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Stability and bifurcation analysis of an SIR epidemic model with logistic growth and saturated treatment☆

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ABSTRACT

In this paper, we introduce the saturated treatment and logistic growth rate into an SIR epidemic model with bilinear incidence. The treatment function is assumed to be a continuously differential function which describes the effect of delayed treatment when the medical condition is limited and the number of infected individuals is large enough. Sufficient conditions for the existence and local stability of the disease-free and positive equilibria are established. And the existence of the stable limit cycles also is obtained. Moreover, by using the theory of bifurcations, it is shown that the model exhibits backward bifurcation, Hopf bifurcation and Bogdanov–Takens bifurcations. Finally, the numerical examples are given to illustrate the theoretical results and obtain some additional interesting phenomena, involving double stable periodic solutions and stable limit cycles.

1. Introduction

In the past decades, mathematical modeling has been playing a dramatically vital role in the theory of epidemiology. Various epidemic models have been established and investigated extensively, which leads to the huge progress in the studies of disease control and prevention (See, for example [1–8]). In classical epidemic models, it is usually assumed that the recovered rate of the infective is proportional to the number of the infective. However, every country should have a maximal capacity treatment for diseases. Therefore, it is vital to describe the limited capacity for treatment [9]. Wang and Ruan in [10], introduced the following constant treatment function of diseases into an SIR epidemic model,

$$T(I)=\begin{cases} r, & I>0,\\ 0, & I=0, \end{cases} \tag{1.1}$$

which simulated a limited capacity for treatment. Later, Wang [11] considered the piecewise linear treatment function

$$T(I)=\begin{cases} kI, & 0\le I\le I_0,\\ kI_0, & I>I_0, \end{cases} \tag{1.2}$$

where k and I_0 are positive constants. This means that the treatment rate is proportional to the number of the infective before the capacity of treatment is reached and takes the maximal capacity kI_0, otherwise. Recently, Zhang and Liu [12] introduced a continuously differentiable treatment function

$$h(I)=\frac{rI}{1+\alpha I}$$

to describe the saturation phenomenon of the limited medical resources, and proposed the following SIR epidemic model with saturated incidence rate

$$\begin{cases} \dfrac{dS}{dt}=\Lambda-\dfrac{\beta SI}{1+kI}-dS,\\ \dfrac{dI}{dt}=\dfrac{\beta SI}{1+kI}-(d+\gamma+\varepsilon)I-\dfrac{rI}{1+\alpha I},\\ \dfrac{dR}{dt}=\gamma I+\dfrac{rI}{1+\alpha I}-dR. \end{cases} \tag{1.3}$$

The dynamical behaviors, include the backward bifurcation and local stability of equilibria are studied. In [13], the authors took a deeper investigation for the above model. The sufficient conditions for the existence of backward bifurcation, as well as the existence, stability and the direction of Hopf bifurcation are established.

In [10–14], we see that models with saturated incidence rate are assumed to have a constant input of the susceptible. However, in many realistic problems the assumption of logistic growth input of the susceptible may be more reasonable for a relatively long-lasting disease or a disease with high death rate. Actually, varying total population models have been studied widely already

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(See, for example [15–17]). In this paper, we suppose that the susceptible population of a country follows logistic growth. For simplicity, we assume that newborns directly enter into the susceptible class and the infected or recovered ones do not contribute to births and deaths in susceptible class.

Thus, our SIR epidemic model is proposed in the following form.

$$\begin{cases} \dfrac{dS}{dt} = rS(1-\dfrac{S}{K}) - \beta SI, \\ \dfrac{dI}{dt} = \beta SI - (\mu+\alpha+\sigma)I - \dfrac{\lambda I}{1+\varepsilon I}, \\ \dfrac{dR}{dt} = \sigma I + \dfrac{\lambda I}{1+\varepsilon I} - \mu R, \end{cases} \tag{1.4}$$

where $S(t)$, $I(t)$ and $R(t)$ denote the numbers of the susceptible, infectious and recovery at time t, respectively. r is the intrinsic growth rate of susceptible population, K denotes the carrying capacity of the country ignoring the infection and recovered persons, β denotes the transmission rate, μ is natural death rate, α is the disease-induced death rate, σ represents the recovered rate, λ represents the maximal medical resources supplied per united time, and ε is half-saturation constant, which measures effect of being delayed for treatment. It is assumed in this paper that λ is nonnegative constant and other parameters are positive constants.

