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Role of limited medical resources in an epidemic model with media report and general birth rate

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ABSTRACT

This paper formulates an SEIRSHM epidemic model with general birth rate, media report and limited medical resources. Firstly, the well-posedness of the solutions and the extinction of the disease are discussed. Then, the existence of the endemic equilibrium is discussed and we find when $R^* > 1$ and $R_0 = 1$, there exhibits a backward bifurcation, if $R^* < 1$ and $R_0 = 1$, there exhibits a forward bifurcation. Finally, numerical simulations are carried out to illustrate the main results and show that media report and limited medical resources have a great impact on disease transmission.

1. Introduction

Throughout human history, infectious diseases have always been one of the significant factors threatening our health and survival. With the process of globalization, the speed and scope of disease transmission have greatly increased, which makes its prevention and control more complex and challenging. As one of the main tools for studying the dynamics of disease transmission, mathematical models naturally receive the favor of many researchers. For example, Hamer (Hamer, 1906) proposed a discrete-time model to explain recurrent measles epidemics. Ross (Ross, 1911) used a differential equation model to study malaria transmission between mosquitoes and humans. In 1927, Kermack and McKendrick (Kermack & McKendrick, 1927) constructed the famous SIR model for studying the Black Death and plague, and developed the famous threshold theory that distinguishes the prevalence of disease in the subsequent research (Kermack and McKendrick, 1932, 1933). Since then, more and more scholars have expanded the SIR models to multiple types, such as the time-delay models (Goel et al., 2023; Guo, He, & Cui, 2023; Luo, Zhang, Teng, & Zheng, 2021a), the reaction-diffusion models (Luo, Zhang, Teng, & Zheng, 2021b;

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Zheng, Luo, & Teng, 2023), etc. In particular, theoretical research based on mathematical modeling played a key role in the fight against the COVID-19 epidemic(see (Tang et al., 2020; Wang et al., 2020) and its references).

In fact, medical resources are one of the important factors that cannot be ignored in the process of disease prevention and control. For example, in the fight against COVID-19, Wuhan Vulcan Mountain Hospital is a grand project completed under the witness of the whole country and the world, and medical staff from all over the country have rushed to the epidemic area to support it. From the perspective of mathematical modeling, many researchers have introduced limited medical resources into mathematical models, and analyzed the impact of limited medical resources on disease transmission (Hu et al., 2022; Huang et al., 2023; Li et al., 2020; Shan & Zhu, 2014; Sylvie Diane & Farai, 2018; Wei, Xu, & Liu, 2023; Yang, Chen, Tan, Liu, & Cheke, 2023; Yu, Wan, & Zhu, 2023; Zhou & Fan, 2012). Among them, Hu et al. (Hu et al., 2022) introduced a half-saturated detection rate function to describe the effect of medical resources and showed that the supply of medical resources can influence the transmission of disease. Li et al. (Li et al., 2020) integrated the number of beds in the Square Cabin hospital into the mathematical model, and found that the investment time of the Square Cabin hospital can determine the spread trend of COVID-19 in Wuhan. Wei et al. (Wei et al., 2023) introduced a stochastic switching SIR epidemic model in their study, which focused on the impact of the limited medical treatment on disease dynamics. Their findings indicated that the increase in medical resources and improvement in supply efficiency could facilitate the transition from a persistent state to an extinct state of the epidemic. These conclusions were largely based on the analysis of parameters characterizing the influence of limited medical resources on disease outcomes. In fact, because medical resources mainly affect the medical treatment of infected people, and the number of existing inpatients and the maximum capacity of patients admitted by the hospital will determine the degree of medical treatment of infected people, therefore, this paper follows the ideas of Liu et al. (Liu, Wang, Ren, et al, 2023) to portray the impact of limited medical resources by introducing a new hospital compartment.

In addition, media coverage is also one of the important factors that cannot be ignored in the process of disease prevention and control. How to introduce media reporting factors into mathematical models to investigate their effect on disease dynamics, different scholars have different methods (Chang, Liu, Jin, & Wang, 2020; Cui, Tao, & Zhu, 2008; Huo, Yang, & Xiang, 2018; Liu, Luo, & Teng, 2023; Song & Xiao, 2022; Yan et al., 2016). To the best of our knowledge, the current research mainly includes the following two perspectives: one is to change the incidence of infectious diseases, that is, through various forms of non-linear incidence to characterize the direct or indirect impact of media reports on the incidence; the other is to describe the dynamic process of media coverage over time by introducing a separate compartment for media coverage. For instance, Cui et al. (Cui et al., 2008) studied the impact of media coverage on disease transmission by using the general incidence function $c_1 - c_2 f(I)$ as the exposure rate, and concluded that the important role of media coverage in the spread of infectious diseases. Tang et al. (Yan et al., 2016) used media reports as state variables to study the complex dynamic behavior of the model first. Huo et al. (Huo et al., 2018) showed that media can be a valuable tool in controlling the emergence and spread of epidemic diseases by proposing a new SEIS epidemic model that includes the impact of media. Chang et al. (Chang et al., 2020) analyzed the effect of media coverage on the spread of the COVID-19 epidemic by introducing an SIHRS epidemic model. They also highlighted the potential for extensive media coverage to shape public perceptions of epidemics and foster collective efforts to combat the outbreaks.

However, most of epidemic models mentioned above considered the total population size to be constant. In fact, it is unreasonable to describe a long-term disease that causes death and reduces the chance of regeneration. Hence, in recent years, researchers have proposed some models to describe disease transmission in non-constant populations (Cooke, van den Driessche, & Zou, 1999; Gao et al., 1992; Liu & Beretta, 2006; Luo et al., 2018; Zhang, Liu, & Teng, 2010). For example, Cooke et al. (Cooke et al., 1999) considered a nonlinear birth term $B(N)$, and they found that the form $B(N)N$ is significant in determining the qualitative dynamics. In the absence of disease, Zhang et al. (Zhang et al., 2010) assume that the total population size N changes according to a population growth equation $N' = B(N)N - dN$, here $d > 0$ is the death rate constant, $B < d$, and $B(N)N$ is a birth rate function. We aim to formulate and analyze an epidemic model in a varying population of size $N(t)$.

