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Research article

A generalized delay-induced SIRS epidemic model with relapse

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Abstract: In this paper, a generalized delay-induced $SIRS$ epidemic model with nonlinear incidence rate, latency and relapse is proposed. Our epidemic model is a generalized one, and the published epidemic models are the special cases of ours under some conditions. By using LaSalle's invariance principle and Lyapunovi's direct method, the dynamical behaviors are investigated and the results show that the disease free-equilibrium Q_0 is globally asymptotically stable if the basic reproduction number $R_0 < 1$ for any time delay. However, if the basic reproduction number $R_0 > 1$, there exists a unique endemic equilibrium Q_* which is locally asymptotically stable under some conditions. Moreover, the effects of latency and relapse on the transmission dynamics of the diseases are analyzed by some numerical experiments which conducted based on $ODE45$ in Matlab.

Keywords: a delay-induced model; generalized nonlinear incidence; dynamical behavior; numerical experiment
Mathematics Subject Classification: 37C75, 92B05, 92D25, 93D20

1. Introduction

Mathematical models are widely used to expound the transmission dynamics of some infectious disease, these mathematical models of the epidemic diseases paly a great role in presenting the prevent and control measures [1]. Most epidemic models are described by the ordinary differential equations [2–6]. For those models, populations are divided into the different classes which have corresponding attributes. the classic SIR epidemic model was proposed by Kermack and McKendrick [7], which was separated the population into three compartments named as the susceptible compartment $S(t)$, the infective compartment $I(t)$ and the recovered compartment $R(t)$. Unlike the SIR model, the $SIRS$ model shows the effect of the impermanent immunity of the removed individuals. That is to say, the removed individuals will become susceptible again after a

period [8]. A famous example is the seasonal influenza which can be immunized for several years due to the high mutation of the virus [9]. Many researchers have investigated $SIRS$ models to reveal the transmission dynamics of the diseases. Chen and Sun [10] studied an $SIRS$ epidemic model with vaccination on the heterogeneous networks and gave some theoretical methods. They obtained the sufficient conditions for the global stability of the disease-free equilibrium and the endemic equilibrium by constructing suitable Lyapunov functions. An $SIRS$ epidemic model with the graded cure and the recovery rate had been investigated by Muroya [11]. By using two kinds of the Lyapunov functionals, one was a quadratic type and the other was a Volterra type, they established complete global dynamics of system. Hao et al. [12] presented an $SIRS$ epidemic model with the birth pulse, the pulse vaccination and the saturation incidence, and the flip bifurcation of the endemic periodic solution was discussed according to the Poincaré map and center manifold theorem. Qi et al. [13] investigated the stability of an $SEIRS$ model with the vertical transmission, the nonlinear incidence, and the time delay which means the immunity period. They established the conclusion that the immunity period and the vertical transmission can significantly influence the dynamical behaviors of the $SEIRS$ system. Therefore, the incidence rate plays an important role in the transmission dynamics of the epidemic diseases.

In fact, the incidence rate defines as the newly infected individuals which are infected by the infectious individuals per unit of time. Its general form is written as $\beta_0 U(N)\frac{S}{N}I$, where $U(N)$ is the contact rate related to the total population size N, β_0 is the probability of contact to a transmitted disease per unit of time, $\beta_0 U(N)$ is called as the effective contact rate [14]. The published researches show that there are usually two types of effective contact rates. One is a kN and the other is a constant k. If the number of the total population is not very large, it would be more appropriate to adopt the contact rate kN, and hence the incidence rate is bilinear form βSI [15]. Moreover, if the number of the total population is relatively large, the contact between the infected individuals and the susceptible individuals should be limited, It would be more realistic to adopt the contact rate k, and hence the corresponding incidence rate is the standard rate $\beta\frac{S}{N}I$ [13]. In fact, those both contact rates are based on the principle of the mass action and the assumption of the homogeneously mixing of populations. These rules are better suited to the infectious diseases such as influenza, tuberculosis, etc. However, for some sexually transmitted diseases or age-structured infectious diseases, this assumption is unreasonable. Furthermore, the spread of diseases is a complex process, and the traditional linear incidence may need to be further modified due to factors such as population structure and heterogeneous mixing [5, 13–16]. These factors have prompted scholars to consider the nonlinear incidence rate. In 1978, Capasso and Serio [16] proposed a saturated incidence rate, namely $\frac{kI}{1+\alpha I}$. A nonlinear incidence rate also arises from saturation effects. If the proportion of infected ones in the total population is increasing, they will be death due to the disease. Hence the spread of the disease will be slower compared with the linear growth. Since the saturated incidence is presented by Capasso and Serio, many scholars have considered various nonlinear forms of the incidence rates. For example, there exists the incidence forming $\beta I^p S^q$ (where p and q are positive constants) in model [17]. Epidemic models with this incidence rate have been studied by Liu et al. [18] and later by Hethcote et al. [19] and many others. More complex forms of the nonlinear incidence were also considered. For instance, Briggs and Godfray [20] studied infection of insects, considered a non-linear pathogen transmission of the form $kS\ln(1+\frac{\upsilon P}{k})$ (where P is the density of the pathogen particles). Ruan and Wang [21] investigated an epidemic model with a nonlinear incidence rate $\frac{kI^2S}{1+\alpha I^2}$.

Liu et al. [17] studied the general incidence rate $\frac{kI^pS}{1+\alpha I^q}$. Moreover, a model with a generalized nonlinear incidence $\beta g(I)S$ was studied by Hethcote [22]. Korobeinikov and Maini [23] considered the incidence of the form $f(S)g(I)$ in various types of epidemic models. In addition, a nonlinear incidence $f(S, I)$ was also investigated in many literatures [24–27]. These generalized incidence rates must satisfy some strict conditions£ and make these incidences limit in applications. Therefore, a more generalized incidence with the fewer limitations and the more widely applications should be presented, and the correspondingly epidemic models should be investigated.

Moreover, once individuals are infected by some certain diseases, they have to go through a certain incubation period before they can infect other susceptible individuals. Hence, there exists a infected delay. Some epidemic models incorporate the effect of the time delay have been investigated. Sekiguchi and Ishiwata [28] proposed a discretized $SIRS$ epidemic model with a distributed time delay and obtained some sufficient conditions for the global dynamics of the solution. Zhang et al. [29] investigated the global stability of an SIR epidemic model with a constant infectious period. The persistence of SIR epidemic model with the distributed delay was considered by Ma et al. [30]. They obtained the results that the SIR epidemic model was permanent for any time delay if and only if the endemic equilibrium existed. In addition, for some diseases such as herpes and human tuberculosis [31,32], it is showed that the recovering individuals will be infected again and become to the infected individuals. Many $SIRI$ models considered the relapses of some certain diseases [33–35]. The epidemic model with the nonlinear incidence and the relapse was studied and the two types of controlling methods were conducted to reduce the number of infectious. The controlling functions were incorporated to the cure and the relapse terms, and the optimal control theory was applied to reveal the model function. Hence, it is obvious that the delay effect plays an important roles in determining the dynamical behaviors of the epidemic models and the transmission mechanisms of the epidemic diseases. Therefore, the presentation of a epidemic models with the effect of the time delay is more necessary, interesting and realistic.

