Journal of Nonlinear Modeling and Analysis
Volume 5, Number 3, September 2023, 524–539
http://jnma.ca; http://jnma.ijournal.cn
DOI:10.12150/jnma.2023.524

Mathematical Analysis of SIR Epidemic Model with Piecewise Infection Rate and Control Strategies

Yu Yang[1], Tariq Q. S. Abdullah[1], Gang Huang[1,†] and Yueping Dong[2]

Abstract The limitation of contact between susceptible and infected individuals plays an important role in decreasing the transmission of infectious diseases. Prevention and control strategies contribute to minimizing the transmission rate. In this paper, we propose SIR epidemic model with delayed control strategies, in which delay describes the response and effect time. We study the dynamic properties of the epidemic model from three aspects: steady states, stability and bifurcation. By eliminating the existence of limit cycles, we establish the global stability of the endemic equilibrium, when the delay is ignored. Further, we find that the delayed effect on the infection rate does not affect the stability of the disease-free equilibrium, but it can destabilize the endemic equilibrium and bring Hopf bifurcation. Theoretical results show that the prevention and control strategies can effectively reduce the final number of infected individuals in the population. Numerical results corroborate the theoretical ones.

Keywords Epidemic model, prevention and control strategy, piecewise infection rate, Hopf bifurcation

MSC(2010) 34D20, 34K20.

1. Introduction

Mathematical model is an important tool of describing the transmission dynamics of infectious diseases and evaluating the control strategies for curbing epidemics, which has become a modern and interesting direction in the infectious diseases field. Currently, the transmission of infectious diseases modeling has been widely studied, including classical SI, SIR compartment models and so on [2, 13, 19, 21]. In the process of infectious diseases modeling, disease incidence plays a key role in the long-term dynamic behaviour of the model. Most traditional models of infectious diseases assume that the incidence is standard or bilinear. However, the saturation incidence sometimes gives a better representation of the dynamics of infectious diseases [7, 15, 18].

[†]The corresponding author.
Email address: yuyang@cug.edu.cn (Y. Yang), tariq.almoqri@tu.edu.ye (T. Q. S. Abdullah), huanggang@cug.edu.cn (G. Huang), ypdong@mail.ccnu.edu.cn (Y. Dong)
[1]School of Mathematics and Physics, China University of Geosciences, Wuhan, Hubei 430074, China
[2]School of Mathematics and Statistics, Central China Normal University, Wuhan, Hubei 430079, China

Various dynamic models have been proposed by mathematicians to investigate the spread and evolution of infectious diseases. Chang, Meng and Zhang [1] investigated a nonlinear stochastic SIS epidemic system with multiplicative noise. Qi et al., [20] proposed two new stochastic non-autonomous SEIS epidemic dynamical models with latent and active patients. He, Wang and Huang [8] studied a nonlocal and time-delayed reaction-diffusion epidemic model with the vaccination strategy in a heterogeneous habitat.

As a means of prevention and control strategies, media reports attract people's attention by reporting the number of infected individuals, deaths and preventive policies. Then, people can reduce the risk of infection by wearing masks and washing their hands frequently. Many researchers have proposed some different mathematical functions or models to describe the impact of media reports on infection rate [3, 22, 27]. Moreover, threshold policy is often used in the control measures [3, 24]. It is often the case that when the number of cases exceeds a certain value, the government will remind the public of avoiding contacting infectious sources and suspected patients for the purpose of crisis awareness of the disease. Thus, it will effectively reduce the incidence of the disease. Therefore, piecewise incidence has been established in many pieces of literature [24, 28].

For many infectious diseases, it is important to consider the influences of delays on the disease dynamics. Time delay is very common in the transmission of infectious diseases and can be caused by various factors. The most notable reasons for a delay are the latency of the infection in a vector and the latent period in an infected host [9, 10, 23, 25]. In these cases, the infection takes some time in the infected host or vector and develops itself during a short period to become in the infection transmission stage. Many scholars have considered mathematical modeling of population dynamics with time delay [4, 12]. Introducing time delay can present more complex transmission dynamics properties.

In real life, prevention and control strategies may not be applied promptly, which means that the infection rate is related to the number of previously infected people. In this paper, we introduce the piecewise infection rate with delayed control strategies into the SIR epidemic model in order to describe the impact of control strategies on the development trend of infectious diseases. By studying the dynamic properties of the model, we show that control strategies can effectively reduce the number of infected individuals. To reduce fluctuations in the number of infected individuals, it is essential to ensure that control strategies are implemented in time.

The paper is organized as follows. The derivation and transformation of the epidemic model are presented in Section 2. We investigate the critical conditions for extinction and permanence of infectious diseases by Filippov system in Section 3. Section 4 contains dynamic properties analysis of the derived model without delay. In Section 5, we discuss the effect of delay on dynamic properties of the equilibria by the bifurcation theory. In Section 6, we make numerical simulations to verify the theoretical results. Our conclusions are given in Section 7 to end this work.

2. Model derivation

To illustrate our ideas, we begin with a simple compartment SI epidemic model. Assuming that the birth and death rate are zero (i.e., no demography), a simple SI model with saturation incidence is given by the following system of ordinary

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11

differential equations

$$\dot{S}(t) = -\frac{\beta S(t)I(t)}{1+I(t)}, \quad \dot{I}(t) = \frac{\beta S(t)I(t)}{1+I(t)} \tag{2.1}$$

with initial values $N = S_0 + I_0$. N means that the total population size is a constant. Here, $S(t)$ and $I(t)$ represent the number of susceptible individuals and infected individuals in the population at time t respectively. β represents the infection rate, and it is assumed to be a positive constant. Since $S(t) = N - I(t)$, a modified model from (2.1) reads

$$\dot{I}(t) = \frac{\beta(N - I(t))I(t)}{1+I(t)}. \tag{2.2}$$

It is known that if $I_0 > 0$, then there exists $\lim\limits_{t\to\infty} I(t) = N$, which implies that all the individuals in the population will be infected.