Based on the discussion above, and considering that many diseases will go through an incubation period and most people cannot obtain lifelong immunity after recovery. Therefore, the paper will study the influence of media coverage and limited treatment resources on the dynamics of infectious diseases based on the basic SEIRS model. The rest of the paper is arranged as follows. In Section 2, we formulate the model and give some preliminaries that will be used in the following sections. Section 3 is dedicated to the basic reproduction number and the extinction. Section 4 is devoted to the study of the existence of the endemic equilibrium and the backward bifurcation. In Section 5, we propose numerical simulations to illustrate our theoretical results. We end with a conclusion and some research perspectives.

2. Model formulation and preliminaries

To formulate the model, we denote the compartments susceptible, latent, infected, hospital and recovered as $S(t)$, $E(t)$, $I(t)$, $H(t)$ and $R(t)$ in that order, and the total number of individuals $N(t)$ at time t is $N(t) = S(t) + E(t) + I(t) + R(t) + H(t)$, we consider the nonlinear birth rate $B(N) = \frac{A}{N} + B, A > 0, B > 0$, where A represents a constant immigration rate and BN represents a linear birth term. We start by giving a flowchart of disease transmission(see Fig. 1).

Then the SEIRSHM model below with nonlinear birth rate, media coverage and limited treatment resources is established.

$$\begin{cases} \dfrac{dS}{dt} = \left(\dfrac{A}{N} + B\right)N - \dfrac{\beta SI}{1+qM} - dS + \delta R, \\ \dfrac{dE}{dt} = \dfrac{\beta SI}{1+qM} - (\epsilon + d)E, \\ \dfrac{dI}{dt} = \epsilon E - \gamma I\left(1 - \dfrac{H}{K}\right) - (d + \gamma_1 + d_1)I, \\ \dfrac{dR}{dt} = \gamma_1 I + \gamma_2 H - (d+\delta)R, \\ \dfrac{dH}{dt} = \gamma I\left(1 - \dfrac{H}{K}\right) - (d + \gamma_2 + d_2)H, \\ \dfrac{dM}{dt} = aI - \tau M, \\ S(0) = S_0, E(0) = E_0, I(0) = I_0, R(0) = R_0, H(0) = H_0, M(0) = M_0. \end{cases} \tag{1}$$

All parameters are positive and their meanings are shown in Table 1.

The subsequent sections of this paper will address the presence of the backward bifurcation. Before delving into this topic, we present the following Lemma 1.

Consider the system with the parameter φ as follows

$$\frac{d\mathbf{x}}{dt} = f(\mathbf{x}, \varphi), f : R^n \times R \to R^n, f \in C^2(R^n \times R), \tag{2}$$

where $\mathbf{0}$ is an equilibrium for system (1), satisfying $f(\mathbf{0}, \varphi) = \mathbf{0}$. Assume.

(A1) $A = D_x f(\mathbf{0}, 0) = (\frac{\partial f_i}{\partial x_j}(\mathbf{0}, 0))$ is the linearized matrix of system (2) at $\mathbf{x} = \mathbf{0}$, $\varphi = 0$. 0 is the simple eigenvalue of A, and the other eigenvalues of A have negative real parts;

(A2) The zero eigenvalue of A has a non-negative right eigenvector $\mathbf{w}$ and a left eigenvector $\mathbf{v}$.

Lemma 1. (Castillon-Charez & Song, 2004) *Let f_k be the k-th component of f, define*

$$a = \sum_{k,i,j=1}^{n} \mathbf{v}_k \mathbf{w}_i \mathbf{w}_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathbf{0}, 0), \quad b = \sum_{k,i=1}^{n} \mathbf{v}_k \mathbf{w}_i \frac{\partial^2 f_k}{\partial x_i \partial \varphi}(\mathbf{0}, 0),$$

then the local dynamics of system (2) at $\mathbf{x} = \mathbf{0}$ are completely determined by a, b.

1) *If $a > 0, b > 0$. When $\varphi < 0, |\varphi| \ll 1, \mathbf{x} = \mathbf{0}$ is locally asymptotically stable, and exists a positive unstable equilibrium; When $0 < \varphi \ll 1, \mathbf{x} = \mathbf{0}$ is unstable, and there is a negative and locally asymptotically stable equilibrium;*
2) *If $a < 0, b < 0$. When $\varphi < 0, |\varphi| \ll 1, \mathbf{x} = \mathbf{0}$ is unstable; When $0 < \varphi \ll 1, \mathbf{x} = \mathbf{0}$ is locally asymptotically stable, and exists a positive unstable equilibrium;*
3) *If $a > 0, b < 0$. When $\varphi < 0, |\varphi| \ll 1, \mathbf{x} = \mathbf{0}$ is unstable, and there is a negative and locally asymptotically stable equilibrium; When $0 < \varphi \ll 1, \mathbf{x} = \mathbf{0}$ is stable, and exists a positive unstable equilibrium;*
4) *If $a < 0, b > 0$. When the φ changes from negative to positive, the stability of $\mathbf{x} = \mathbf{0}$ changes from stable to unstable, and the corresponding negative unstable equilibrium becomes a positive locally asymptotically stable equilibrium.*

From the above Lemma 1, we can know that when $a > 0, b > 0$, the system will have the backward bifurcation(BB) at $\varphi = 0$. If $a < 0, b > 0$, the system has the forward bifurcation(FB) at $\varphi = 0$.

Next, we give the well-posedness of the model (1).

Table 1
The meanings of all parameters in model (1).

Param.	Meanings	Param.	Meanings
β	the infection rate with the susceptible	δ	the rate at which recovered people lose immunity
q	the cover rate of information	ϵ	the rate at which latent people become infected
γ	the rate of the infectious into the hospitalized	K	the maximum carrying capacity of the hospital
γ_1	the recovery rate of the infected	d_1	the mortality rate of the infected
γ_2	the recovery rate of the hospitalized	d_2	the mortality rate of the hospitalized
τ	the elimination rate of media coverage	a	the impact of infected quantity change on media

Theorem 1. *Assume $S_0 \geq 0, E_0 \geq 0, I_0 \geq 0, R_0 \geq 0, H_0 \geq 0, M_0 \geq 0$, then $(S(t), E(t), I(t), R(t), H(t), M(t))$ of the model (1) is non-negative for any $t > 0$ and is ultimately bounded.*

Proof. Let

$$\frac{dS}{dt} = SL_1(S, E, I, R, H, M),$$

where

$$L_1(S, E, I, R, H, M) = \frac{A}{S} + \frac{BN}{S} - \frac{\beta I}{1 + qM} - d + \frac{\delta R}{S}.$$

The expressions of right-hand sides of model (1) are continuous as well as Lipschitzian locally on R^6_+. Hence, the unique solution along with $S_0 \geq 0$ exists on $t > 0$. And then

$$S(t) = S_0 e^{\int_0^t L_1(S(s), E(s), I(s), R(s), H(s), M(s))ds} > 0.$$

Similarly, we can get $E(t) > 0$, $I(t) > 0$, $R(t) > 0$, $H(t) > 0$, $M(t) > 0$ for any $t > 0$.