The organization of this paper is as follows. In Section 2, we analyze the existence of equilibria, backward bifurcation and the local dynamics of equilibria. In Section 3, we focus on the discussion of Hopf bifurcation at critical values. In Section 4, the existence of Bogdanov–Takens bifurcations is discussed. In Section 5, we present some numerical examples to verify our theoretical results and find some other meaningful phenomena.

2. Equilibria and local dynamics

We notice that the recovery R does not appear in the first two equations of model (1.4), thus, it is equivalent to investigate the following subsystem of model (1.4)

$$\begin{cases} \dfrac{dS}{dt} = rS(1-\dfrac{S}{K}) - \beta SI, \\ \dfrac{dI}{dt} = \beta SI - (\mu+\alpha+\sigma)I - \dfrac{\lambda I}{1+\varepsilon I}. \end{cases} \tag{2.1}$$

For the convenience, we denote $m = \mu+\alpha+\sigma$. Firstly, we make scalings: $(r', \beta', \lambda', \varepsilon') = (\frac{r}{m}, \frac{\beta K}{m}, \frac{\lambda}{m}, K\varepsilon)$ and $(x, y, \tau) = (\frac{S}{K}, \frac{I}{K}, mt)$. To avoid the abuse of mathematical notation, we still denote (r', β', λ', ε', τ) by (r, β, λ, ε, t). Then model (2.1) becomes

$$\begin{cases} \dfrac{dx}{dt} = rx(1-x) - \beta xy, \\ \dfrac{dy}{dt} = \beta xy - y - \dfrac{\lambda y}{1+\varepsilon y}. \end{cases} \tag{2.2}$$

Theorem 2.1. *All solutions $(x(t), y(t))$ of model (2.2) with initial conditions $x(0) > 0, y(0) > 0$ are positive and bounded for all $t \geq 0$.*

Proof. Let $m(t) = \min\{x(t), y(t)\}$, then $m(0) > 0$. Assume that there exists $\bar{t} > 0$ such that $m(\bar{t}) = 0$ and $m(t) > 0$ for all $t \in [0, \bar{t})$. If $m(\bar{t}) = x(\bar{t})$, from the first equation of model (2.2), we have

$$x(\bar{t}) = x(0)\exp(r(1-x(\bar{t})) - \beta y(\bar{t})) > 0,$$

which leads to a contradiction. Similarly, when $m(\bar{t}) = y(\bar{t})$, we also can obtain the contradiction. Hence, $m(t) > 0$ for all $t \geq 0$, and thereby, $(x(t), y(t))$ is positive for all $t \geq 0$.

Consider Lyapunov function $V = x + y$. Calculating the derivative of $V(x, y)$ along the solution of model (2.2), we obtain

$$\frac{dV}{dt} = rx(1-x) - y - \frac{\lambda y}{1+\varepsilon y} \leq r(1-x)x - y,$$

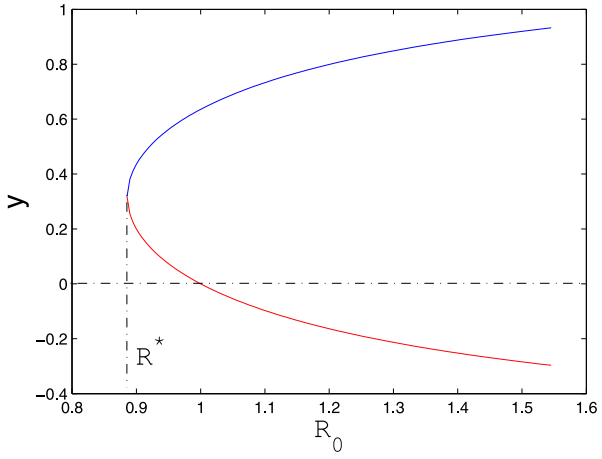

Fig. 1. The infective sizes at equilibria versus $\mathcal{R}_0$ when take $\lambda = 0.1 : 0.01 : 0.92$, $\beta = 1.7$, $\varepsilon = 3$ and $r = 4$, which satisfies condition $r > \frac{\beta^2}{\varepsilon(\beta-1)} > 0$.