With the spread of the epidemic, public health authorities provide a lot of information and reports through mass media on the risk of disease, transmission routes, and protection methods, which attracts much attention. It makes people change their behaviours which will reduce the probability of the disease transmission in the population. This case shows that the infection rate should be assumed as a decreasing function of the number of the infected individuals. When the number of infected individuals exceeds a certain threshold, the government will strictly control the contact between susceptible individuals and infected individuals, for example, the isolation and lockdown of the community, which implies that the infection rate highly decrease. Thus, motivated by the works [3,4,11,27], we construct a piecewise function to describe the impact of prevention and control strategy on infection rate by

$$\beta(I) = \begin{cases} \beta(1-\delta I), & 0 \le I \le \frac{1}{\delta}, \\ 0, & I > \frac{1}{\delta}, \end{cases} \tag{2.3}$$

where β is the maximum infection rate, δ represents the intensity of prevention and control strategies, and $\frac{1}{\delta}$ means the exposure infection threshold. In addition, $0 \le \delta \le 1$, $\delta = 0$ implies that the prevention and control strategies are not taken, while $\delta = 1$ implies the maximum prevention and control efforts. The piecewise infection rate function is more realistic and effective than the constant infection rate in describing the state of epidemic transmission. For the sake of reality, we assume that the exposure infection threshold is smaller than the total population (i.e., $\frac{1}{\delta} < N$). The equation for describing infectious diseases by (2.2) and (2.3) is as follows.

$$\dot{I}(t) = \frac{\beta \mathbf{max}\{0, 1-\delta I(t)\}(N - I(t))I(t)}{1+I(t)}. \tag{2.4}$$

Obviously, equation (2.4) can be expressed as the following segmented model

$$\dot{I}(t) = \frac{\beta(1-\delta I(t))(N - I(t))I(t)}{1+I(t)}, \quad 0 \le I \le \frac{1}{\delta} \tag{2.5}$$

and

$$\dot{I}(t) = 0, \quad \frac{1}{\delta} < I < N. \tag{2.6}$$

We give a simple analysis for model (2.5). There always exists a disease-free equilibrium $I_0 = 0$ and a positive equilibrium $I^* = \frac{1}{\delta}$. For the stability of equilibria of (2.5), we have the following results.

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11

Theorem 2.1. *For model* (2.5)*, the following results are obtained.*

(i) The disease-free equilibrium I_0 is always unstable;

(ii) The positive equilibrium I^* is asymptotically stable.

Proof. (i) Let $g(I) = \frac{\beta(1-\delta I)(N-I)I}{1+I}$. Then we obtain

$$g^{'}(I) = \frac{\beta(2\delta I^3 + (3\delta - \delta N - 1)I^2 - (2 + 2\delta N)I + N)}{(1+I)^2}.$$

Since $g^{'}(I)|_{I=I_0} = \beta N > 0$, the disease-free equilibrium I_0 of (2.5) is always unstable.

(ii) For the positive equilibrium I^* of (2.5), we get $g^{'}(I)|_{I=I^*} = -\frac{\beta(\delta N-1)}{1+\delta} < 0$. That is, I^* is locally asymptotically stable. Further, we discuss the global stability of the positive equilibrium I^* of (2.5) by classical Lyapunov method. Define a function

$$V(t) = I - N - N\ln\frac{I}{N}.$$

Notice that the function $h(x) = x - 1 - \ln x$ is non-negative. Then $V(t)$ is also non-negative for all $t > 0$. The derivative of $V(t)$ along the solutions of (2.5) is

$$\frac{dV(t)}{dt} = (1 - \frac{N}{I})\frac{\beta(1-\delta I)(N-I)I}{1+I} = -\frac{\beta(1-\delta I)(N-I)^2}{1+I}.$$

It follows from the solution set for (2.5) that $N - I > 0$ for all $t > 0$, which implies that $\frac{dV(t)}{dt} = 0$, if and only if $I = \frac{1}{\delta}$. According to Lasalle Invariant Principle [16], the positive equilibrium I^* of (2.5) is globally asymptotically stable . □

Theorem 2.1 implies that the number of infected individuals eventually reaches the exposure infection threshold and will decrease with the increase of prevention and control efforts. Compared with the analysis results of (2.2), the prevention and control efforts can reduce the number of infected individuals at the equilibrium point, which inhibits the spread of infectious diseases.

In fact, it takes time for prevention and control strategies to have an impact on the spread of infectious diseases. To be specific, patients are not immediately diagnosed as infectious, and in cases where patients do not know they are infected, infection rate is related to the number of infected individuals over time. Therefore, we propose the infection rate function with discrete delay

$$\beta(I) = \begin{cases} \beta(1 - \delta I(t-\tau)), & 0 \le I \le \frac{1}{\delta}, \\ 0, & I > \frac{1}{\delta}, \end{cases} \tag{2.7}$$

where time delay τ refers to the time when the benefit of prevention and control strategy comes out and the patient is clearly confirmed to be infectious. Since the population movement is an important factor for infectious diseases to geographically spread over the time, we include the demographic process to explore the long-term persistence and endemic dynamics here. Based on the above considerations, we establish an epidemic model with the segmented infection rate under delayed prevention and control strategies by

$$\begin{cases} \dot{S}(t) &= \Lambda - dS(t) - \frac{\beta\mathbf{max}\{0,1-\delta I(t-\tau)\}S(t)I(t)}{1+I(t)}, \\ \dot{I}(t) &= \frac{\beta\mathbf{max}\{0,1-\delta I(t-\tau)\}S(t)I(t)}{1+I(t)} - cI(t) - dI(t), \\ \dot{R}(t) &= cI(t) - dR(t), \end{cases} \tag{2.8}$$

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11

where all parameters are positive, Λ is the intrinsic growth rate of the human population, d is the natural death rate, β is the maximum infection rate, δ is the prevention and control strategies efforts, and c is the recovery rate. Since $R(t)$ does not appear in the first two equations of system (2.8), we can simplify to the following segmented model

$$\begin{cases} \dot{S}(t) &= \Lambda - dS(t) - \frac{\beta \mathbf{max}\{0,1-\delta I(t-\tau)\}S(t)I(t)}{1+I(t)}, \\ \dot{I}(t) &= \frac{\beta \mathbf{max}\{0,1-\delta I(t-\tau)\}S(t)I(t)}{1+I(t)} - pI(t), \end{cases} \tag{2.9}$$

where $p = c + d$. The initial conditions of model (2.9) are given as

$$S(\theta) = \phi_1(\theta) > 0, \quad I(\theta) = \phi_2(\theta) > 0, \quad \theta \in [-\tau, 0]. \tag{2.10}$$

We denote by $\mathbb{C}$ the Banach space of continuous functions $\phi : [-\tau, 0] \to \mathbf{R}^2$ equipped with the suitable norm. Furthermore, we have

$$\mathbb{C}_+ = \phi = (\phi_1, \phi_2) \in \mathbb{C} : \phi_i(\theta) \geq 0 \text{ for all } \theta \in [-\tau, 0], \quad i = 1, 2.$$

By means of the fundamental theory of functional differential equations [6, 14], we can verify that there is a unique solution for system (2.9) with initial conditions (2.10). Then, we discuss the positivity and boundedness of the solution. Letting $(S(t), I(t))$ be any solution of system (2.9) satisfying conditions (2.10) and $N(t) = S(t) + I(t)$, we get

$$\dot{N}(t) \leq \Lambda - dN(t).$$

It is clear that the positive invariant set of system (2.9) with initial conditions (2.10) is

$$\Omega = \{(S, I) \in \mathbf{R}^2_+ : 0 \leq S, I \leq N \leq \frac{\Lambda}{d}\}.$$

Furthermore, model (2.9) is well posed.