In accordance with

$$\frac{dN}{dt} = \left(\frac{A}{N} + B\right)N - dN - d_1 I - d_2 H \leq A + (B - d)N,$$

then

$$\limsup_{t\to\infty} N(t) \leq \frac{A}{d - B}.$$

Therefore, there has $t_1 > 0$ such that

$$I(t) \leq \frac{A}{d - B}, \ t > t_1.$$

Hence, by the last equation of model (1), one has

$$\frac{dM}{dt} = aI - \tau M \leq \frac{Aa}{d - B} - \tau M, \ t > t_1,$$

thus

$$\limsup_{t\to\infty} M(t) \leq \frac{Aa}{(d - B)\tau}.$$

In summary, the solution of the model (1) is ultimately bounded, which completes the proof.

Building on the foregoing discussion, it can be inferred that the model (1) encompasses the following feasible range

$$\Gamma = \left\{(S(t), E(t), I(t), R(t), H(t), M(t)) \in R^6_+ : S(t) + E(t) + I(t) + R(t) + H(t) \leq \frac{A}{d - B}; 0 \leq M(t) \leq \frac{Aa}{(d - B)\tau}\right\}.$$

3. The extinction of the disease

Obviously, the model (1) exists a disease-free equilibrium $P_0 = (\frac{A}{d-B}, 0, 0, 0, 0, 0)$. Further, the basic reproduction number R_0 of model (1) is defined by using the method in (van den et al., 2002).

Let

$$F = \begin{pmatrix} 0 & \frac{\beta A}{d - B} \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} d + \epsilon & 0 \\ -\epsilon & \gamma + d + \gamma_1 + d_1 \end{pmatrix},$$

then

$$R_0 = \rho(FV^{-1}) = \frac{\beta A\epsilon}{(d+\epsilon)(\gamma+d+\gamma_1+d_1)(d-B)}.$$

Theorem 2. *If $R_0 < 1$, then P_0 is locally asymptotically stable(LAS). If $R_0 > 1$, then P_0 is unstable.*

Proof. The Jacobian matrix J at P_0 is

$$J(P_0) = \begin{pmatrix} -d & 0 & -\frac{\beta A}{d-B} & \delta & 0 & 0 \\ 0 & -(d+\epsilon) & \frac{\beta A}{d-B} & 0 & 0 & 0 \\ 0 & \epsilon & -(\gamma+d+\gamma_1+d_1) & 0 & 0 & 0 \\ 0 & 0 & \gamma_1 & -(d+\delta) & \gamma_2 & 0 \\ 0 & 0 & \gamma & 0 & -(d+\gamma_2+d_2) & 0 \\ 0 & 0 & a & 0 & 0 & -\tau \end{pmatrix}.$$

Then

$$(\lambda+d)(\lambda+d+\delta)(\lambda+d+\gamma_2+d_2)(\lambda+\tau)\left[\left(\lambda+d+\epsilon)(\lambda+\gamma+d+\gamma_1+d_1\right) - \left(-\frac{\beta A}{d-B}\right)(-\epsilon)\right] = 0.$$

Thus

$$\lambda_1 = -d,\ \lambda_4 = -(d+\delta),\ \lambda_5 = -(d+\gamma_2+d_2),\ \lambda_6 = -\tau,$$

$$\lambda_2 = \frac{-(\gamma+d+\gamma_1+d_1+d+\epsilon) - \sqrt{(\gamma+d+\gamma_1+d_1+d+\epsilon)^2 + 4(R_0-1)(d+\epsilon)(\gamma+d+\gamma_1+d_1)}}{2},$$

$$\lambda_3 = \frac{-(\gamma+d+\gamma_1+d_1+d+\epsilon) + \sqrt{(\gamma+d+\gamma_1+d_1+d+\epsilon)^2 + 4(R_0-1)(d+\epsilon)(\gamma+d+\gamma_1+d_1)}}{2}.$$

Obviously, when $R_0 < 1$, we can get $\lambda_2 < 0$, $\lambda_3 < 0$, and $\lambda_1 < 0$, $\lambda_4 < 0$, $\lambda_5 < 0$, $\lambda_6 < 0$, thus, P_0 is LAS by the Routh-Hurwitz criteria (Dejesus & Kaufman, 1987). However, when $R_0 > 1$, $\lambda_3 > 0$, which shows that P_0 is unstable. This proof is complete.

Theorem 3. *If $R_0^* = \frac{R_0(d+\gamma+\gamma_1+d_1)}{d+d_1} < 1$, then P_0 is global asymptotical stable(GAS) on Γ.*

Proof. Define

$$V = S - S_0 - S_0 \ln\frac{S}{S_0} + E + \frac{d+\epsilon}{\epsilon}(I+R+H),$$

then

$$\begin{aligned}\frac{dV}{dt} &= A + BN - dS + \delta R - \frac{S_0}{S}(A+BN) + \frac{\beta S_0 I}{1+qM} + dS_0 - \frac{S_0}{S}\delta R + \frac{d+\epsilon}{\epsilon}[-(d+d_1)I - (d+\delta)R - (d+d_2)H] \\ &\le -dS_0\left(\frac{S}{S_0} + \frac{S_0}{S} - 2\right) + \left(1 - \frac{S_0}{S}\right)\delta R + \frac{I(d+\epsilon)}{\epsilon(1+qM)}(d+d_1)(R_0^* - 1).\end{aligned}$$

Obviously, $\frac{dV}{dt} \le 0$ when $R_0^* \le 1$, $\frac{dV}{dt} = 0$ if and only if $S = S_0$, $I = 0$. Hence if $R_0^* < 1$, the maximal positively invariant set of V in $\left\{\left(S,E,I,R,H,M\right) \in \Gamma : \frac{dV}{dt} = 0\right\}$ is P_0. Thus P_0 is GAS by the LaSalle's Invariance Principle (LaSalle, 1976).