Motivated by these, this paper proposes a generalized incidence rate and incorporates the generalized incidence function and the latent delay into a $SIRS$ epidemic model. This paper is organized as follows. In Section 2, we give the description of the $SIRS$ model and discuss the existence of equilibrium points based on the basic reproduction number R_0. In Section 3, the global stability of disease-free equilibrium and the local stability of endemic equilibrium without the latent period (i.e., $\tau = 0$) are considered. Furthermore, the dynamic behaviors of the model with $\tau > 0$ is obtained in Section 4. The numerical experiments are conducted in Section 5 to test and verify our theoretical results. Finally, some conclusions are drawn in Section 6.

2. Model description and preliminaries

In this paper, we propose the following $SIRS$ model with a generalized incidence rate and relapse. According to the research published by Ma et al. [3], the generalized incidence rate could be proposed based on the following assumptions:

($A1$) The infectious probability of the infectious subpopulation $P(I)$ which can efficaciously make each susceptible individual to be infected is assumed as a function of the infectious individuals and expressed as $P(I) = KT(I)\varphi(I)$, where K is the intrinsically infectious rate, $T(I)$ is the available infectious time of the infectious subpopulation, the function $\varphi(I)$ means the total number of the

infectious individuals which contribute to the spread of the infectious diseases, and is mathematically assumed a continuously differentiable function at the interval $[0, +\infty]$.

$(A2)$ The available infectious time $T(I)$ is assumed as the minus of the average life-span T and the search time of the infectious individuals $h(I)P(I)$, where $h(I)$ is the search time of each infectious individuals. The search time $h(I)P(I)$ means the total time being spent by the infectious subpopulation for the purpose of survival which being carrying the infectious virus and being spreading over the infectious diseases. Hence, the available infectious time $T(I)$ is expressed as $T - h(I)P(I)$. This assumption means that the infectious subpopulation must spend some times to physically survive, rather than transmit the epidemic diseases in their life-span.

According to the above assumptions, the infectious probability of the infectious subpopulation for each susceptible individuals $P(I)$ could be obtained by solving the following equations

$$\begin{cases} P(I) = KT(I)\varphi(I), \\ T(I) = T - h(I)P(I). \end{cases}$$

Solving the above equations, it is obtained that

$$P(I) = \frac{KT\varphi(I)}{1 + Kh(I)\varphi(I)}.$$

Hence, the infectious probability which make each susceptible individuals to be effectively infected per unit time is expressed as the following form

$$\frac{P(I)}{T} = \frac{K\varphi(I)}{1 + Kh(I)\varphi(I)}.$$

Setting $Kh(I)\varphi(I) = \psi(I)$, thus the incidence function should be proposed as

$$\frac{KS\varphi(I)}{1 + \psi(I)}.$$

The above incidence function satisfies the classic assumptions for the incidence rate [3]. That is to say, the above nonlinear incidence function is proposed based on the two classic assumptions. One is that the number of effective contacts between the infective and susceptible individuals may be saturated at high infective levels due to the crowding effect of the infective individuals or due to the self-protection effect of the susceptible individuals. The other is that the number of effective contacts is decreasing and the inhibition effect is weakened when the number of infective individuals is large enough.

Furthermore, according to the natural factors, it is assumed that $\varphi(I)$ and $\psi(I)$ are positive and monotonically increasing functions for all $I > 0$, $\varphi(0) = \psi(0) = 0$. Clearly, our incidence function is more generalized and and some published incidence rates will become special cases of ours under some assumptions. If $\psi(I) = 0$, $\varphi(I) = I$, the incidence is classic bilinear $\beta S I$ [15]; When we take $\psi(I) = \alpha I$, $\varphi(I) = kI$, the incidence rate $\frac{kI}{1+\alpha I}$ was studied by Capasso and Serio [16]; If $\psi(I) = I^p$, $\varphi(I) = \alpha I^q$, the incidence rate $\beta I^p S^q$ was mentioned in references [17–19]; If $\psi(I) = 0$, $\varphi(I) = \ln(1 + \frac{\upsilon P}{k})$ the incidence rate $kS \ln(1 + \frac{\upsilon P}{k})$ was considered in reference [20]; The case of $\psi(I) = 0$ was studied in reference [22].

In this paper, the function $\varphi(I)$ also satisfies the following assumptions

$$\varphi'(I) > 0, \ \ \psi'(I) > 0, \ \ \forall I > 0,$$

and

$$0 < \varphi(I) \leq \varphi'(0)I, \ \ \forall I > 0.$$

Considering all the previous assumptions, our model is proposed as following form

$$\begin{cases} \frac{dS}{dt} = b - dS - \frac{KS\varphi(I(t-\tau))}{1+\psi(I(t-\tau))} + \gamma R, \\ \frac{dI}{dt} = \frac{KS\varphi(I(t-\tau))}{1+\psi(I(t-\tau))} - (d+\mu)I + \sigma R, \\ \frac{dR}{dt} = \mu I - (d+\sigma+\gamma)R, \end{cases} \tag{2.1}$$

where b, d, K, γ, μ, τ, σ are positive constant number and the epidemiological meanings of all parameters are given in Table 1.

Table 1. Definition of the parameters of model (2.1).

Parameter	Definitions
b	The birth rate of the population in all compartment
d	The natural death rate of the population in all compartment
K	The effective contact rate between the susceptible individuals and the infectious individuals
γ	The transmission rate from the recovered compartment to the susceptible compartment
μ	The transmission rate from the infectious compartment to the removed compartment
τ	The latent period of the epidemic disease
σ	The recover rate from the infectious compartment to the removed compartment

Molde (2.1) are more realistic and the latent period which is incorporated in model (2.1) generally exists for many epidemic diseases, such as AIDS/HIV who's latent period could be more than ten years, COVID-19 who's latent period is about 7–14 days. The span of the latent period for the epidemic diseases is very important in presenting the controlling methods.