3. Equilibrium analysis of segmented model

The existence condition of equilibria of system (2.9) is discussed below. Since the prevention and control strategy is primarily aimed at reducing the number of infected individuals, we consider the case where the exposure infection threshold is smaller than the total population (i.e., $\frac{1}{\delta} < \frac{\Lambda}{d}$). Define $Z = (S, I)^T$, and

$$F_{G_1}(Z) = (\Lambda - dS(t) - \frac{\beta(1-\delta I(t))S(t)I(t)}{1+I(t)}, \frac{\beta(1-\delta I(t))S(t)I(t)}{1+I(t)} - pI(t))^T,$$
$$F_{G_2}(Z) = (\Lambda - dS(t), -pI(t))^T.$$

Thus, when $\tau = 0$, model (2.9) is equivalent to the following system

$$\dot{Z}(t) = \begin{cases} F_{G_1}(Z), & Z \in G_1, \\ F_{G_2}(Z), & Z \in G_2, \end{cases} \tag{3.1}$$

where

$$G_1 = \{Z \in R^2_+ : 0 \leq I < \frac{1}{\delta}\}, \quad G_2 = \{Z \in R^2_+ : \frac{1}{\delta} \leq I \leq \frac{\Lambda}{d}\}.$$

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11

In addition, the switching surface separating the areas G_1 and G_2 is $\Sigma = \{Z \in R_+^2 : I = \frac{1}{\delta}\}$. Therefore, we call the part of system (3.1) in region G_1 as system S_{G_1} and the part of system (3.1) in region G_2 as system S_{G_2}. System (3.1) is a special form of Filippov system [5]. For the convenience of discussion, the following definitions are given for each equilibrium of piecewise smooth system (3.1).

Definition 3.1. If Z_* satisfies $F_{S_{G_1}}(Z_*) = 0$, $Z_* \in G_1$ or $F_{S_{G_2}}(Z_*) = 0$, $Z_* \in G_2$, then it is called the regular equilibrium state of system (3.1). If Z_* satisfies $F_{S_{G_1}}(Z_*) = 0$, $Z_* \in G_2$ or $F_{S_{G_2}}(Z_*) = 0$, $Z_* \in G_1$, then it is called the virtual equilibrium state of system (3.1).

First, we analyze the existence of all possible equilibria of system S_{G_1}. Obviously, there always exists a disease-free equilibrium $E_0 = (\frac{\Lambda}{d}, 0)$. Then, if the endemic equilibrium $E_* = (S_*, I_*)$ exists, S_* must satisfy $S_* = \frac{p(1+I_*)}{\beta(1-\delta I_*)}$, and I_* is determined by the following equation

$$H(I) = p\beta\delta I^2 - (\Lambda\beta\delta + dp + p\beta)I + \Lambda\beta - dp = 0 \tag{3.2}$$

and

$$\begin{aligned}\Delta &= (\Lambda\beta\delta + dp + p\beta)^2 - 4p\beta\delta(\Lambda\beta - dp)\\ &= (\Lambda\beta\delta - p\beta)^2 + d^2p^2 + 2(\Lambda\beta\delta + p\beta)dp + 4\beta\delta dp^2\\ &> 0.\end{aligned}$$

Therefore, equation (3.2) has two roots

$$I_1 = \frac{(\Lambda\beta\delta + dp + p\beta) - \sqrt{\Delta}}{2p\beta\delta}, \quad I_2 = \frac{(\Lambda\beta\delta + dp + p\beta) + \sqrt{\Delta}}{2p\beta\delta}.$$

To make sure that S_* is positive, we must have $0 < I_* < \frac{1}{\delta}$. It follows from equation (3.2) that $H(\frac{1}{\delta}) = -dp(1 + \frac{1}{\delta}) < 0$, then we obtain $I_2 > \frac{1}{\delta}$, and I_2 is omitted. Now, notice that $0 < I_1 < \frac{1}{\delta}$, if and only if $H(0) = \Lambda\beta - dp > 0$, which indicates that if $R_0 = \frac{\Lambda\beta}{dp} > 1$, system S_{G_1} has a unique endemic equilibrium $E_* = (S_*, I_*)$, and it is the regular equilibrium state of system (3.1), where

$$S_* = \frac{p(1 + I_*)}{\beta(1 - \delta I_*)}, \quad I_* = I_1 = \frac{(\Lambda\beta\delta + dp + p\beta) - \sqrt{\Delta}}{2p\beta\delta}.$$

Second, we analyze the existence of all possible equilibria of system S_{G_2}. Clearly, $\bar{E}_0 = (\bar{S}_0, \bar{I}_0) = (\frac{\Lambda}{d}, 0)$ is a unique equilibrium of system S_{G_2}. By Definition (3.1), it shows that $\bar{E}_0$ is the virtual equilibrium state of system (3.1). Finally, we conclude that model (2.9) and model (3.1) have the same equilibrium point.

4. Dynamic properties of segmented model without time delay

In this section, we continue to study the stability of equilibria state $\bar{E}_0$, E_0 and E_* of segmented model (2.9) without time delay. When $\tau = 0$, model (2.9) simplifies to

$$\begin{cases}\dot{S}(t) &= \Lambda - dS(t) - \frac{\beta\mathbf{max}\{0,1-\delta I(t)\}S(t)I(t)}{1+I(t)},\\ \dot{I}(t) &= \frac{\beta\mathbf{max}\{0,1-\delta I(t)\}S(t)I(t)}{1+I(t)} - pI(t).\end{cases} \tag{4.1}$$

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11

Since model (3.1) is equivalent to model (4.1), here we will discuss the dynamic properties of model (3.1) in the same way as in [17, 29].