4. The endemic equilibrium and the backward bifurcation

4.1. The existence of the endemic equilibrium(EE)

In this subsection, suppose (S^*, E^*, I^*, R^*, H^*, M^*) is the positive equilibrium of model (1), then

$$\begin{cases} \left(\frac{A}{N^*}+B\right)N^* - \frac{\beta S^* I^*}{1+qM^*} - dS^* + \delta R^* = 0, \\ \frac{\beta S^* I^*}{1+qM^*} - (d+\epsilon)E^* = 0, \\ \epsilon E^* - \gamma I^*\left(1-\frac{H^*}{K}\right) - (d+\gamma_1+d_1)I^* = 0, \\ \gamma_1 I^* + \gamma_2 H^* - (d+\delta)R^* = 0, \\ \gamma I^*\left(1-\frac{H^*}{K}\right) - (d+\gamma_2+d_2)H^* = 0, \\ aI^* - \tau M^* = 0. \end{cases} \tag{3}$$

By calculating we get

$$S^* = \frac{(d+\epsilon)\left(1+\frac{aqI^*}{\tau}\right)}{\beta\epsilon}\left(d+\gamma_1+d_1+\frac{\gamma(d+\gamma_2+d_2)}{d+\gamma_2+d_2+\gamma\frac{I^*}{K}}\right),$$

$$E^* = \frac{I^*}{\epsilon}\left(d+\gamma_1+d_1+\frac{\gamma(d+\gamma_2+d_2)}{d+\gamma_2+d_2+\gamma\frac{I^*}{K}}\right),\ M^* = \frac{aI^*}{\tau},$$

$$R^* = \frac{I^*}{d+\delta}\left(\gamma_1+\frac{\gamma\gamma_2}{d+\gamma_2+d_2+\gamma\frac{I^*}{K}}\right),\ H^* = \frac{\gamma I^*}{d+\gamma_2+d_2+\gamma\frac{I^*}{K}}.$$

Substituting S^*, E^*, R^*, H^*, M^* expressions into the first equation of (3), then we get

$$A_1(I^*)^2 + B_1 I^* + C = 0, \tag{4}$$

where

$$A_1 = \frac{\gamma\{(d+\gamma_1+d_1)(d+\delta)(B-d)[\beta\tau+aq(d+\epsilon)] + \beta\tau\epsilon[(d+\delta+\gamma_1)(B-d) - d_1(d+\delta)]\}}{K\epsilon\beta\tau(d+\delta)},$$

$$B_1 = \frac{(B-d)(d+\gamma_2+d_2)(\gamma+d+\gamma_1+d_1)\left[\beta K+\frac{aqK}{\tau}(d+\epsilon)\right]}{\beta\epsilon K} + \frac{\gamma(d-B)(d+\epsilon)[R_0(d+\gamma+d_1+\gamma_1)-(d+d_1+\gamma_1)]}{\beta\epsilon K}$$
$$+\frac{B-d}{d+\delta}[\gamma\gamma_2+\gamma_1(d+\gamma_2+d_2)] + (B-d)(d+\gamma+d_2+\gamma_2) - d_1(d+d_2+\gamma_2) - d_2\gamma,$$

$$C = A(d+\gamma_2+d_2)\left(1-\frac{1}{R_0}\right).$$

Obviously, $A_1 < 0$. R_0 and C satisfy

$$R_0 > 1 \Leftrightarrow C > 0, R_0 = 1 \Leftrightarrow C = 0, R_0 < 1 \Leftrightarrow C < 0.$$

The case of the root of equation (4) is discussed below.

(i) When $R_0 > 1$, $C > 0$, equation (4) exists the only positive root.
(ii) When $R_0 = 1$, $C = 0$. If $B_1 > 0$, equation (4) must exist a positive root; if $B_1 < 0$, equation (4) does not exist the positive root.
(iii) When $R_0 < 1$, $C < 0$. Let

$$\overline{R_0} = \frac{B_1^2 + 4A_1(d+\gamma_2+d_2)(\gamma+d+\gamma_1+d_1)(d-B)\frac{d+\epsilon}{\beta\epsilon}}{4A_1(d+\gamma_2+d_2)(\gamma+d+\gamma_1+d_1)(d-B)\frac{d+\epsilon}{\beta\epsilon}}, \Delta = B_1^2 - 4A_1C.$$

Then, the following statements hold

$$\Delta = 0 \Leftrightarrow R_0 = \overline{R_0}, \Delta > 0 \Leftrightarrow R_0 > \overline{R_0}, \Delta < 0 \Leftrightarrow R_0 < \overline{R_0}.$$

Thus, when $R_0 < 1$ and $B_1 > 0$, $R_0 = \overline{R_0}$ if and only if equation (4) has one positive root; $R_0 > \overline{R_0}$ if and only if equation (4) has two positive roots; $R_0 < \overline{R_0}$ if and only if equation (4) has not any positive root. Furthermore, when $R_0 < 1$ and $B_1 < 0$, equation (4) does not exist a positive root. With the above findings, we present the following theorem.

Theorem 4. *The following statements hold:*

(1) *If* $R_0 > 1$, *the model* (1) *exists the only EE;*
(2) *If* $R_0 \leq 1$ *and* $B_1 < 0$, *the model* (1) *does not exist EE;*
(3) *If* $R_0 = 1$ *and* $B_1 > 0$, *the model* (1) *exists the only EE;*
(4) *If* $\overline{R_0} < R_0 < 1$ *and* $B_1 > 0$, *the model* (1) *exists two EE;*
(5) *If* $R_0 = \overline{R_0} < 1$ *and* $B_1 > 0$, *the model* (1) *exists the only EE;*
(6) *If* $R_0 < \overline{R_0} < 1$ *and* $B_1 > 0$, *the model* (1) *does not exist an EE.*

4.2. The backward bifurcation

In this subsection, we will use the backward bifurcation theorem in Refs (Castillon-Charez & Song, 2004) and Lemma 1 to analyze whether model (1) will exhibit a BB. Denote

$$R^* = \frac{\epsilon\delta K\tau(d+\epsilon)^2(\gamma+d+\gamma_1+d_1)(d-B)[\gamma\gamma_2+\gamma_1(d+\gamma_2+d_2)] + A\gamma^2\epsilon^2 d\tau(d+\delta)(d+\gamma_2+d_2)}{K(d+\delta)(d+\gamma_2+d_2)(d+\epsilon)^2(\gamma+d+\gamma_1+d_1)[\tau(d+\epsilon)(\gamma+d+\gamma_1+d_1)(d-B)+A\epsilon daq]}.$$