Add the three equations of model (2.1) and denoting $N(t) = S(t) + I(t) + R(t)$, we have

$$\frac{dN(t)}{dt} = b - dN(t).$$

Then

$$N(t) = \frac{1}{d}(b - (b - dN(t_0))e^{-d(t-t_0)}), \lim_{t\to\infty} N(t) = \frac{b}{d}. \tag{2.2}$$

Hence, the following limit equations of system (2.1) is obtained

$$\begin{cases} \frac{dI}{dt} = \frac{K\varphi(I(t-\tau))}{1+\psi(I(t-\tau))}(\frac{b}{d} - I - R) - (d+\mu)I + \sigma R, \\ \frac{dR}{dt} = \mu I - (d+\sigma+\gamma)R. \end{cases} \tag{2.3}$$

We will obtain the dynamical behavior of system (2.1) by studying the stability of the equilibrium of system (2.3). By a biological meaning, we further assume that the initial condition is given such that

$$S(0) > 0, I(0) > 0, R(0) > 0.$$

The epidemic models usually have a threshold parameter, known as the basic reproduction number, R_0. It is the number of secondary cases which one case would produce in a completely susceptible

population, and it can be obtained as dominant eigenvalue of the next generation matrix [36]. The reproduction number R_0 of model (2.3) is defined by the theory of the next generation matrix [36, 37]

$$R_0 = \rho((DF\mid_{Q_0}) \bullet (DV\mid_{Q_0})^{-1}),$$

in which F is the regeneration matrix matrix and V is the transition matrix of model (2.3).

$$F = \begin{pmatrix} \frac{K\varphi(I(t-\tau))}{1+\psi(I(t-\tau))}(\frac{b}{d} - I - R) + \sigma R \\ 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} (d+\mu)I \\ -\mu I + (d+\sigma+\gamma)R \end{pmatrix}.$$

Hence, it is obtained that

$$DF\mid_{Q_0} = \begin{pmatrix} \frac{kb\varphi'(0)}{d} & \sigma \\ 0 & 0 \end{pmatrix},$$

and

$$DV\mid_{Q_0} = \begin{pmatrix} d+\mu & 0 \\ -\mu & d+\sigma+\gamma \end{pmatrix}.$$

Thus, we have

$$(DV\mid_{Q_0})^{-1} = \frac{1}{(d+\sigma+\gamma)(d+\mu)} \begin{pmatrix} d+\sigma+\gamma & 0 \\ \mu & (d+\mu) \end{pmatrix}.$$

Therefore, the basic reproduction number is

$$R_0 = \rho((DF\mid_{Q_0}) \bullet (DV\mid_{Q_0})^{-1}) = \frac{Kb\varphi'(0)}{d(d+\mu)} + \frac{\sigma\mu}{(d+\mu)(d+\gamma+\sigma)}.. \tag{2.4}$$

Hence, the following result on the existence of the equilibrium for system (2.3) are obtained.

The positivity and boundness of solutions for system (2.3) could be found in Proposition 1 in Appendix. Furthermore, system (2.3) has two equilibrium points: the disease-free equilibrium $Q_0 = (0, 0)$ and the unique endemic equilibrium $Q_* = (I_*, R_*)$. The proof of the existence of the equilibrium points could also be found in Appendix.

3. Stability analysis of system (2.3) with $\tau = 0$

In this section, we analyze the stability of the two equilibria of system (2.3) with $\tau = 0$.

Theorem 3.1. The disease-free equilibrium Q_0 of system (2.3) is globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. We first demonstrate that the disease-free equilibrium Q_0 of system (2.3) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

The Jacobian matrix of system (2.3) at Q_0 is

$$M_0 = \begin{pmatrix} \frac{Kb\varphi'(0)}{d} - (d+\mu) & \sigma \\ \mu & -(d+\sigma+\gamma) \end{pmatrix}.$$

We compute the determinant and trace of M_0 as following

$$Det(M_0) = -(d+\mu)(d+\sigma+\gamma)(R_0-1),$$

$$Tr(M_0) = \frac{Kb\varphi'(0)}{d} - (d+\mu) - (d+\sigma+\gamma).$$

Hence, the $Det(M_0) > 0$ if and only if $R_0 < 1$. The $Tr(M_0) < 0$ if and only if $\frac{Kb\varphi'(0)}{d} < (d+\mu) + (d+\sigma+\gamma)$.

Since $\frac{Kb\varphi'(0)}{d} < (d+\mu) - \frac{\sigma\mu}{d+\sigma+\gamma}$ when $R_0 < 1$, then one can see that $Tr(M_0) < 0$ if $R_0 < 1$. Thus, the disease-free equilibrium Q_0 of system (2.3) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

To verify the global stability of the disease-free equilibrium Q_0, we take the Lyapunov function

$$V(t) = I(t) + \frac{\sigma}{d+\sigma+\gamma}R(t).$$

The derivative of the function $V(t)$ along solutions of system (2.3) is

$$\begin{aligned} V'(t) &= I'(t) + \tfrac{\sigma}{d+\sigma+\gamma}R'(t) \\ &= \tfrac{K(\frac{b}{d}-I-R)\varphi(I)}{1+\psi(I)} - (d+\mu)I + \tfrac{\sigma\mu}{d+\sigma+\gamma}I. \end{aligned}$$

Since $1+\psi(I) > 1$, $\varphi(I) \le \varphi'(0)I$ and $\frac{b}{d} - I - R \le \frac{b}{d}$, we have

$$\begin{aligned} V'(t) &\le \tfrac{Kb}{d}\varphi'(0)I - (d+\mu)I + \tfrac{\sigma\mu}{d+\sigma+\gamma}I \\ &= (d+\mu)(\tfrac{Kb\varphi'(0)}{d(d+\mu)} + \tfrac{\sigma\mu}{(d+\mu)(d+\gamma+\sigma)} - 1)I \\ &= (d+\mu)(R_0-1) \\ &\le 0. \end{aligned}$$

Since $R_0 < 1$, $V'(t) = 0$ holds only when $I(t) = 0$. It follows from system (2.3) that $I(t) \to 0$ and $R(t) \to 0$, and then Q_0 is the largest invariant subset in the set where $V'(t) = 0$. Hence, the disease-free equilibrium Q_0 of system (2.3) is globally asymptotically stable if $R_0 < 1$.
This completes the proof.

Now, we turn to consider the stability analysis of the unique endemic equilibrium Q_*.

Theorem 3.2. If $R_0 > 1$ and $\frac{K(\frac{b}{d}-I_*-R_*)\varphi'(I_*)}{I_*\psi'(I_*)} < d + \frac{\mu(d+\gamma)}{d+\sigma+\gamma}$, the endemic equilibrium Q_* of system (2.3) is locally asymptotically stable.

Proof. The Jacobian matrix of system (2.3) at Q_* is

$$M_* = \begin{pmatrix} \frac{K(\varphi'(I_*)(1+\psi(I_*))-\varphi(I_*)\psi'(I_*))}{(1+\psi(I_*))^2}(\frac{b}{d} - I_* - R_*) - \frac{K\varphi(I_*)}{1+\psi(I_*)} - (d+\mu) & -\frac{K\varphi(I_*)}{1+\psi(I_*)} + \sigma \\ \mu & -(d+\sigma+\gamma) \end{pmatrix}.$$

We compute the determinant and trace of M_* as following

$$Det(M_*) = -\frac{K(\varphi'(I_*)(1+\psi(I_*))-\varphi(I_*)\psi'(I_*))(d+\sigma+\gamma)}{(1+\psi(I_*))^2} + \frac{K\varphi(I_*)(d+\sigma+\gamma)}{1+\psi(I_*)} + \frac{K\varphi(I_*)\mu}{1+\psi(I_*)} + (d+\mu)(d+\gamma) + d\sigma,$$

$$Tr(M_*) = \frac{K(\varphi'(I_*)(1+\psi(I_*)) - \varphi(I_*)\psi'(I_*))(\frac{b}{d} - I_* - R_*)}{(1+\psi(I_*))^2} - \frac{K\varphi(I_*)}{1+\psi(I_*)} - (d+\mu) - (d+\sigma+\gamma).$$

Hence, the $Det(M_*) > 0$ and $Tr(M_*) < 0$ if

$$C = [(1+\psi(I_*))\varphi'(I_*) - \psi'(I_*)\varphi(I_*)] < 0, \tag{3.1}$$

that is

$$\frac{K(\frac{b}{d} - I_* - R_*)\varphi'(I_*)}{I_*\psi'(I_*)} < d + \frac{\mu(d+\gamma)}{d+\sigma+\gamma}. \tag{3.2}$$

Therefore, if $R_0 > 1$ and $\frac{K(\frac{b}{d}-I_*-R_*)\varphi'(I_*)}{I_*\psi'(I_*)} < d + \frac{\mu(d+\gamma)}{d+\sigma+\gamma}$, the endemic equilibrium Q_* of system (2.3) is locally asymptotically stable. This completes the proof.