Theorem 4.1. *For system S_{G_1}, the disease-free equilibrium E_0 is locally asymptotically stable, when $R_0 < 1$, while the endemic equilibrium E_* is locally asymptotically stable, when $R_0 > 1$. For system S_{G_2}, the virtual equilibrium $\bar{E}_0$ is locally asymptotically stable.*

Proof. For system S_{G_1}, the Jacobian matrix of system S_{G_1} at E_0 satisfies

$$J(E_0) = \begin{pmatrix} -d & -\frac{\Lambda\beta}{d} \\ 0 & \frac{\Lambda\beta}{d} - p. \end{pmatrix}.$$

If $R_0 = \frac{\Lambda\beta}{dp} < 1$, the eigenvalues of $J(E_0)$ are $\lambda_1 = -d < 0$, and $\lambda_2 = \frac{\Lambda\beta}{d} - p < 0$, which indicates that the disease-free equilibrium E_0 is locally asymptotically stable. Similarly, for system S_{G_2}, we can obtain that the eigenvalues of $J(\bar{E}_0)$ are $\bar{\lambda}_1 = -d < 0$ and $\bar{\lambda}_2 = -p < 0$. Therefore, the virtual equilibrium $\bar{E}_0$ is also locally asymptotically stable.

By setting the equations in system (4.1) to be zero, it is clear that the following equations hold at the endemic equilibrium E_*.

$$\Lambda - dS_* - \frac{\beta(1-\delta I_*)S_*I_*}{1+I_*} = 0, \quad \frac{\beta(1-\delta I_*)S_*}{1+I_*} - p = 0.$$

Hence, the Jacobian matrix of system S_{G_1} at E_* can be given by

$$J(E_*) = \begin{pmatrix} -\frac{\Lambda}{S_*} & -\frac{\beta S_*(1-2\delta I_* - \delta {I_*}^2)}{(1+I_*)^2} \\ \frac{\Lambda}{S_*} - d & \frac{\beta S_*(1-2\delta I_* - \delta {I_*}^2)}{(1+I_*)^2} - p \end{pmatrix}.$$

Then, the associated characteristic equation of system S_{G_1} at E_* can be described as

$$\lambda^2 + (\frac{\Lambda}{S_*} - \frac{\beta S_*(1-2\delta I_* - \delta {I_*}^2)}{(1+I_*)^2} + p)\lambda + \frac{p\Lambda}{S_*} - \frac{d\beta S_*(1-2\delta I_* - \delta {I_*}^2)}{(1+I_*)^2} = 0. \tag{4.2}$$

From (4.2), we can derive that the eigenvalues λ_3 and λ_4 have the following relationship

$$\lambda_3 + \lambda_4 = \frac{\beta S_*(1-2\delta I_* - \delta {I_*}^2)}{(1+I_*)^2} - \frac{\Lambda}{S_*} - p,$$
$$\lambda_3\lambda_4 = \frac{p\Lambda}{S_*} - \frac{d\beta S_*(1-2\delta I_* - \delta {I_*}^2)}{(1+I_*)^2}.$$

When $R_0 = \frac{\Lambda\beta}{dp} > 1$, we have

$$\begin{aligned} \lambda_3 + \lambda_4 &= \frac{p(1-2\delta I_* - \delta {I_*}^2)}{(1+I_*)(1-\delta I_*)} - \frac{\Lambda\beta(1-\delta I_*)}{p(1+I_*)} - p \\ &= -\frac{p^2(\delta I_* + I_*) + \Lambda\beta(1-\delta I_*)^2}{p(1+I_*)(1-\delta I_*)} \end{aligned}$$

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11

$$< 0.$$

$$\begin{aligned}\lambda_3\lambda_4 &= \frac{\Lambda\beta(1-\delta I_*)^2 - dp(1-2\delta I_* - \delta {I_*}^2)}{(1+I_*)(1-\delta I_*)} \\ &> \frac{dp(1-\delta I_*)^2 - dp(1-2\delta I_* - \delta {I_*}^2)}{(1+I_*)(1-\delta I_*)} \\ &= \frac{dp(\delta^2 {I_*}^2 + \delta {I_*}^2)}{(1+I_*)(1-\delta I_*)} \\ &> 0.\end{aligned}$$

Thus, the characteristic equation (4.2) has two eigenvalues with negative real parts. It implies that E_* is locally asymptotically stable. □

To further discuss the global stability of each equilibrium point, we introduce the following Lemma [26]. For simplicity, the two equations on the right side of the equation of system (4.1) are expressed by h_1, h_2 respectively.

Lemma 4.1. *If the continuous function G is continuously differentiable in $\mathbf{R}^2_+$, when $I \neq \frac{1}{\delta}$, and*

$$\frac{\partial(Gh_1)}{\partial S} + \frac{\partial(Gh_2)}{\partial I}$$

is either strictly positive in $\mathbf{R}^2_+$ or strictly negative in $\mathbf{R}^2_+$, when $I \neq \frac{1}{\delta}$, then system (4.1) has no limit cycles.

Theorem 4.2. *System (4.1) has no limit cycles.*

Proof. With regards to system (4.1), we obtain

$$\begin{aligned}h_1 &= \Lambda - dS(t) - \frac{\beta\mathbf{max}\{0, 1-\delta I(t)\}S(t)I(t)}{1+I(t)}, \\ h_2 &= \frac{\beta\mathbf{max}\{0, 1-\delta I(t)\}S(t)I(t)}{1+I(t)} - pI(t).\end{aligned}$$

Considering the continuous Dulac function $G(S, I) = \frac{1}{I}$, we can calculate that, if $0 \leq I < \frac{1}{\delta}$,

$$\frac{\partial(Gh_1)}{\partial S} + \frac{\partial(Gh_2)}{\partial I} = -\frac{d}{I} - \frac{\beta(1-\delta I)}{1+I} - \frac{\beta S(1+\delta)}{(1+I)^2} < 0,$$

and if $\frac{1}{\delta} \leq I \leq \frac{\Lambda}{d}$,

$$\frac{\partial(Gh_1)}{\partial S} + \frac{\partial(Gh_2)}{\partial I} = -\frac{d}{I} < 0.$$

By Lemma 4.1, it is shown that system (4.1) has no limit cycles. □

Theorem 4.3. *If $R_0 < 1$, the disease-free equilibrium E_0 of (4.1) is globally asymptotically stable. If $R_0 > 1$, the endemic equilibrium E_* of (4.1) is globally asymptotically stable.*

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11

Proof. Notice that the virtual equilibrium $\bar{E}_0$ of system S_{G_2} is locally asymptotically stable, and it is located in region G_1. Thus, all curves of the starting point in region G_2 will eventually enter region G_1.