Theorem 5. *If* $R^* > 1$ *and* $R_0 = 1$, *then model* (1) *exhibits a BB; If* $R^* < 1$ *and* $R_0 = 1$, *then model* (1) *exhibits a FB.*

Proof. Choose the infectious rate β as the bifurcation parameter. Denote $x = (x_1, x_2, x_3, x_4, x_5, x_6) = (S, E, I, R, H, M)$, then the model (1) can be represented as

$$\frac{dx}{dt} = f(x, \beta),$$

where the specific form of $f(x, \beta) = (f_1(x, \beta), f_2(x, \beta), f_3(x, \beta), f_4(x, \beta), f_5(x, \beta), f_6(x, \beta))$ is determined by the right-end function of the equation in the model (1). Let

$$\beta^* = \frac{(d+\epsilon)(\gamma+d+\gamma_1+d_1)(d-B)}{A\epsilon},$$

then when $\beta = \beta^*$, $R_0 = 1$.

The linearization matrix at the (P_0, β^*) of the model (1) is

$$A = \begin{pmatrix} -d & 0 & -\frac{\beta^* A}{d-B} & \delta & 0 & 0 \\ 0 & -(d+\epsilon) & \frac{\beta^* A}{d-B} & 0 & 0 & 0 \\ 0 & \epsilon & -(\gamma+d+\gamma_1+d_1) & 0 & 0 & 0 \\ 0 & 0 & \gamma_1 & -(d+\delta) & \gamma_2 & 0 \\ 0 & 0 & \gamma & 0 & -(d+\gamma_2+d_2) & 0 \\ 0 & 0 & a & 0 & 0 & -\tau \end{pmatrix}.$$

Then

$$\lambda(\lambda+d)(\lambda+\tau)(\lambda+d+\delta)(\lambda+d+\gamma_2+d_2)(\lambda+\gamma+2d+\gamma_1+d_1+\epsilon)=0.$$

It is obviously that 0 is the simple eigenvalue, and the remaining eigenvalues all have negative real parts.

Let ω is the right eigenvector of the zero eigenvalue of A, then ω satisfies

$$\begin{pmatrix} -d & 0 & -\frac{\beta^* A}{d-B} & \delta & 0 & 0 \\ 0 & -(d+\epsilon) & \frac{\beta^* A}{d-B} & 0 & 0 & 0 \\ 0 & \epsilon & -(\gamma+d+\gamma_1+d_1) & 0 & 0 & 0 \\ 0 & 0 & \gamma_1 & -(d+\delta) & \gamma_2 & 0 \\ 0 & 0 & \gamma & 0 & -(d+\gamma_2+d_2) & 0 \\ 0 & 0 & a & 0 & 0 & -\tau \end{pmatrix} \begin{pmatrix} \omega_1 \\ \omega_2 \\ \omega_3 \\ \omega_4 \\ \omega_5 \\ \omega_6 \end{pmatrix} = 0.$$

Further

$$\omega=\left[\frac{\delta}{d}\frac{\gamma\gamma_2+\gamma_1(d+\gamma_2+d_2)}{(d+\delta)(d+\gamma_2+d_2)}-\frac{\beta^* A}{d(d-B)},\frac{\beta^* A}{(d-B)(d+\epsilon)},1,\frac{\gamma\gamma_2+\gamma_1(d+\gamma_2+d_2)}{(d+\delta)(d+\gamma_2+d_2)},\frac{\gamma}{d+\gamma_2+d_2},\frac{a}{\tau}\right]^T.$$

Similarly

$$\nu=\left[0,\frac{\beta^* A}{(d-B)(\gamma+d+\gamma_1+d_1)},1,0,0,0\right].$$

Next, based on Lemma 1, by computing the second-order partial derivatives of $f_i(x,\beta)$ $(i=1,2,3,4,5,6)$ at (P_0,β^*), one has

$$\frac{\partial^2 f_1}{\partial x_1\partial x_3}=\frac{\partial^2 f_1}{\partial x_3\partial x_1}=-\beta^*,\quad \frac{\partial^2 f_2}{\partial x_1\partial x_3}=\frac{\partial^2 f_2}{\partial x_3\partial x_1}=\beta^*,$$

$$\frac{\partial^2 f_3}{\partial x_3\partial x_5}=\frac{\partial^2 f_3}{\partial x_5\partial x_3}=\frac{\gamma}{K},\quad \frac{\partial^2 f_5}{\partial x_3\partial x_5}=\frac{\partial^2 f_5}{\partial x_5\partial x_3}=-\frac{\gamma}{K},$$

$$\frac{\partial^2 f_1}{\partial x_3\partial x_6}=\frac{\partial^2 f_1}{\partial x_6\partial x_3}=\frac{\beta^* Aq}{d-B},\quad \frac{\partial^2 f_2}{\partial x_3\partial x_6}=\frac{\partial^2 f_2}{\partial x_6\partial x_3}=-\frac{\beta^* Aq}{d-B},$$

$$\frac{\partial^2 f_1}{\partial x_3\partial\beta}=-\frac{A}{d-B},\quad \frac{\partial^2 f_2}{\partial x_3\partial\beta}=\frac{A}{d-B},$$

$$\frac{\partial^2 f_i}{\partial x_j^2}=0, i,j=1,2,3,4,5,6,\quad \frac{\partial^2 f_i}{\partial x_j\partial x_k}=0, i=4,6, j,k=1,2,3,4,5,6, j\neq k,$$

$$\frac{\partial^2 f_i}{\partial x_j\partial\beta}=0, i=1,2, j=1,2,4,5,6,\quad \frac{\partial^2 f_i}{\partial x_j\partial\beta}=0, i=3,4,5,6, j=1,2,3,4,5,6,$$

$$\frac{\partial^2 f_i}{\partial x_j\partial x_k}=0, i=1,2, j,k=1,2,3,4,5,6, j\neq k, j=1,k\neq 3; j=3,k\neq 1,$$

$$\frac{\partial^2 f_i}{\partial x_j\partial x_k}=0, i=3,5, j,k=1,2,3,4,5,6, j\neq k, j=3,k\neq 5; j=5,k\neq 3.$$