4. Stability analysis of system (2.3) with $\tau > 0$

In this section, we will turn to consider the stability of the two equilibria of system (2.3) with $\tau > 0$.

Theorem 4.1. If $R_0 < 1$, the disease-free equilibria Q_0 of system (2.3) is globally asymptotically stable.

Proof. We firstly give the local stability of Q_0. Linearize system (2.3) at Q_0 and obtain the characteristic equation

$$Det(\lambda E - A_0 - e^{-\lambda\tau}B_0) = 0,$$

where E is the unit matrix and

$$A_0 = \begin{pmatrix} -(d+\mu) & \sigma \\ \mu & -(d+\sigma+\gamma) \end{pmatrix},$$

$$B_0 = \begin{pmatrix} \frac{Kb\varphi'(0)}{d} & 0 \\ 0 & 0 \end{pmatrix}.$$

Then we have

$$\lambda^2 + (d+\sigma+\gamma+(d+\mu))\lambda + (d+\mu)(d+\sigma+\gamma) - \sigma\mu = \frac{Kb\varphi'(0)}{d}e^{-\lambda\tau}(\lambda + (d+\sigma+\gamma)). \tag{4.1}$$

From Theorem 3.1, the real parts of all roots of Eq (4.1) are negative if $R_0 < 1$ and $\tau = 0$. We assume that $\lambda = i\omega$ is a purely imaginary root of the Eq (4.1), where ω is a positive real number, then by separating the real and imaginary parts, we get

$$\begin{cases} -\omega^2 + (d+\mu)(d+\sigma+\gamma) - \sigma\mu = \frac{Kb\varphi'(0)}{d}((d+\sigma+\gamma)\cos\omega\tau + \omega\sin\omega\tau), \\ (d+\sigma+\gamma+(d+\mu))\omega = \frac{Kb\varphi'(0)}{d}(\omega\cos\omega\tau - (d+\sigma+\gamma)\sin\omega\tau). \end{cases}$$

By a simple calculation, we have

$$\omega^4 + a_1\omega^2 + a_2 = 0, \tag{4.2}$$

where

$$a_1 = (d+\sigma+\gamma)^2 + 2\sigma\mu + (d+\mu+\frac{Kb\varphi'(0)}{d})(d+\mu-\frac{Kb\varphi'(0)}{d}),$$

$$a_2 = ((d+\mu)(d+\sigma+\gamma-\sigma\mu))^2 - (\frac{Kb\varphi'(0)}{d}(d+\sigma+\gamma))^2.$$

If $R_0 < 1$, then $a_1 > 0$, $a_2 > 0$. then the Eq (4.2) does not have positive roots. Therefore, the characteristic Eq (4.1) does not have purely imaginary roots, and all roots of the Eq (4.1) have negative real parts for any $\tau > 0$ if $R_0 < 1$. Thus, Q_0 is locally asymptotically stable if $R_0 < 1$ for any $\tau > 0$.

Next, we show that the Q_0 is globally asymptotically stable if $R_0 < 1$. Construct the Lyapunov functional

$$V(I(t), R(t)) = I(t) + \frac{\sigma}{d+\sigma+\gamma}R(t) + \frac{Kb}{d}\int_{t-\tau}^{t}\frac{\varphi(u)}{1+\psi(u)}du.$$

Clearly, $V(0,0) = 0$ and $V(I(t), R(t)) > 0$ in the interior of R^2_+.

Since $R_0 < 1$, we have

$$\begin{aligned}
\tfrac{dV(I(t),R(t))}{dt}\,|_{(4.1)} &= I'(t) + \tfrac{\sigma}{d+\sigma+\gamma}R'(t) + \tfrac{Kb\varphi(I)}{d(1+\psi(I))} - \tfrac{Kb}{d}\tfrac{\varphi(I(t-\tau))}{1+\psi(I(t-\tau))} \\
&= -\tfrac{K\varphi(I(t-\tau))}{1+\psi(I(t-\tau))}(I+R) - (d+\mu)I + \tfrac{\sigma\mu}{d+\sigma+\gamma}I + \tfrac{Kb}{d}\varphi(I) - \tfrac{Kb}{d}\varphi(I) + \tfrac{Kb\varphi(I)}{d(1+\psi(I))} \\
&\le -\tfrac{K\varphi(I(t-\tau))}{1+\psi(I(t-\tau))}(I+R) + [\tfrac{Kb}{d}\varphi'(0) + \tfrac{\sigma\mu}{d+\sigma+\gamma} - (d+\mu)]I - \tfrac{Kb\varphi(I)\psi(I)}{d(1+\psi(I))} \\
&= -\tfrac{K\varphi(I(t-\tau))}{1+\psi(I(t-\tau))}(I+R) - \tfrac{Kb\varphi(I)\psi(I)}{d(1+\psi(I))} + (d+\mu)(R_0-1) \\
&< 0.
\end{aligned}$$

Furthermore, the set

$$\{(I(t), R(t)) | \frac{dV(I(t), R(t))}{dt}\,|_{(4.1)} = 0, \forall t \ge 0\}$$

has a unique point Q_0. It follows from the Lyapunov-Lasalle invariant principle that the Q_0 is globally asymptotically stable. *This completes the proof.*

Now, we turn to the study of the stability of the endemic equilibrium Q_* of system (2.3) with $\tau > 0$. To do this, we calculate the linearization of system (2.3) at Q_* and obtain the characteristic equation

$$Det(\lambda E - A_* - e^{-\lambda\tau}B_*) = 0,$$

where E is the unit matrix and

$$A_* = \begin{pmatrix} -(d+\mu) & \sigma \\ \mu & -(d+\sigma+\gamma) \end{pmatrix},$$

$$B_* = \begin{pmatrix} \frac{KC}{(1+\psi(I_*))^2}(\frac{b}{d} - I_* - R_*) - \frac{K\varphi(I_*)}{1+\psi(I_*)} & -\frac{K\varphi(I_*)}{1+\psi(I_*)} \\ 0 & 0 \end{pmatrix}.$$