According to Theorem 4.1, when $R_0 < 1$, the disease-free equilibrium E_0 of system S_{G_1} is locally asymptotically stable, and it is a regular equilibrium state of system S_{G_1}. Meanwhile, the endemic equilibrium E_* does not exist. It is obvious that the disease-free equilibrium E_0 of system S_{G_1}, and the virtual equilibrium $\bar{E}_0$ of system S_{G_2} are coincident. By Theorem 4.2, there is no limit cycle in system (4.1). It can be concluded that all curves of the starting point in region G_1 and G_2 will eventually approach E_0. It can be deduced that the disease-free equilibrium E_0 is globally asymptotically stable.

When $R_0 > 1$, the disease-free equilibrium E_0 in region G_1 is unstable. The endemic equilibrium E_* of system S_{G_1} is locally asymptotically stable, and it is a regular equilibrium state of system S_{G_1}. All the curves in region G_1 tend to E_*. By Theorem 4.2, there is no limit cycle in system (4.1), so all the curves starting from region G_1 and G_2 will approach E_* at last. It means that E_* is globally asymptotically stable. □

5. Dynamic properties of segmented model with time delay

In this section, we discuss the effects of time delay τ on the stability of E_0 and E_* of segmented model (2.9), where $0 < I_* < \frac{1}{\delta}$. Therefore, we have $\mathbf{max}\{0, 1-\delta I_*\} = 1 - \delta I_*$.

Theorem 5.1. *For model* (2.9)*, if* $R_0 < 1$*, the disease-free equilibrium* E_0 *is locally asymptotically stable for any time delay* $\tau > 0$*. If* $R_0 > 1$*, the disease-free equilibrium* E_0 *is unstable for any time delay* $\tau > 0$*.*

Proof. When $\tau > 0$, linearizing system (2.9) at $\hat{E} = \{\hat{S}, \hat{I}\}$, we obtain

$$\begin{cases} \dot{S}(t) &= \left(-d - \frac{\beta\hat{I}(1-\delta\hat{I})}{1+\hat{I}}\right) S(t) - \frac{\beta\hat{S}(1-\delta\hat{I})}{(1+\hat{I})^2} I(t) + \frac{\beta\delta\hat{S}\hat{I}}{1+\hat{I}} I(t-\tau), \\ \dot{I}(t) &= \frac{\beta\hat{I}(1-\delta\hat{I})}{1+\hat{I}} S(t) + \left(\frac{\beta\hat{S}(1-\delta\hat{I})}{(1+\hat{I})^2} - p\right) I(t) - \frac{\beta\delta\hat{S}\hat{I}}{1+\hat{I}} I(t-\tau), \end{cases} \tag{5.1}$$

where $\hat{E} = (\hat{S}, \hat{I})$ denotes any regular equilibrium of system (2.9). Let $\vec{M}(t) = (S(t), I(t))^T$, and system (5.1) can be transformed as

$$\frac{d\vec{M}(t)}{dt} = O\vec{M}(t) + P\vec{M}(t-\tau), \tag{5.2}$$

where

$$O = \begin{pmatrix} -d - \frac{\beta\hat{I}(1-\delta\hat{I})}{1+\hat{I}} & -\frac{\beta\hat{S}(1-\delta\hat{I})}{(1+\hat{I})^2} \\ \frac{\beta\hat{I}(1-\delta\hat{I})}{1+\hat{I}} & \frac{\beta\hat{S}(1-\delta\hat{I})}{(1+\hat{I})^2} - p \end{pmatrix}, \quad P = \begin{pmatrix} 0 & \frac{\beta\delta\hat{S}\hat{I}}{1+\hat{I}} \\ 0 & -\frac{\beta\delta\hat{S}\hat{I}}{1+\hat{I}} \end{pmatrix}.$$

Substituting $\vec{M}(t) = e^{\lambda t}\vec{\eta}$, $\vec{\eta} \neq \vec{0}$ into (5.2), then we have

$$\lambda e^{\lambda t}\vec{\eta} = Oe^{\lambda t}\vec{\eta} + Pe^{\lambda(t-\tau)}\vec{\eta},$$

in other words,

$$(\lambda E - O - Pe^{-\lambda\tau})\vec{\eta} = \vec{0},$$

where E is an identity matrix. Therefore, the associated characteristic equation of system (2.9) is given by

$$det(\lambda E - O - Pe^{-\lambda\tau}) = \begin{vmatrix} \lambda + d + \frac{\beta\hat{I}(1-\delta\hat{I})}{1+\hat{I}} & \frac{\beta\hat{S}(1-\delta\hat{I})}{(1+\hat{I})^2} - \frac{\beta\delta\hat{S}\hat{I}}{1+\hat{I}}e^{-\lambda\tau} \\ -\frac{\beta\hat{I}(1-\delta\hat{I})}{1+\hat{I}} & \lambda - \frac{\beta\hat{S}(1-\delta\hat{I})}{(1+\hat{I})^2} + p + \frac{\beta\delta\hat{S}\hat{I}}{1+\hat{I}}e^{-\lambda\tau} \end{vmatrix} = 0. \tag{5.3}$$

It is clear that the associated characteristic equation of system (2.9) at $E_0 = (\frac{\Lambda}{d}, 0) = (\hat{S}, \hat{I})$ becomes

$$(\lambda + d)(\lambda - \frac{\Lambda\beta}{d} + p) = 0. \tag{5.4}$$

Thus, equation (5.4) has two eigenvalues $\lambda_1 = -d < 0$, $\lambda_2 = \frac{\Lambda\beta}{d} - p$. When $R_0 < 1$, $\lambda_2 < 0$, which implies that E_0 is stable for any time delay $\tau > 0$. Similarly, when $R_0 > 1$, $\lambda_2 > 0$, then E_0 is unstable for any time delay $\tau > 0$. □

Theorem 5.2. *For system* (2.9)*, if (A1) holds, the endemic equilibrium E_* is locally asymptotically stable when $\tau \in (0, \tau_0)$. If (A2) holds, system* (2.9) *undergoes Hopf bifurcation at τ_0 and the endemic equilibrium E_* is unstable when $\tau \in (\tau_0, +\infty)$.*