Therefore

$$a = \frac{2}{A\epsilon^3 d(d+\delta)(d+\gamma_2+d_2)K\tau}\Big\{\epsilon\delta K\tau(d+\epsilon)^2(\gamma+d+\gamma_1+d_1)(d-B)[\gamma\gamma_2+\gamma_1(d+\gamma_2+d_2)]$$
$$-K\tau(d+\delta)(d+\gamma_2+d_2)(d+\epsilon)^3(\gamma+d+\gamma_1+d_1)^2(d-B)$$
$$-A\epsilon Kd(d+\delta)(d+\gamma_2+d_2)aq(d+\epsilon)^2(\gamma+d+\gamma_1+d_1)+A\gamma^2\epsilon^2 d\tau(d+\delta)(d+\gamma_2+d_2)\Big\},$$
$$b = \frac{A(d+\epsilon)}{\epsilon(d-B)}.$$

Obviously, $b > 0$. In line with R^*, we get

$$a > 0 \Leftrightarrow R^* > 1, a < 0 \Leftrightarrow R^* < 1.$$

Hence, based on the first conclusion of Lemma 1, we can find that the model (1) exhibits a BB at $R_0 = 1$ if $R^* > 1$, and from the fourth conclusion of Lemma 1, there exhibits a FB at $R_0 = 1$ if $R^* < 1$.

5. Numerical simulations

5.1. Verification of theoretical results

Firstly, we select initial values (50, 30, 20, 0, 2, 4), (70, 50, 40, 0, 8, 10), (90, 70, 60, 0, 14, 16) and the other parameters values are chosen in Table 2, we get $R_0 = 0.8333 < 1$, and the corresponding solutions of model (1) as depicted in Fig. 2, which sustains the findings as stated in Theorem 3, that is P_0 is GAS.

To verify the conclusion (1) of Theorem 4, we choose $\beta = 0.03$, $K = 20$, $a = 0.3$, the remaining parameter values and initial values remain unchanged, then we calculate $R_0 = 3.1250 > 1$, and the corresponding solutions of model (1) as depicted in Fig. 3, which indicates that there exists an EE, and the EE is GAS.

5.2. Sensitivity analysis

In this subsection, by using MCMC, we perform a sensitivity analysis of the model parameters with respect to R_0, as shown in Fig. 4 (a). It follows from Fig. 4 (a) that R_0 is the most sensitive to the infection rate β and the numbers of recruitment A. Fig. 4 (a) indicates that a 1% decrease in the rate at which latent people exhibit infectious traits and become infected ϵ is connected with a 0.1667% decrease in R_0. Conversely, a 1% decrease in the recovery rate γ_1 of $I(t)$ is associated with a 0.05% increase in R_0 and so on. Fig. 4(b) and (c) and (d) show that when γ, γ_1 and d_1 increase, then $I(t)$ decreases, that is, the number of infected decreases when the infected are sent to the hospital for treatment and the recovery rate of the infected is increased, further, the death rate of infected can also affect the spread of the disease.

According to Figs. 5 and 6, reducing the recruitment numbers A, the birth rate B and reducing infection rate β of contact between S and I or the rate ϵ at which latent people exhibit infectious traits and become infected can postpone the peak arrival time, decrease the maximum number of infected cases, and lower the overall peak size. It suggests that minimizing the intensity of people's interaction with each other, such as restricting travel, wearing masks, and closing public places, can be effective in stopping the spread of diseases. Fig. 7 (a), (b) show that both improved media coverage q and maximum hospital capacity K for patients can reduce the number of infected people.

5.3. Backward bifurcation

From the expression of R^*, we can see that when the K gets larger, the R^* will be smaller, and the model (1) is less likely to exhibit a BB. We select the parameter values: $A = 20$, $\gamma = 0.5$, $\gamma_1 = 0.1$, $\gamma_2 = 0.3$, $d = 0.008$, $d_2 = 0.005$, $B = 0.001$, $q = 0.004$, $d_1 = 0.01$, $a = 0.002$, $\tau = 0.0001$, $\delta = 0.9$, $\epsilon = 0.1$. The K takes the values 2, 8, 10, and 42, and the corresponding R^* are 16.109978769, 4.200008881, 3.406010889, and 0.986207483, respectively, the numerical simulation yields in Fig. 8. We can

Table 2
Values of all parameters in model (1).

Param.	Value	Param.	Value	Param.	Value
a	0.5	A	5	d	0.06
B	0.01	d_1	0.2	d_2	0.1
β	0.008	γ	0.5	γ_2	0.1
γ_1	0.04	q	0.03	K	40
τ	0.2	δ	0.6	ϵ	0.3

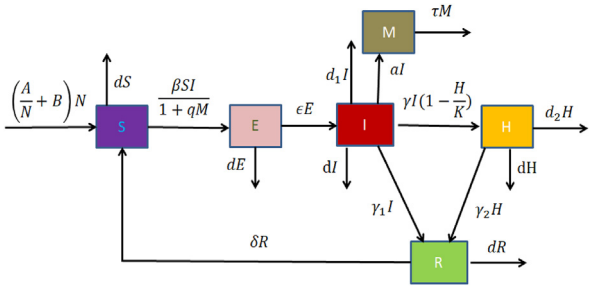

Fig. 1. The flowchart of the model.

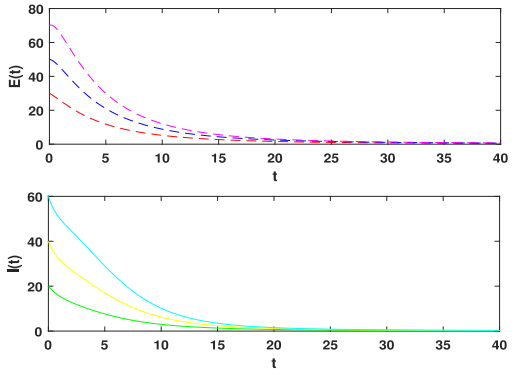

Fig. 2. The evolutionary trend of each compartment in the system as $R_0 = 0.8333 < 1$.

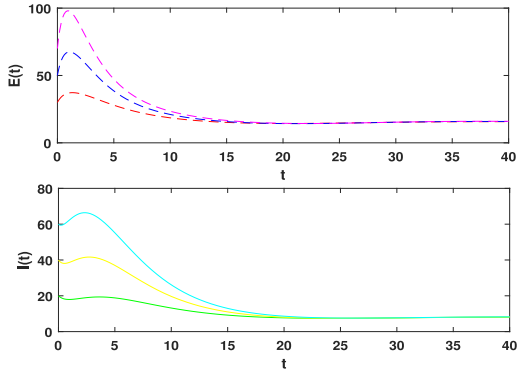

Fig. 3. The evolutionary trend of each compartment in the system as $R_0 = 3.1250 > 1$.

see that as K increases, R^* decreases, and the area where the model (1) appears to BB is smaller. When the K increases to a certain extent, $R^* < 1$, BB is lost, and FB appears, which is consistent with the results of Theorem 5.