Then the characteristic equation becomes

$$\begin{aligned}
&\lambda^2 + (d+\sigma+\gamma+(d+\mu))\lambda + (d+\mu)(d+\sigma+\gamma) - \sigma\mu \\
&= [\tfrac{KC}{(1+\psi(I_*))^2}(\tfrac{b}{d} - I_* - R_*)(\lambda + (d+\sigma+\gamma)) - \tfrac{K\mu\varphi(I_*)}{1+\psi(I_*)} - \\
&\quad \tfrac{K\varphi(I_*)}{1+\psi(I_*)}(\lambda + (d+\sigma+\gamma))]e^{-\lambda\tau}.
\end{aligned} \tag{4.3}$$

From Theorem 3.2, the real parts of all roots of Eq (4.3) are negative if $R_0 > 1$ and $\tau = 0$. We assume that $\lambda = i\omega$ is a purely imaginary root of the Eq (4.3), where ω is a positive real number, then by separating the real and imaginary parts, we get

$$\begin{cases} -\omega^2 + (d+\mu)(d+\sigma+\gamma) - \sigma\mu = D_1 \cos\omega\tau + D_2 \sin\omega\tau, \\ \omega(d+\sigma+\gamma+(d+\mu)) = -D_1 \sin\omega\tau + D_2 \cos\omega\tau, \end{cases}$$

where

$$D_1 = \frac{KC(\frac{b}{d} - I_* - R_*)(d+\sigma+\gamma)}{(1+\psi(I_*))^2} - \frac{K\varphi(I_*)(\mu + (d+\sigma+\gamma)}{1+\psi(I_*)}),$$

$$D_2 = \frac{KC\omega(\frac{b}{d} - I_* - R_*)}{(1+\psi(I_*))^2} - \frac{K\omega\varphi(I)}{1+\psi(I)}.$$

By a simple computation, we have

$$\omega^4 + a_1\omega^2 + a_2 = 0, \tag{4.4}$$

where

$$a_1 = (d+\sigma+\gamma)^2 + (d+\mu)^2 + 2\sigma\mu - (\frac{KC(\frac{b}{d} - I_* - R_*)}{(1+\psi(I_*))^2} - \frac{K\varphi(I_*)}{1+\psi(I_*)})^2,$$

$$a_2 = ((d+\mu)(d+\sigma+\gamma) - \sigma\mu)^2 - (\tfrac{KC(\frac{b}{d}-I_*-R_*)(d+\sigma+\gamma)}{(1+\psi(I_*))^2} - \tfrac{K\mu\varphi(I_*)}{1+\psi(I_*)} - \tfrac{K\varphi(I_*)(d+\sigma+\gamma)}{1+\psi(I_*)})^2.$$

Hence, if $a_1 > 0$ and $a_2 > 0$, the Eq (4.4) has no positive real root, and the real parts of all roots of Eq (4.3) are negative if $R_0 > 1$ and $\tau > 0$. Thus, the endemic equilibrium Q_* of system (2.3) with $\tau > 0$ is locally asymptotically stable. In the following, we can obtain the conditions of the $a_1 > 0$ and $a_2 > 0$.

From the Eq (3.1), we can obtain that Q_* with $\tau = 0$ is locally asymptotically stable when $C < 0$. Using $C < 0$, we deduce that

$$\begin{aligned} a_2 &= ((d+\mu)(d+\sigma+\gamma) - \sigma\mu)^2 - [-(\tfrac{K(-C)(\frac{b}{d}-I_*-R_*)(d+\sigma+\gamma)}{(1+\psi(I_*))^2} + \tfrac{K\mu\varphi(I_*)}{1+\psi(I_*)} + \tfrac{K\varphi(I_*)(d+\sigma+\gamma)}{1+\psi(I_*)})]^2 \\ &= ((d+\mu)(d+\sigma+\gamma) - \sigma\mu)^2 - (\tfrac{K(-C)(\frac{b}{d}-I_*-R_*)(d+\sigma+\gamma)}{(1+\psi(I_*))^2} + \tfrac{K\mu\varphi(I_*)}{1+\psi(I_*)} + \tfrac{K\varphi(I_*)(d+\sigma+\gamma)}{1+\psi(I_*)})^2. \end{aligned}$$

Then $a_2 > 0$ if and only if

$$(d+\mu)(d+\sigma+\gamma) - \sigma\mu > \frac{K(-C)(\frac{b}{d} - I_* - R_*)(d+\sigma+\gamma)}{(1+\psi(I_*))^2} + \frac{K\mu\varphi(I_*)}{1+\psi(I_*)} + \frac{K\varphi(I_*)(d+\sigma+\gamma)}{1+\psi(I_*)},$$

which implies

$$|C| < \tfrac{(1+\psi(I_*))^2(d+\mu)}{K(\frac{b}{d}-I_*-R_*)} - \tfrac{(1+\psi(I_*))\varphi(I_*)}{\frac{b}{d}-I_*-R_*} - \tfrac{(1+\psi(I_*))^2\sigma\mu}{K(\frac{b}{d}-I_*-R_*)(d+\sigma+\gamma)} - \tfrac{(1+\psi(I_*))\varphi(I_*)\mu}{(\frac{b}{d}-I_*-R_*)(d+\sigma+\gamma)}. \tag{4.5}$$

Furthermore, we have

$$\begin{aligned} a_1 &= (d+\sigma+\gamma)^2 + (d+\mu)^2 + 2\sigma\mu - (\tfrac{KC(\frac{b}{d}-I_*-R_*)}{(1+\psi(I_*))^2} - \tfrac{K\varphi(I_*)}{1+\psi(I_*)})^2 \\ &\geq (d+\mu)^2 - (\tfrac{KC(\frac{b}{d}-I_*-R_*)}{(1+\psi(I_*))^2} - \tfrac{K\varphi(I_*)}{1+\psi(I_*)})^2. \end{aligned}$$

Let

$$a_3 = (d+\mu)^2 - (\frac{KC(\frac{b}{d} - I_* - R_*)}{(1+\psi(I_*))^2} - \frac{K\varphi(I_*)}{1+\psi(I_*)})^2. \tag{4.6}$$

Since $C < 0$, then

$$\begin{aligned} a_3 &= (d+\mu)^2 - [-(\tfrac{K(-C)(\frac{b}{d}-I_*-R_*)}{(1+\psi(I_*))^2} + \tfrac{K\varphi(I_*)}{1+\psi(I_*)})]^2 \\ &= (d+\mu)^2 - (\tfrac{K(-C)(\frac{b}{d}-I_*-R_*)}{(1+\psi(I_*))^2} + \tfrac{K\varphi(I_*)}{1+\psi(I_*)})^2. \end{aligned}$$

Thus $a_3 > 0$ if and only if

$$d+\mu > \frac{K(-C)(\frac{b}{d} - I_* - R_*)}{(1+\psi(I_*))^2} + \frac{K\varphi(I_*)}{1+\psi(I_*)},$$

then

$$|C| < \frac{(1+\psi(I_*))^2(d+\mu)}{K(\frac{b}{d} - I_* - R_*)} - \frac{(1+\psi(I_*))\varphi(I_*)}{\frac{b}{d} - I_* - R_*}. \tag{4.7}$$