then there exist constants $\delta > 0$ and $\eta > 0$ such that

$$\frac{dV}{dt} \leq \delta - \eta(x+y) = \delta - \eta V.$$

Hence, $\limsup_{t\to\infty} V \leq \frac{\delta}{\eta}$. This shows that the solution is ultimately bounded, and the theorem is proved. □

It is clear that model (2.2) always has a unique disease-free equilibrium $P_0(1, 0)$. The positive equilibria of model (2.2) can be gained by solving equations

$$rx(1-x) - \beta xy = 0, \quad \beta xy - y - \frac{\lambda y}{1+\varepsilon y} = 0, \tag{2.3}$$

which yields

$$\beta^2\varepsilon y^2 + (\beta^2 + r\varepsilon(1-\beta))y + r(1+\lambda-\beta) = 0. \tag{2.4}$$

Denote

$$\mathcal{R}_0 = \frac{\beta}{1+\lambda}.$$

From (2.4), it is clear that if $\beta \leq 1$ and $\mathcal{R}_0 < 1$, then model (2.2) has no positive equilibrium. Computing the discriminant of (2.4), we get

$$\Delta = (\beta^2 + r\varepsilon(1-\beta))^2 - 4\beta^2\varepsilon r(1+\lambda-\beta).$$

Define $\mathcal{R}^* = 1 - \frac{[\beta^2 + r\varepsilon(1-\beta)]^2}{4\beta^2\varepsilon r(1+\lambda)}$. We have that $\Delta > 0$ is equivalent to $\mathcal{R}^* < \mathcal{R}_0$. Furthermore, we also have $\Delta = \kappa^2 - 4\beta^2\varepsilon r\lambda$ with $\kappa = \beta^2 + r\varepsilon(\beta-1)$. Clearly, we can obtain the following results.

Theorem 2.2.

(1) *Model (2.2) always has a disease-free equilibrium P_0.*
(2) *If $\mathcal{R}_0 = 1$ and $r > \frac{(1+\lambda)^2}{\varepsilon\lambda}$, model (2.2) has a unique positive equilibrium $P_1(x_1, y_1)$, where $y_1 = \frac{r\varepsilon\lambda - \beta^2}{\beta^2\varepsilon}$ and $x_1 = \frac{\varepsilon y_1 + 1 + \lambda}{\beta(1+\varepsilon y_1)}$.*
(3) *Assume $r > \frac{\beta^2}{\varepsilon(\beta-1)} > 0$, then we have*
   (a) if $\mathcal{R}^* < \mathcal{R}_0 < 1$, model (2.2) has two positive equilibria $P_2(x_2, y_2)$ and $P_3(x_3, y_3)$, where $y_2 = \frac{r\varepsilon(\beta-1) - \beta^2 - \sqrt{\Delta}}{2\beta^2\varepsilon}$, $y_3 = \frac{r\varepsilon(\beta-1) - \beta^2 + \sqrt{\Delta}}{2\beta^2\varepsilon}$ and $x_i = \frac{\varepsilon y_i + 1 + \lambda}{\beta(1+\varepsilon y_i)}$, $i = 2, 3$;
   (b) if $\mathcal{R}^* = \mathcal{R}_0$, model (2.2) has a unique positive equilibrium $P_4(x_4, y_4)$, where $y_4 = \frac{r\varepsilon(\beta-1) - \beta^2}{2\beta^2\varepsilon}$ and $x_4 = \frac{\varepsilon y_4 + 1 + \lambda}{\beta(1+\varepsilon y_4)}$.
(4) *If $\mathcal{R}_0 > 1$, model (2.2) has a unique positive equilibrium $P_5(x_5, y_5)$, where $y_5 = \frac{r\varepsilon(\beta-1) - \beta^2 + \sqrt{\Delta}}{2\beta^2\varepsilon}$ and $x_5 = \frac{\varepsilon y_5 + 1 + \lambda}{\beta(1+\varepsilon y_5)}$.*
(5) *Model (2.2) has no positive equilibrium in other cases.*

On the basis of results above and Fig. 1, the following result can be obtained naturally.