6. Conclusion

Considering the synthetical influence of media coverage, limited medical resources and the demographic structure on the disease dynamics, this paper formulates an SEIRSHM epidemic model, where we select the nonlinear birth rate function to describe the varied population, select M as a new compartment to model the media coverage, and choose logistic growth function to reflect limited medical resources. Using the model mentioned above to study the dynamics of the epidemic models makes it possible to reflect different properties in real life. Not only the media coverage and the limited medical

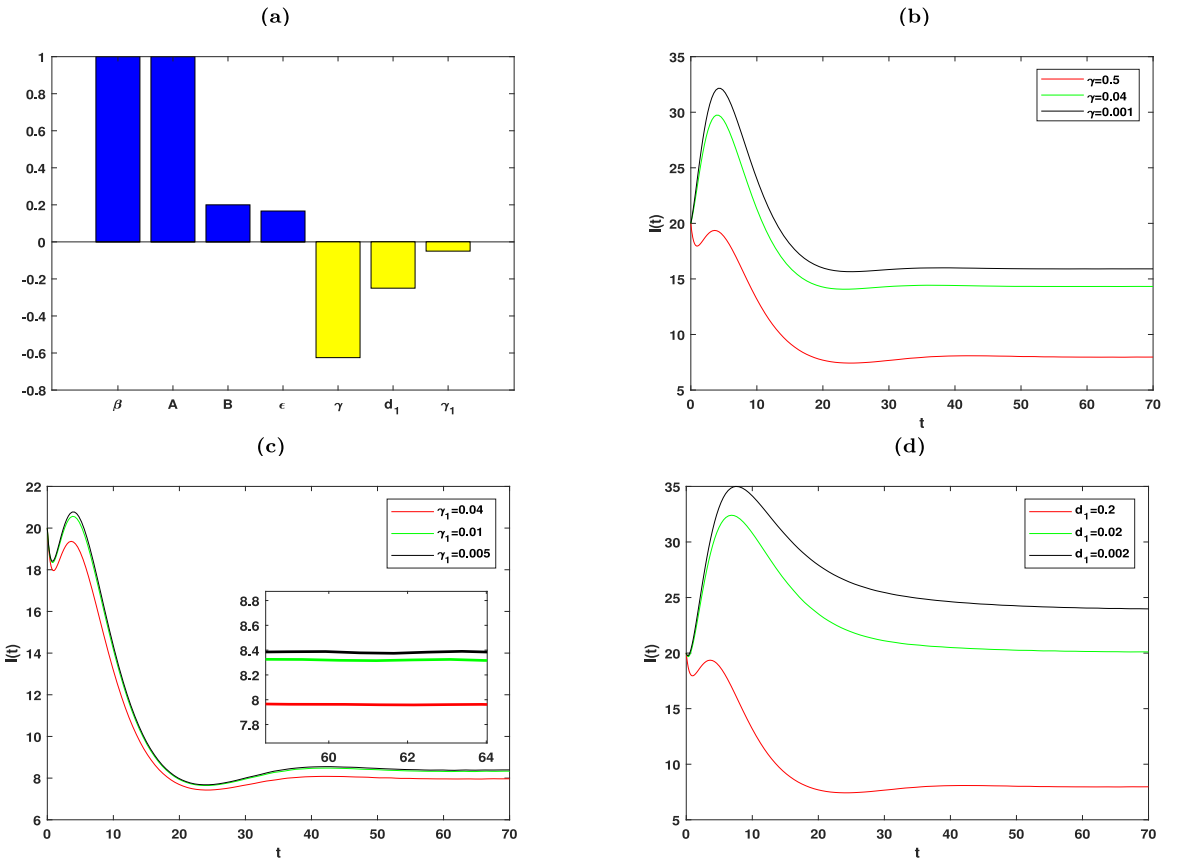

Fig. 4. Effect of changes in parameters γ, γ_1 and d_1 on the number of infected people.

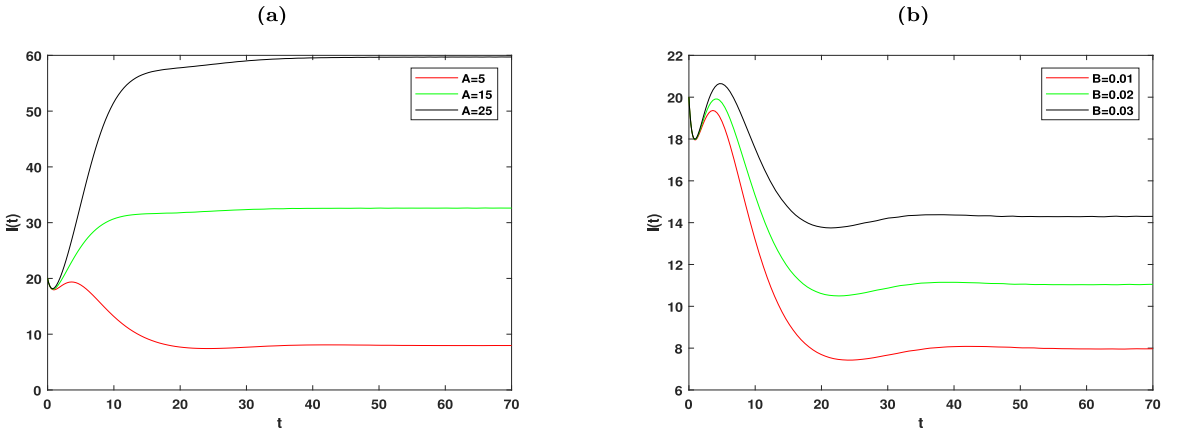

Fig. 5. Effect of changes in parameters A and B on the number of infected people.

resources covered in the model, but also the incubation period and the temporary acquired immunity are considered in the model. Nevertheless, it poses greater challenges to analyzing such systems.