Proof. According to (5.3), the associated characteristic equation of system (2.9) at $E_* = (S_*, I_*)$ can be written as the form

$$\lambda^2 + A\lambda + B + (C\lambda + D)e^{-\lambda\tau} = 0, \tag{5.5}$$

where

$$\begin{aligned} A &= \frac{\Lambda}{S_*} + \frac{pI_*}{1+I_*}, \\ B &= \frac{\Lambda p}{S_*} - \frac{dp}{1+I_*}, \\ C &= \frac{\beta\delta S_* I_*}{1+I_*}, \\ D &= \frac{d\beta\delta S_* I_*}{1+I_*}. \end{aligned}$$

Since (5.5) is a transcendental equation, we consider whether there exists a pair of pure imaginary roots. Let $\omega > 0$ and suppose that $\lambda = i\omega$ is a pure imaginary root of equation (5.5). By separating the real and imaginary parts, we get

$$\begin{cases} B - \omega^2 + C\omega sin(\omega\tau) + Dcos(\omega\tau) &= 0, \\ A\omega + C\omega cos(\omega\tau) - Dsin(\omega\tau) &= 0. \end{cases} \tag{5.6}$$

Thus,

$$Q(\omega) = \omega^4 + (A^2 - 2B - C^2)\omega^2 + B^2 - D^2 = 0. \tag{5.7}$$

Define $\Delta_1 = (A^2 - 2B - C^2)^2 - 4(B^2 - D^2)$.

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11

(A1): Suppose $\Delta_1 > 0$, $B^2 - D^2 < 0$.
Equation (5.7) has a positive root ω_*, where

$$\omega_* = \left(\frac{-(A^2 - 2B - C^2) + \sqrt{\Delta_1}}{2} \right)^{\frac{1}{2}}.$$

According to (5.6), we get

$$\tau^{(j)} = \frac{1}{\omega_*} arccos\left(\frac{D\omega_*^2 - AC\omega_*^2 - BD}{C^2\omega_*^2 + D^2} \right) + \frac{2j\pi}{\omega_*}, \quad j = 0, 1, 2, 3, ...$$

Define $\tau_0 = \tau^{(0)}$. Next, we continue to discuss the transversality conditions for Hopf bifurcation at $\tau = \tau_0$.

Let $\lambda(\tau) = v(\tau) + i\omega(\tau)$ be the root of equation (5.5). When $\tau = \tau_0$, we have $v(\tau_0) = 0$ and $\omega(\tau_0) = \omega_*$. Differentiating equation (5.5) with respect to τ, we have

$$\left(\frac{d\lambda}{d\tau} \right)^{-1} = \frac{2\lambda + A + Ce^{-\lambda\tau} - \tau(C\lambda + D)e^{-\lambda\tau}}{\lambda(C\lambda + D)e^{-\lambda\tau}} = \frac{(2\lambda + A)e^{\lambda\tau} + C}{\lambda(C\lambda + D)} - \frac{\tau}{\lambda},$$

and by (5.6), we can derive

$$\begin{aligned}
\mathbf{sign}\left[\frac{d(\mathrm{Re}(\lambda))}{d\tau} \right]_{\tau=\tau_0} &= \mathbf{sign}\left[\mathrm{Re}\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\tau} \right)^{-1} \right]_{\tau=\tau_0} \\
&= \mathbf{sign}\left[\mathrm{Re}\left(\frac{(2\mathrm{i}\omega_* + \mathrm{A})\mathrm{e}^{\mathrm{i}\omega_*\tau_0} + \mathrm{C}}{\mathrm{i}\omega_*(\mathrm{C}\mathrm{i}\omega_* + \mathrm{D})} \right) \right] \\
&= \mathbf{sign}\left[\frac{2\omega_*^2 + A^2 - 2B - C^2}{C^2\omega_*^2 + D^2} \right] \\
&= \mathbf{sign}\left[Q'(\omega_*^2) \right].
\end{aligned}$$

Hence, if the following condition (A2): $Q'(\omega_*^2) \neq 0$ holds, then the transversality condition for Hopf bifurcation is satisfied.

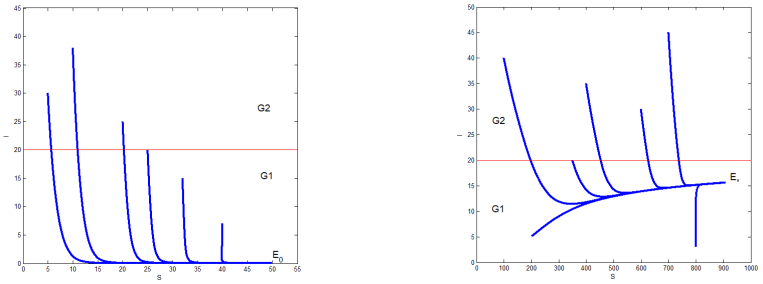
□

6. Numerical simulations

In this section, we give some numerical simulations to illustrate the main results. First, we numerically study the globally asymptotically stability of the disease-free equilibrium E_0 and the endemic equilibrium E_* of model (2.9), when $\tau = 0$. We choose a set of parameters with values $\Lambda = 1$, $\beta = 0.01$, $d = 0.02$, $p = 0.52$, $\delta = 0.05$. Then, direct calculation shows that $R_0 < 1$. Model (2.9) has a disease-free equilibrium $E_0 = (50, 0)$. From Figure 1(a), it is easy to see that all the curves at different starting points tend to approach E_0, which means that E_0 is globally asymptotically stable, when $\tau = 0$. Similarly, setting $\Lambda = 20$, $\beta = 0.01$, $d = 0.02$, $p = 0.12$, $\delta = 0.05$, we get $R_0 > 1$. Hence, model (2.9) has a unique endemic equilibrium $E_* = (906.3787, 15.6035)$. Figure 1(b) shows that E_* is globally asymptotically stable, when $\tau = 0$.