Thus, according to the inequalities of (4.5) and (4.7), we obtain $a_1 > 0$ and $a_2 > 0$ when

$$|C| < \tfrac{(1+\psi(I_*))^2(d+\mu)}{K(\frac{b}{d}-I_*-R_*)} - \tfrac{(1+\psi(I_*))\varphi(I_*)}{\frac{b}{d}-I_*-R_*} - \tfrac{(1+\psi(I_*))^2\sigma\mu}{K(\frac{b}{d}-I_*-R_*)(d+\sigma+\gamma)} - \tfrac{(1+\psi(I_*))\varphi(I_*)\mu}{(\frac{b}{d}-I_*-R_*)(d+\sigma+\gamma)}. \tag{4.8}$$

Based on the above analyses, we obtain the following results

Theorem 4.2. If $R_0 > 1$ and

$$|C| < \tfrac{(1+\psi(I_*))^2(d+\mu)}{K(\frac{b}{d}-I_*-R_*)} - \tfrac{(1+\psi(I_*))\varphi(I_*)}{\frac{b}{d}-I_*-R_*} - \tfrac{(1+\psi(I_*))^2\sigma\mu}{K(\frac{b}{d}-I_*-R_*)(d+\sigma+\gamma)} - \tfrac{(1+\psi(I_*))\varphi(I_*)\mu}{(\frac{b}{d}-I_*-R_*)(d+\sigma+\gamma)},$$

the endemic equilibrium Q_* of system (2.3) with $\tau > 0$ is locally asymptotically stable.

5. Numerical results

In this section, we apply some numerical simulations of system (2.3) to illustrate our analytical results above. The model parameter values are shown in Table 2. For simplicity, we set $\varphi(I) = I$, $\psi(I) = 10I$.

Table 2. Table of parameter values used in the numerical simulation.

b	d	K	γ	μ	σ	$\varphi(I)$	$\psi(I)$
0.2	0.21	0.3	0.04	0.18	0.01	I	$10I$

Some of these parameter values have been taken from literatures [11,30]. According to the Eq (2.4), we can compute $R_0 = 0.75 < 1$. It follows from Theorems 3.1 and 4.1 that the disease-free equilibrium Q_0 is globally asymptotically stable for any latent period, and we know that the disease will disappear. These results have been shown in Figure 1 for $\tau = 0$ and $\tau = 40$ respectively. Also it can be seen that the rate of disease extinction slows down as τ increases. It shows that the time delay has an effect on the dynamic behaviors of system (2.3), but it will eventually reach a stable state, which is consistent with the theoretical analysis results of Theorem 4.1.

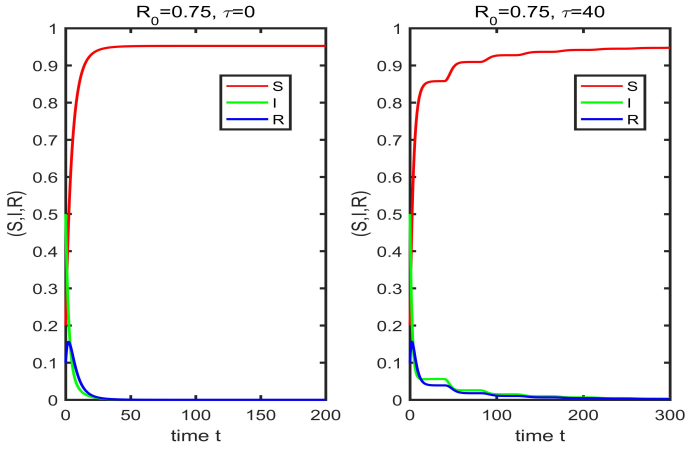

Figure 1. The disease-free equilibrium $Q_0 = (0, 0)$ is globally asymptotically stable if $R_0 < 1$ as $\tau = 0$ and $\tau = 40$.

In order to investigate the influence of nonlinear incidence rate on disease transmission, we take $\psi(I) = 10I^3$, and the values of other parameters used for this simulation are shown in Table 1. The simulation results are shown in Figure 2. Figure 2 illustrates the more complicated the incidence rate function is, the slower the disease dies out. Since $R_0 = 0.75 < 1$, the disease-free equilibrium Q_0 still tends to be stable, the disease will disappear from the environment.

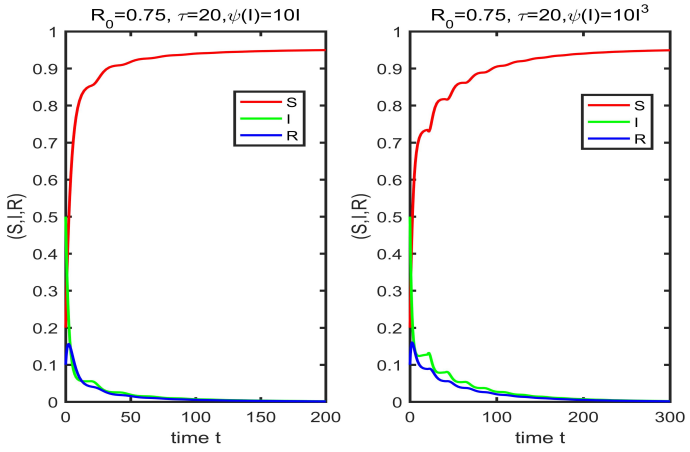

Figure 2. The disease-free equilibrium $Q_0 = (0, 0)$ is globally asymptotically stable if $R_0 < 1$, $\tau = 20$ as $\psi(I) = 10I$ and $\psi(I) = 10I^3$

To better understand how the relapse of the disease affects the dynamic behaviors of the system, we set $\sigma = 0.04$ and $\sigma = 0.3$. The other parameters are the same as in Table 2. We can get $R_0 = 0.7$ when $\sigma = 0.04$ and $R_0 = 0.9$ when $\sigma = 0.3$. Since $R_0 < 1$, the disease-free equilibrium Q_0 is globally asymptotically stable with $\tau = 20$, (see Figure 3(a)). The Figure 3(b) can be obtained by locally magnifying the Figure 3(a). Figure 3(b) illustrates the influence of the relapse on the disease risk. We observe that as σ becomes larger , the number of infected individuals I grows, the recovered individuals R decreases at the same time, indicating that a larger relapse tends to increase the risk of epidemic disease. It's also in line with our common sense of life.