First, we give the proof of the well-posedness and get the feasible domain of the model, and then, R_0 is calculated and discuss the extinction of the disease: P_0 is LAS when $R_0 < 1$, P_0 is GAS when $R_0^* < 1$, and then the existence of the EE is studied. Next, we investigate the existence of BB, and find that the threshold dynamics of the model do not depend solely on R_0: when $R^* > 1$ and $R_0 = 1$, the model will exhibit a BB. In numerical simulations, we study the effects of nonlinear birth rate, media coverage and limited treatment resources on the model respectively through sensitivity analysis. The results indicate that

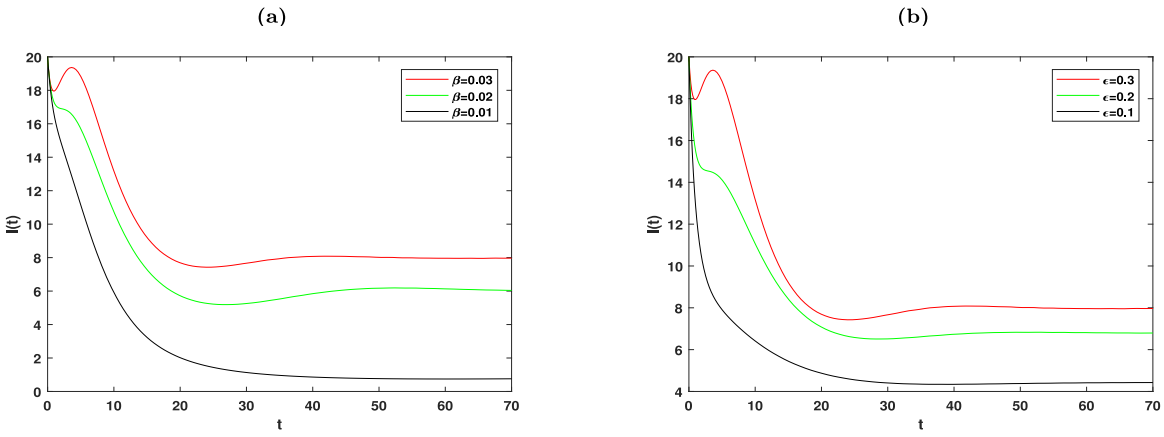

Fig. 6. Effect of changes in parameters β and ϵ on the number of infected people.

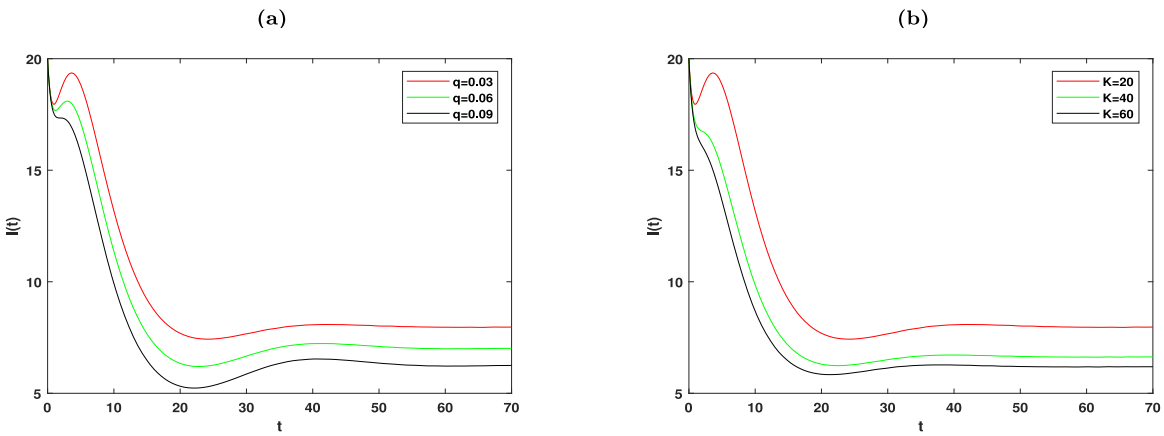

Fig. 7. Effect of changes in parameters q and K on the number of infected people.

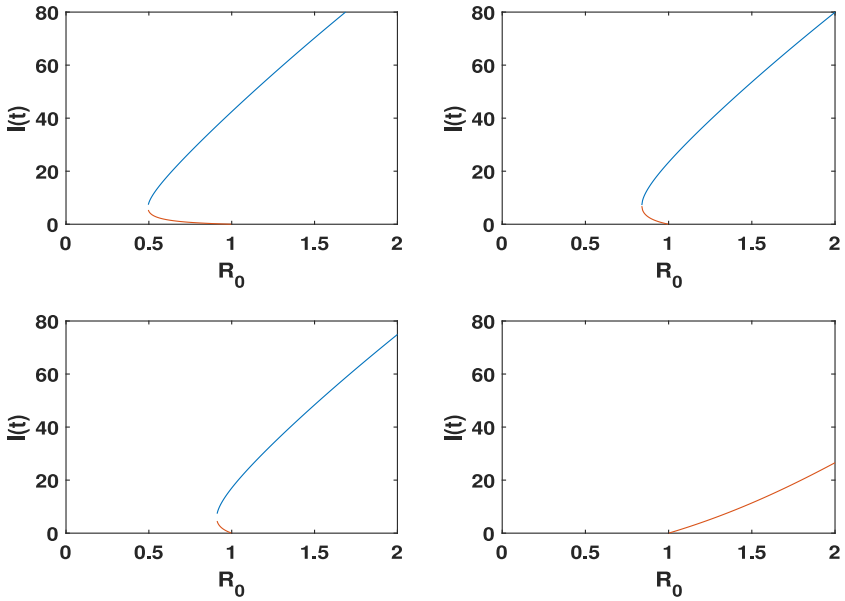

Fig. 8. The bifurcation of the model (1).

improving the media coverage and increasing medical resources can control the number of infected. In addition, changes in the birth rate can also have an impact on the spread of the disease, which accelerates when the birth rate increases.

Furthermore, there still exist several open problems, such as when proving the GAS of P_0, we can not obtain it with $R_0 < 1$, and the local and global stability of the EE is not discussed due to the high-dimension. These are all the significant work that we will take effort into in the future.

CRediT authorship contribution statement

Yicheng Hao: Writing – original draft, Software, Methodology, Conceptualization. **Yantao Luo:** Writing – review & editing, Validation, Supervision, Funding acquisition, Conceptualization. **Zhidong Teng:** Validation, Supervision, Conceptualization.

Data availability

The manuscript has no associated data.

Declaration of competing interest

The authors have no relevant financial or non-financial interests to disclose.

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