Then, we numerically analyze the effect of time delay τ on the stability of the endemic equilibrium E_* of model (2.9). Setting $\Lambda = 20$, $\beta = 0.01$, $d = 0.02$,

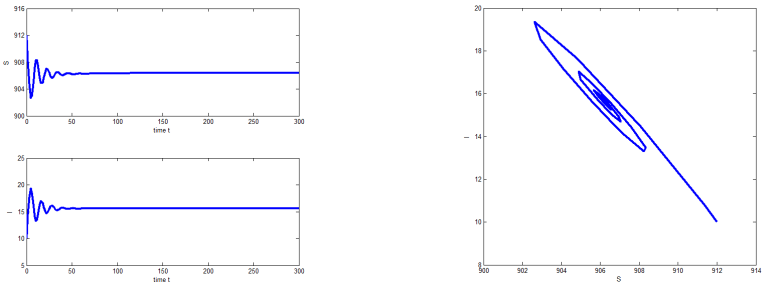
DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11



(a) (b)

Figure 1. The phase diagram of $S(t)$ and $I(t)$ of model (2.9), when $\tau = 0$: (a)$R_0 < 1$, the disease-free equilibrium E_0 of model (2.9) is globally asymptotically stable; (b)$R_0 > 1$, the endemic equilibrium E_* of model (2.9) is globally asymptotically stable

$p = 0.12$, $\delta = 0.05$, we have $R_0 > 1$, $E_* = (906.3787, 15.6035)$, $\omega_* = 0.4106$, $\tau_0 = 4.4905$, $Q'(\omega_*^2) = 0.1690 > 0$. In this case, the transversality condition for Hopf bifurcation of model (2.9) holds. It can be seen from Figure 2 that E_* is locally asymptotically stable, when $\tau = 3 < \tau_0$. Figure 3 indicates that when $\tau = 5 > \tau_0$, the number of susceptible and infected individuals changes periodically (see Figure 3(a)), and there are periodic solutions near E_* (see Figure 3(b)).

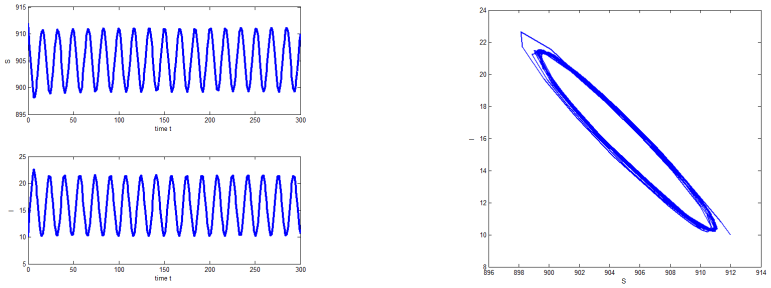


(a) (b)

Figure 2. $R_0 > 1$, $\tau = 3$: (a) the time series diagram of $S(t)$ and $I(t)$ of model (2.9); (b) the phase diagram of $S(t)$ and $I(t)$ of model (2.9)

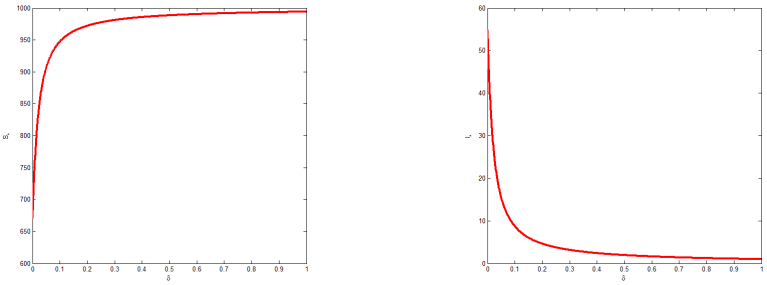
Next, in order to investigate the influence of the prevention and control efforts on the epidemic disease, we draw the graphs of S_* and I_* at the endemic equilibrium with respect to the prevention and control efforts δ. Figure 4 shows that as the prevention and control efforts δ increase, susceptible individuals increase while infected individuals decrease at the endemic equilibrium. However, the graph trajectories of infected individuals decrease more slowly as δ increases, which implies that high prevention and control efforts have little impact on reducing the number of infected individuals at the endemic equilibrium. From Figure 5, we numerically study the effect of the prevention and control efforts on the dynamics of model (2.9), when $\tau = 0$. As the prevention and control efforts δ increase, susceptible individuals increase while infected individuals decrease. The graph trajectories of infected individuals converge more quickly as δ increases, which implies that the increase of the prevention and control efforts can delay the infection progress of epidemics.

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11



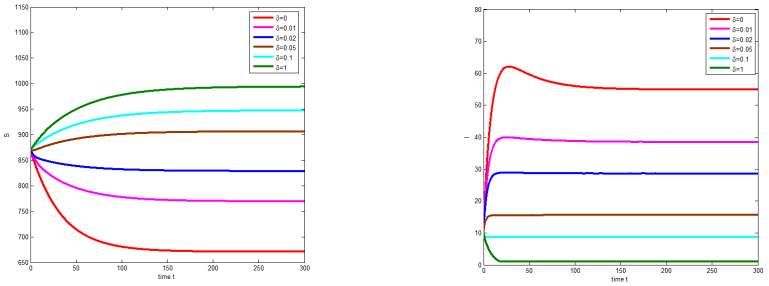
(a) (b)

Figure 3. $R_0 > 1$, $\tau = 5$: (a) the time series diagram of $S(t)$ and $I(t)$ of model (2.9); (b) the phase diagram of $S(t)$ and $I(t)$ of model (2.9)



(a) (b)

Figure 4. Graphs of the number of S_* and I_* at the endemic equilibrium with the change of prevention and control efforts δ



(a) (b)

Figure 5. Under different prevention and control efforts δ, the time series diagram of $S(t)$ and $I(t)$ of model (2.9), when $\tau = 0$

7. Conclusions

This paper considers the effects of delayed prevention and control strategies on the infection rate and proposes a delayed SIR epidemic model with the piecewise infection rate function. By analyzing the existence of the endemic equilibrium E_*, we obtain the basic reproduction number R_0. When $\tau = 0$, by investigating corresponding characteristic equations, the local stability of each of the feasible

DOI https://doi.org/10.12150/jnma.2023.524 | Generated on 2024-11-19 20:40:11

equilibria has been established. Further, the global stability of the disease-free equilibrium E_0 and the endemic equilibrium E_* has been completely established by eliminating the existence of limit cycles. The theoretical analysis results show that if $R_0 < 1$, the disease-free equilibrium E_0 is globally asymptotically stable at $\tau = 0$. In this case, the disease will fade out. If $R_0 > 1$, the endemic equilibrium E_* is globally asymptotically stable at $\tau = 0$. In this case, the disease becomes endemic. Next, we analyze the effects of time delay τ on the stability of the disease-free equilibrium E_0 and the endemic equilibrium E_*. Theorem 5.1 shows that the time delay does not change the stability of E_0. When $R_0 > 1$, the time delay can destabilize the endemic equilibrium E_* causing Hopf bifurcation at $\tau = \tau_0$, and there are periodic solutions near E_*.

