* - I^* - R^*) f(I^*) - (\mu + \gamma_1 + \gamma_2 + \alpha) I^* &= 0, \\ p\lambda + \gamma_2 I^* - (\mu + \delta) R^* &= 0. \end{aligned} \tag{6}$$

From the second equation of (6), we have

$$(N^* - I^* - R^*) - (\mu + \gamma_1 + \gamma_2 + \alpha) \frac{I^*}{f(I^*)} = 0.$$

Thus, the model (4) can be rewritten as

$$\begin{aligned} \frac{dN}{dt} &= -\mu(N - N^*) - \alpha(I - I^*), \\ \frac{dI}{dt} &= f(I)\{(N - N^*) - (I - I^*) - (R - R^*) - (\mu + \gamma_1 + \gamma_2 + \alpha)\left[\frac{I}{f(I)} - \frac{I^*}{f(I^*)}\right]\}, \\ \frac{dR}{dt} &= \gamma_2(I - I^*) - (\mu + \delta)(R - R^*). \end{aligned} \tag{7}$$

Theorem 4. *If $\mathscr{R}_0 > 1$, then the endemic equilibrium $\bar{E}^*$ of model* (4) *is globally asymptotically stable in* Ω.

Proof. We define a Lyapunov function

$$V_1(t) = \frac{1}{2\alpha}(N - N^*)^2 + \int_{I^*}^{I} \frac{U - I^*}{f(U)} dU + \frac{1}{2\gamma_2}(R - R^*)^2.$$

The time derivative of V_1 along solutions of system (7) is

$$\begin{aligned} \frac{dV_1}{dt} &= \frac{1}{\alpha}(N - N^*)\frac{dN}{dt} + \frac{I - I^*}{f(I)}\frac{dI}{dt} + \frac{1}{\gamma_2}(R - R^*)\frac{dR}{dt} \\ &= \frac{1}{\alpha}(N - N^*)[-\mu(N - N^*) - \alpha(I - I^*)] \\ &\quad + \frac{I - I^*}{f(I)} f(I)\{(N - N^*) - (I - I^*) - (R - R^*) - (\mu + \gamma_1 + \gamma_2 + \alpha)[\frac{I}{f(I)} - \frac{I^*}{f(I^*)}]\} \\ &\quad + \frac{1}{\gamma_2}(R - R^*)[\gamma_2(I - I^*) - (\mu + \delta)(R - R^*)] \end{aligned}$$

$$
\begin{aligned}
= & -\frac{\mu}{\alpha}(N-N^*)^2-(N-N^*)(I-I^*)+(N-N^*)(I-I^*)-(I-I^*)^2 \\
& -(I-I^*)(R-R^*)-(\mu+\gamma_1+\gamma_2+\alpha)(I-I^*)[\frac{I}{f(I)}-\frac{I^*}{f(I^*)}] \\
& +(I-I^*)(R-R^*)-\frac{\mu+\delta}{\gamma_2}(R-R^*)^2 \\
= & -\frac{\mu}{\alpha}(N-N^*)^2-(I-I^*)^2-\frac{\mu+\delta}{\gamma_2}(R-R^*)^2-(\mu+\gamma_1+\gamma_2+\alpha)(I-I^*)[\frac{I}{f(I)}-\frac{I^*}{f(I^*)}]
\end{aligned}
$$

Because $\frac{f(I)}{I}$ is a continuous monotonic nonincreasing function, we can obtain that $(\mu+\gamma_1+\gamma_2+\alpha)(I-I^*)[\frac{I}{f(I)}-\frac{I^*}{f(I^*)}]>0$. Therefore, it follows from the Lyapunov stability theorem that the endemic equilibrium $\bar{E^*}$ is globally asymptotically stable in Ω. □

5. Numerical Simulation

In this section, we use numerical simulations to verify the results obtained in sections 3 and 4. We consider the following system

$$
\begin{aligned}
\frac{dS(t)}{dt} &= \lambda(1-p)-\mu S-\frac{\beta SI}{1+\alpha_1 I}+\gamma_1 I+\delta R, \\
\frac{dI(t)}{dt} &= \frac{\beta SI}{1+\alpha_1 I}-(\mu+\gamma_1+\gamma_2+\alpha)I, \\
\frac{dR(t)}{dt} &= p\lambda+\gamma_2 I-(\mu+\delta)R.
\end{aligned} \tag{8}
$$

Obviously, model (8) is a particular case of model (1), which contains the saturate incidence $\frac{\beta SI}{1+\alpha_1 I}$. Besides, it is easy to see that the hypotheses (A_1) and (A_2) are verified. And also the basic reproduction number $\mathscr{R}_0=\frac{\lambda\beta(\mu(1-p)+\delta)}{\mu(\mu+\delta)(\mu+\gamma_1+\gamma_2+\alpha)}$. According to [10], we set $\lambda=10,\mu=0.04,\beta=0.02,\alpha=0.5,\alpha_1=0.3,\gamma_1=0.1,\gamma_2=0.85,\delta=0.005$.