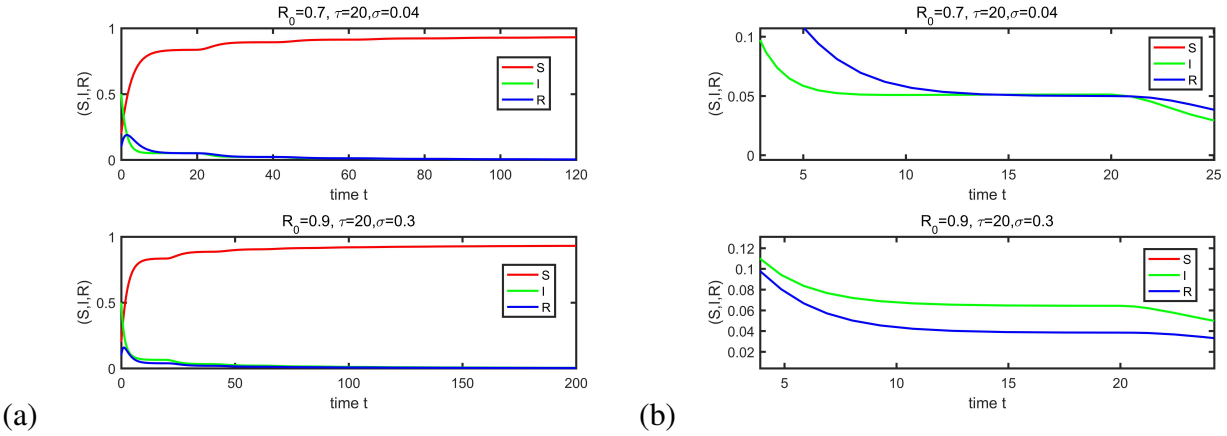

Figure 3. The disease-free equilibrium $Q_0 = (0, 0)$ is globally asymptotically stable if $R_0 < 1$, $\tau = 20$ as $\sigma = 0.04$ and $\sigma = 0.3$.

To investigate the stability of endemic equilibrium Q_* of system (2.3), we take different parameter values as shown in Table 3. For the above set of parameter values, we can get $R_0 = 2.042 > 1$. The existence of endemic equilibrium Q_* is satisfied and the endemic equilibrium for this data is $S_* = 3.21$, $I_* = 0.45$, $R_* = 0.09$. For $\tau = 0$, we have $0.35 < 0.5$, the condition (3.3) is satisfied. It follows from Theorem 3.2 that the endemic equilibrium Q_* is locally asymptotically stable without latent period, (see Figure 4(a)). For $\tau = 20$, we can compute that $0.82 < 1.57$, the condition (4.9) is also satisfied. It follows from Theorem 4.2 that the endemic equilibrium Q_* is locally asymptotically stable for $\tau = 20$, (see Figure 4(b)). From Figure 4, it may be noted that all the variables are approaching to their equilibrium states, which shows the stability of the endemic equilibrium Q_* for any latent period under some conditions. According to Theorems 3.2 and 4.2, the disease will exist everlasting.

Table 3. Table of parameter values used in the numerical simulation.

b	d	K	γ	μ	σ	$\varphi(I)$	$\psi(I)$
1.5	0.4	0.3	0.1	0.2	0.5	I	$10I^3$

If we use the same parameters as in Figure 4 except choosing $\sigma = 0.02$, $\sigma = 0.5$, then, Figure 5 shows the influence of recurrence on stability of the endemic equilibrium Q_*. By simple computations, we can derive $R_0 = 1.882$ when $\sigma = 0.02$ and $R_0 = 2.042$ when $\sigma = 0.5$. If $\sigma = 0.02$, the existence of endemic equilibrium Q_* is satisfied and the endemic equilibrium for this data is $S_* = 3.20$, $I_* = 0.40$, $R_* = 0.15$. We find that S_*, I_* become smaller and R_* becomes larger when the relapse term σ becomes smaller. For $\sigma = 0.02$, we have $0.28 < 1.40$, the condition (4.9) is also satisfied. As what can be see from Figure 5, the disease will persist on a positive steady level, which is in agreement with Theorems 3.2 and 4.2.

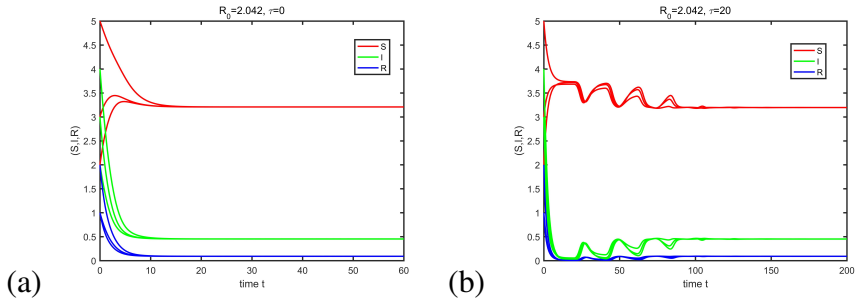

Figure 4. The endemic equilibrium $Q_* = (S_*, R_*)$ is locally asymptotically stable if $R_0 > 1$ as $\tau = 0$ and $\tau = 20$ with different initial values.

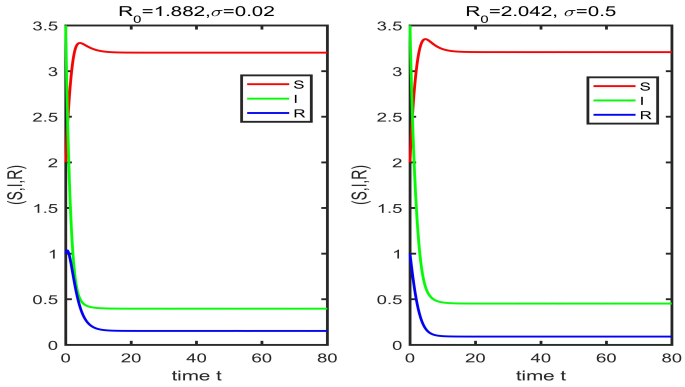

Figure 5. The endemic equilibrium $Q_* = (S_*, R_*)$ is locally asymptotically stable if $R_0 > 1$, $\tau = 20$ as $\sigma = 0.02$ and $\sigma = 0.5$.

6. Discussion and conclusions

In this paper, a delayed $SIRS$ epidemic model with a generalized nonlinear incidence rate and a discrete relapse has been proposed and investigated. our model is more realistic than some published ones because this nonlinear incidence rate can be used to explain the psychological effect. That is to say, the incidence rate will decrease at high infective levels because of the isolation of the infected individuals or the protection measures taken by susceptible individuals.

The nonlinear incidence rate which is presented in this work has a more generalized form, namely $\frac{KS\varphi(I)}{1+\psi(I)}$, and the basic reproduction number R_0 was obtained by using the next generation matrix method. This means that the disease-free equilibrium Q_0 is globally asymptotically stable when $R_0 < 1$ for any latent period. That is to say, all positive solutions converge to the disease-free equilibrium point Q_0, indicating that the disease cannot continue to transmit among individuals and will go extinct eventually. Based on epidemiological issue, the epidemic diseases will be controlled if and only if the basic reproduction number is smaller than one no matter how long latent period takes. According to this conclusion, COVID-19 always could be extinct while the reasonably controlling methods are adopted.

On the other hand, if $R_0 > 1$, the disease-free equilibrium point Q_0 becomes unstable and there occurs the epidemic equilibrium Q_* which is locally asymptotically stable in the absence of delay.