The prevention and control strategies can reduce the number of infected individuals at the equilibrium point, which inhibits the spread of infectious diseases. Therefore, we suggest that the government and communities actively adopt those strategies, for example, meticulous testing and tracking, massive isolation and strict lockdown of people. In order to reduce fluctuations in the number of infected people, it is essential to ensure that control strategies can be implemented in time. Last but not least, according to the relationship between the prevention and control efforts and the number of infected individuals at the endemic equilibrium, the spread of diseases can be avoided without requiring a high level of efforts. We suggest that moderate prevention and control efforts should be recommended to balance economic development situations and control prevention strategies.

Acknowledgments

The authors thank the reviewers and editors for their valuable suggestions that help improve this paper.

References

[1] Z. Chang, X. Meng and T. Zhang, *A new way of investigating the asymptotic behaviour of a stochastic SIS system with multiplicative noise*, Applied Mathematics Letters, 2019, 87, 80–86.

[2] I. Cooper, A. Mondal and C. G. Antonopoulos, *A SIR model assumption for the spread of COVID-19 in different communities*, Chaos, Solitons & Fractals, 2020, 139, Article ID 110057, 14 pages.

[3] J. Cui, Y. Sun and H. Zhu, *The impact of media on the control of infectious diseases*, Journal of Dynamics and Differential Equations, 2008, 20(1), 31–53.

[4] Y. Dong, J. Ren and Q. Huang, *Dynamics of a toxin-mediated aquatic population model with delayed toxic responses*, Mathematical Biosciences and Engineering, 2020, 17(5), 5907–5924.

[5] A. F. Filippov, *Differential Equations with Discontinuous Righthand Sides: Control Systems*, Kluwer Academic, Amsterdam, 1988.

[6] J. K. Hale, *Theory of Functional Differential Equations*, Springer-Verlag, New York, 1997.

[7] L. Hao, G. Jiang, S. Liu and L. Ling, *Global dynamics of an SIRS epidemic model with saturation incidence*, Biosystems, 2013, 114(1), 56–63.

[8] G. He, J. Wang and G. Huang, *Threshold dynamics of an epidemic model with latency and vaccination in a heterogeneous habitat*, Journal of Nonlinear Modeling and Analysis, 2020, 2(3), 393–410.

[9] G. Huang and Y. Takeuchi, *Global analysis on delay epidemiological dynamic models with nonlinear incidence*, Journal of Mathematical Biology, 2011, 63(1), 125–139.

[10] G. Huang, Y. Takeuchi, W. Ma and D. Wei, *Global stability for delay SIR and SEIR epidemic models with nonlinear incidence rate*, Bulletin of Mathematical Biology, 2010, 72(5), 1192–1207.

[11] Q. Huang, L. Parshotam, H. Wang, C. Bampfylde and M. A. Lewis, *A model for the impact of contaminants on fish population dynamics*, Journal of Theoretical Biology, 2013, 334, 71–79.

[12] Q. Huang, G. Seo and C. Shan, *Bifurcations and global dynamics in a toxin-dependent aquatic population model*, Mathematical Biosciences, 2018, 296, 26–35.

[13] W. O. Kermack and A. G. McKendrick, *A contribution to the mathematical theory of epidemics*, Proceedings of the Royal Society Series A, 1927, 115(772), 700–721.

[14] Y. Kuang, *Delay Differential Equations: With Applications In Population Dynamics*, Academic Press, Boston, 1993.

[15] M. A. Kuddus and A. Rahman, *Analysis of COVID-19 using a modified SLIR model with nonlinear incidence*, Results in Physics, 2021, 27(2), Article ID 104478, 11 pages.

[16] J. P. LaSalle, *The Stability of Dynamical Systems*, Regional Conference Series in Applied Mathematics, Society for Industrial and Applied Mathematics, Philadelphia, 1976.

[17] Y. Liu and Y. Xiao, *An epidemic model with saturated media/psychological impact*, Applied Mathematics and Mechanics, 2013, 34(4), 399–407.

[18] M. Lu, J. Huang, S. Ruan and P. Yu, *Bifurcation analysis of an SIRS epidemic model with a generalized nonmonotone and saturated incidence rate*, Journal of Differential Equations, 2019, 267(3), 1859–1898.

[19] S. Pathak, A. Maiti and G. P. Samanta, *Rich dynamics of an SIR epidemic model*, Nonlinear Analysis: Modelling and Control, 2010, 15(1), 71–81.

[20] H. Qi, X. Leng, X. Meng and T. Zhang, *Periodic solution and ergodic stationary distribution of SEIS dynamical systems with active and latent patients*, Qualitative Theory of Dynamical Systems, 2019, 18, 347–369.

[21] Y. Rao, D. Hu and G. Huang, *Dynamical analysis of COVID-19 epidemic model with individual mobility*, Communications in Mathematical Biology and Neuroscience, 2021, 7, 18 pages.

[22] G. P. Sahu and J. Dhar, *Dynamics of an SEQIHRS epidemic model with media coverage, quarantine and isolation in a community with pre-existing immunity*, Journal of Mathematical Analysis and Applications, 2015, 421(2), 1651–1672.

[23] B. Sounvoravong and S. J. Guo, *Dynamics of a diffusive SIR epidemic model with time delay*, Journal of Nonlinear Modeling and Analysis, 2019, 1(3), 319–334.

[24] A. Wang, Y. Xiao and R. Smith, *Multiple equilibria in a non-smooth epidemic model with medical-resource constraints*, Bulletin of Mathematical Biology, 2019, 81(4), 963–994.

[25] J. Wang, G. Huang, Y. Takeuchi and S. Liu, *Sveir epidemiological model with varying infectivity and distributed delays*, Mathematical Biosciences and Engineering, 2011, 8(3), 875-888.

[26] W. Wang, *Backward bifurcation of an epidemic model with treatment*, Mathematical Biosciences, 2006, 201(1–2), 58–71.

[27] Y. Xiao, X. Xu and S. Tang, *Sliding mode control of outbreaks of emerging infectious diseases*, Bulletin of Mathematical Biology, 2012, 74(10), 2403–2422.

[28] Y. Zhang and P. Song, *Dynamics of the piecewise smooth epidemic model with nonlinear incidence*, Chaos, Solitons & Fractals, 2021, 146, Article ID 110903, 7 pages.

[29] Z. Zhang, J. Zhang and Y. Liu, *Dynamical study on a class of SIR discontinuous epidemic model with two control strategies*, Acta Mathematicae Applicatae Sinica, 2020, 43(5), 897–914.