Next, based on different p, we perform numerical simulations with model (8) by using Matlab. Firstly, we take $p=0.9$, then the basic reproduction number $\mathscr{R}_0=0.6711<1$. It follows from Theorem 2 that the disease-free equilibrium E_0 is globally asymptotically stable, which implies that the disease dies out eventually (see Figure 1). This is consistent with the conclusion of Figure 1. Secondly, we replace p with $p=0.5$. It follows that the basic reproduction number $\mathscr{R}_0=1.8043>1$. By Theorem 4, the endemic equilibrium E^* is globally asymptotically stable and the disease will persist (see Figure 2). If we choose the parameter value $p=0.1$, then $\mathscr{R}_0=3.0574>1$ and the endemic equilibrium E^* is globally asymptotically stable (see

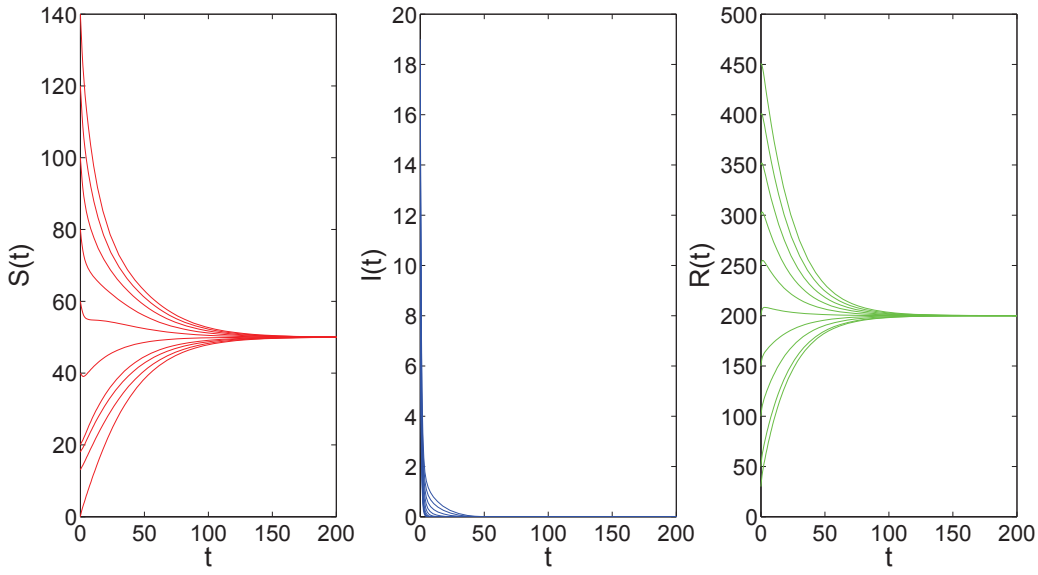

FIGURE 1. Taking $\lambda = 10, \mu = 0.04, \beta = 0.02, \alpha = 0.5, \alpha_1 = 0.3, \gamma_1 = 0.1, \gamma_2 = 0.85, \delta = 0.005, p = 0.9$, then $\mathscr{R}_0 = 0.6711 < 1$ and the disease-free equilibrium $E_0 = (50, 0, 200)$ of model (8) is globally asymptotically stable.