Moreover, the endemic equilibrium Q_* is locally asymptotically stable if $R_0 > 1$, $\tau > 0$ which is shown in Theorem 4.2. In this case, all positive solutions converge to Q_* and the disease will become the endemic disease. In fact, the prediction of the peak time of an epidemic disease is a interesting issue in this field. For example, M. Turkyilmazoglu et al. considered this interesting issue and found the explicit formulae for the peak time of COVID-19. This explicit formulae for the peak time of COVID-19 could enable the governments to take early effective epidemic precautions [38,39].

Furthermore, some numerical simulations are conducted to test the theoretical results, and these provide some comprehensively insights into the corresponding roles of different parameters in the transmission of the epidemic diseases. For example, our numerical cases show that the basic reproduction number R_0 is increasing with the relapse rate σ. That is to say, the recurrence span will become larger and the risk of the disease's transmission gets higher. Thus, the disease control measures will be focused more on reducing the relapse period. For COVID-19, keeping social distance, wearing mask and vaccinating vaccine are the most effective controlling measures.

However, the epidemic model will lose its stability while the time delay exceeds the threshold value, and the Hopf bifurcation of the considered model system should be investigated by using the normal form theory and the center manifold theorem. Meanwhile, the direction of Hopf bifurcation and the stability of bifurcating periodic solutions will be an interesting issue [40]. Furthermore, most epidemics is inevitably affected by random factors in real nature, and many published researches had focused on this field and some more realistic conclusions were obtained [41–45]. Therefore, it is highly necessary to consider some stochastic factors in our $SIRS$ epidemic model. We leave these questions for future work.

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Conflict of interest

The authors declare no conflict of interest.

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Appendix

Proposition 1. The region $S_{\triangle} = \{(I, R)|I \geq 0, R \geq 0, I + R \leq \frac{b}{d}\}$ is positively invariant set and also an absorbing set of system (2.3) in the first quadrant.

Proof. From system (2.3), we can know that on the line $I = 0$, $\frac{dI}{dt} > 0$, and on the line $R = 0$, $\frac{dR}{dt} > 0$. This results ensures that I and R are non-negative, and no orbit of system (2.3) can exit from the first quadrant, with the boundary $R = 0$ and $I = 0$.

From the Eq (2.2), it is obtained that

$$\lim_{t\to\infty} I(t) \leq \lim_{t\to\infty} N(t) = \frac{b}{d}, \lim_{t\to\infty} R(t) \leq \lim_{t\to\infty} N(t) = \frac{b}{d}.$$

Since

$$\begin{aligned} \tfrac{d(I+R)}{dt} \mid_{I+R=\frac{b}{d}} &= \tfrac{dI}{dt} + \tfrac{dR}{dt} \mid_{I+R=\frac{b}{d}} \\ &= \tfrac{K\varphi(I(t-\tau))}{1+\psi(I(t-\tau))}(\tfrac{b}{d} - I - R) - d(I + R) - \gamma R \mid_{I+R=\frac{b}{d}} \\ &= -b - \gamma R < 0, \end{aligned}$$

the orbit of system (2.3) getting at the boundary $I + R = \frac{b}{d}$ must go into the interior of the region $S_{\triangle}$. Thus the region $S_{\triangle} = \{(I, R)|I \geq 0, R \geq 0, I + R \leq \frac{b}{d}\}$ is an absorbing set of system (2.3) in the first quadrant. This completes the proof.

Proposition 2. There exist at most two equilibria in the region $S_{\triangle}$.
(i) If $R_0 \leq 1$, system (2.3) has the disease-free equilibrium $Q_0 = (0, 0)$.
(ii) If $R_0 > 1$ and $\frac{K(\frac{b}{d}-I-R)\varphi'(I)}{I\psi'(I)} < d + \frac{\mu(d+\gamma)}{d+\sigma+\gamma}$, system (2.3) has two equilibria, the disease-free equilibrium $Q_0 = (0, 0)$ and the unique endemic equilibrium $Q_* = (I_*, R_*)$ regardless of the time delay length.
Proof. Obviously, the system (2.3) always has an equilibrium Q_0. Furthermore, the system (2.3) has a positive equilibrium Q_* if and only if it satisfies the following equations

$$\begin{cases} \frac{K\varphi(I)}{1+\psi(I)}(\frac{b}{d} - I - R) - (d + \mu)I + \sigma R = 0, \\ \mu I - (d + \sigma + \gamma)R = 0. \end{cases} \tag{6.1}$$

It is equivalent to I_* being a positive solution of

$$\frac{d(1 + \psi(I))}{K\varphi(I)}(d + \mu - \frac{\sigma\mu}{d + \sigma + \gamma})I + (d + \mu - \frac{(\sigma + \gamma)\mu}{d + \sigma + \gamma})I - b = 0. \tag{6.2}$$

Set

$$F(I) = \frac{d(1 + \psi(I))}{K\varphi(I)}(d + \mu - \frac{\sigma\mu}{d + \sigma + \gamma})I + (d + \mu - \frac{(\sigma + \gamma)\mu}{d + \sigma + \gamma})I - b.$$

Then

$$F(\frac{b}{d}) = d(1 + \psi(\frac{b}{d}))(d + \frac{\mu(d + \gamma)}{d + \sigma + \gamma}) + \frac{K\varphi(\frac{b}{d})\mu d}{d + \sigma + \gamma},$$

$$\lim_{I\to 0^+} F(I) = \frac{d(d + \mu - \frac{\sigma\mu}{d+\sigma+\gamma})}{K\varphi'(0)} - b = -\frac{d(d + \mu)}{K\varphi'(0)}(R_0 - 1).$$

Clearly, it appears that $F(\frac{b}{d}) > 0$. It is easy to see that $\lim_{I\to 0^+} F(I) < 0$ when $R_0 > 1$.
Furthermore

$$F'(I) = \frac{d(1 + \psi(I))}{K\varphi(I)}(d+\frac{\mu(d + \gamma)}{d + \sigma + \gamma})+(d+\frac{\mu d}{d + \sigma + \gamma})-(d+\frac{\mu(d + \gamma)}{d + \sigma + \gamma})I\frac{dK[(1 + \psi(I))\varphi'(I) - \psi'(I)\varphi(I)]}{K^2\varphi^2(I)}.$$

Let $C = (1 + \psi(I))\varphi'(I) - \psi'(I)\varphi(I)$.
It is obtained that $F'(I) > 0$ when

$$C = (1 + \psi(I))\varphi'(I) - \psi'(I)\varphi(I) < 0. \tag{6.3}$$

According to the intermediate value theorem, if $R_0 > 1$ and $C < 0$, we have $F(I)$ is strictly monotone increasing for $I > 0$.
The Eq (2.7) is equivalent to the following inequality

$$\frac{K(\frac{b}{d} - I - R)\varphi'(I)}{I\psi'(I)} < d + \frac{\mu(d + \gamma)}{d + \sigma + \gamma}.$$

This implies that the Eq (2.6) has one unique positive solution and the conclusion in case (ii) holds. This completes the proof.