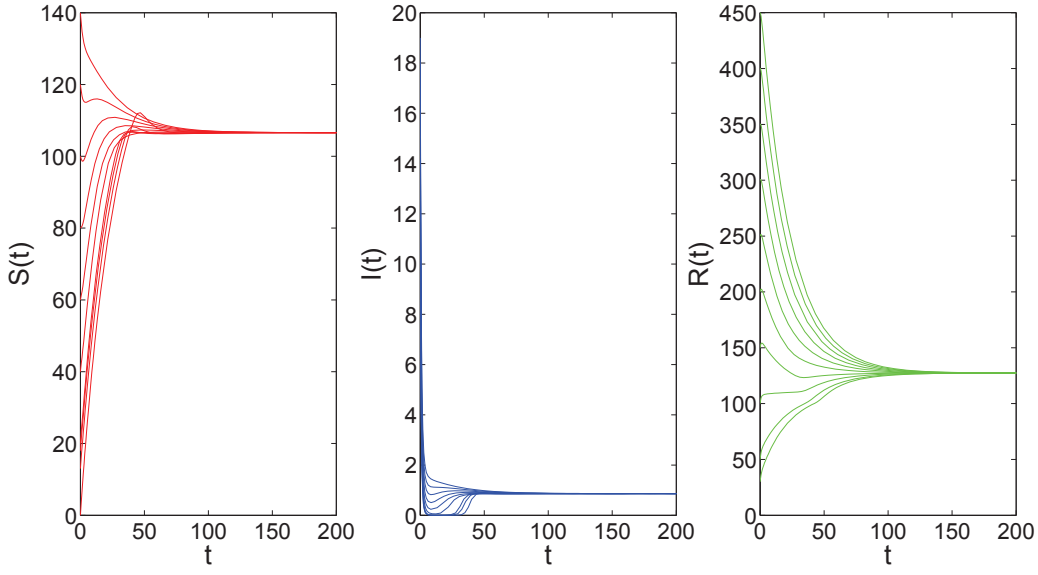

FIGURE 2. Taking $\lambda = 10, \mu = 0.04, \beta = 0.02, \alpha = 0.5, \alpha_1 = 0.3, \gamma_1 = 0.1, \gamma_2 = 0.85, \delta = 0.005, p = 0.5$, then $\mathscr{R}_0 = 1.8043 > 1$ and the endemic equilibrium $E^* = (106.5913, 0.8654, 127.3850)$ of model (8) is globally asymptotically stable.

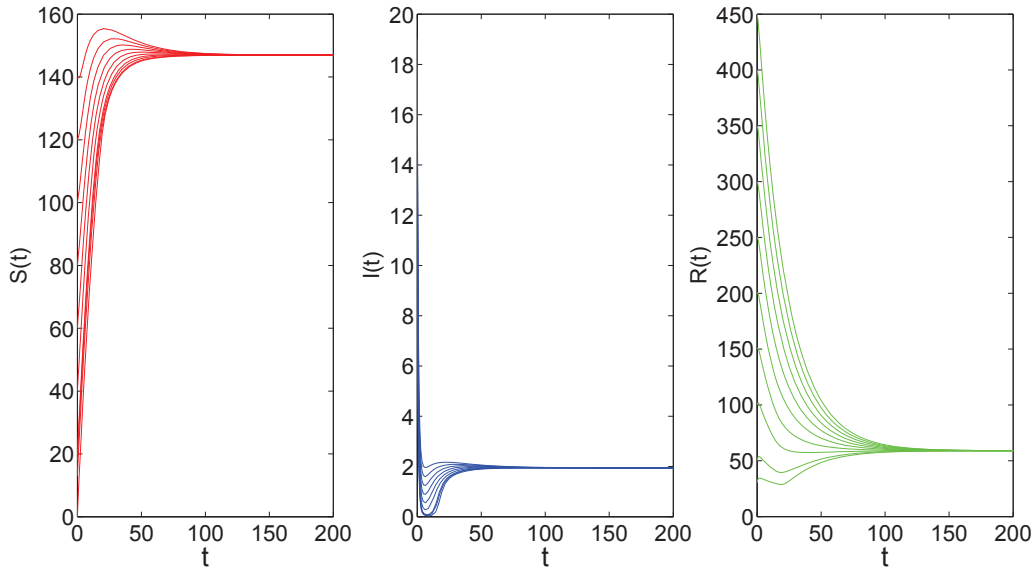

FIGURE 3. Taking $\lambda = 10, \mu = 0.04, \beta = 0.02, \alpha = 0.5, \alpha_1 = 0.3, \gamma_1 = 0.1, \gamma_2 = 0.85, \delta = 0.005, p = 0.1$, then $\mathscr{R}_0 = 3.0574$ and the endemic equilibrium $E^* = (146.9651, 1.9453, 58.9684)$ of model (8) is globally asymptotically stable.

Figure 3). According to our calculations, we can change the value of p to control the basic reproduction number $\mathscr{R}_0$ to measure whether the disease will persist. Compared with Figure 1, Figure 2 and Figure 3, we obtain a higher value of vaccination rate p, which can lead to the disease extinction. On the other hand, when the vaccination rate p is at a lower level, the disease will become popular.

6. DISCUSSION

In this paper, we introduce an SIRS epidemic model with a generalized nonlinear incidence rate and vaccination. In the model, a fraction of infected individuals resolve the infection and the recovered lose immunity in a short period and return to the susceptible compartment. We use the monotonicity of $f(I)$ to show that there is a unique positive equilibrium for our proposed model.

The basic reproduction number $\mathscr{R}_0$ is an important threshold in our model. By using the LaSalles invariance principle and the Lyapunov direct method, we proved that when $\mathscr{R}_0$ is less than one, the disease-free equilibrium is globally asymptotically stable and the infectious

disease eventually dies out over time. When $\mathscr{R}_0$ is greater than one, the endemic equilibrium is globally asymptotically stable and the disease becomes endemic. From the expression of the basic reproduction $\mathscr{R}_0$, we conclude that the vaccinated portion p can effectively reduce the number of infected to transmit the disease to susceptible individuals. Therefore, vaccination, when available, should be given to the maximum extent for the optimal control of infectious diseases in the scenario we discussed in this paper.

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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