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Stability of a delayed SIRS epidemic model with a nonlinear incidence rate

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ABSTRACT

In this paper, an SIRS epidemic model with a nonlinear incidence rate and a time delay is investigated. By analyzing the corresponding characteristic equations, the local stability of an endemic equilibrium and a disease-free equilibrium is discussed. By comparison arguments, it is proved that if the basic reproductive number $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable. If $R_0 > 1$, by means of an iteration technique, sufficient conditions are derived for the global asymptotic stability of the endemic equilibrium.

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1. Introduction

Let $S(t)$ represent the number of individuals who are susceptible to the disease, that is, who are not yet infected at time t; $I(t)$ represent the number of infected individuals who are infectious and are able to spread the disease by contact with susceptible individuals, and $R(t)$ represent the number of individuals who have been infected and then removed from the possibility of being infected again or of spreading at time t. In [15], Mena-Lorca and Hethcote considered the following epidemic model

$$\begin{aligned} \dot{S}(t) &= A - dS(t) - \beta S(t)I(t) + \delta R(t), \\ \dot{I}(t) &= \beta S(t)I(t) - (\gamma + \alpha + d)I(t), \\ \dot{R}(t) &= \gamma I(t) - (\delta + d)R(t), \end{aligned} \tag{1.1}$$

where A is the recruitment rate of the population, d is the natural death rate of the population, γ is the recovery rate of the infective individuals, δ is the rate at which recovered individuals lose immunity and return to the susceptible class, β is the transmission rate, α is the death rate due to disease. Model (1.1) is of SIRS type, which means that susceptible individuals become infectious, then removed with immunity after recovery from infection and then susceptible again when the temporary immunity fades away. Threshold was found in [15] to determine whether the disease dies out or approaches an endemic equilibrium. Many authors have studied different kinds of SIRS epidemic models and a significant body of work has been carried out (see, for example, [6,9,10,16,20] and the references cited therein).

Incidence rate plays an important role in the modelling of epidemic dynamics. It has been suggested by several authors that the disease transmission process may have a nonlinear incidence rate. In many epidemic models, the bilinear incidence rate βSI and the standard incidence rate $\beta SI/N$ are frequently used. The bilinear incidence rate is based on the law of mass action. It has been pointed out that for standard incidence rate, it may be a good approximation if the number of available partners is large enough and everybody could not make more contacts than is practically feasible. In fact, the infection prob-

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ability per contact is likely influenced by the number of infective individuals, because more infective individuals can increase the infection risk. For instance, during the outbreak of *SARS* in 2003, many protection measures and control policies were taken by the Chinese government such as closing schools, closing restaurants, postponing conferences, isolating infectives, etc. These actions greatly reduced the contact number per unit time. In [11,12], Liu et al. proposed the incidence rate $\beta I^p S^q$ and discovered that an SIR model can yield rich dynamics such as bistable equilibria, saddle-node bifurcation and Hopf bifurcation, etc. In [4], Capasso and Serio introduced a saturated incidence rate $g(I)S$ into epidemic models, where $g(I)$ tends to a saturation level when I gets large, i.e.,

$$g(I) = \frac{\beta I}{1+\alpha I},$$

where βI measures the infection force of the disease and $1/(1+\alpha I)$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals. This incidence rate seems more reasonable than the bilinear incidence rate βIS, because it includes the behavioral change and crowding effect of the infective individuals and prevents the unboundedness of the contact rate by choosing suitable parameters.

In this paper, we are concerned with the effect of time delay and a nonlinear incidence rate on the dynamics of an SIRS epidemic model. To this end, we study the following delay differential equations

$$\begin{aligned}
\dot{S}(t) &= A - dS(t) - \frac{\beta S(t) I(t-\tau)}{1+\alpha I(t-\tau)} + \delta R(t),\\
\dot{I}(t) &= \frac{\beta S(t) I(t-\tau)}{1+\alpha I(t-\tau)} - (d+\gamma) I(t),\\
\dot{R}(t) &= \gamma I(t) - (d+\delta) R(t),
\end{aligned} \tag{1.2}$$

where $\tau > 0$ is a fixed time during which the infectious agents develop in the vector and it is only after that time that the infected vector can infect a susceptible human (see, for example, [1–3,5,13,14,18,19].

The initial conditions for system (1.2) take the form

$$\begin{aligned}
&S(\theta) = \phi_1(\theta), I(\theta) = \phi_2(\theta), R(\theta) = \phi_3(\theta),\\
&\phi_i(\theta) \geqslant 0, \theta \in [-\tau, 0], \phi_i(0) > 0 \ (i = 1,2,3),
\end{aligned} \tag{1.3}$$

where $(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in C([-\tau, 0], \mathbb{R}^3_{+0})$, the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^3_{+0}$, here $\mathbb{R}^3_{+0} = \{(x_1, x_2, x_3) : x_i \geqslant 0, i = 1,2,3\}$.

It is well known by the fundamental theory of functional differential equations [7], system (1.2) has a unique solution $(S(t), I(t), R(t))$ satisfying the initial conditions (1.3). It is easy to show that all solutions of system (1.2) with initial conditions (1.3) are defined on $[0, +\infty)$ and remain positive for all $t \geqslant 0$.

The organization of this paper is as follows. In the next section, by analyzing the corresponding characteristic equations, the local stability of a disease-free equilibrium and an endemic equilibrium is discussed. In Section 3, if the reproductive number $R_0 > 1$, by means of an iteration technique, sufficient conditions are derived for the global asymptotic stability of the endemic equilibrium. By comparison arguments, we prove that if the basic reproductive number $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable. A brief discussion is given in Section 4 to conclude this work.

2. Local stability

In this section, we discuss the local stability of an endemic equilibrium and a disease-free equilibrium of system (1.2) by analyzing the corresponding characteristic equations, respectively.

System (1.2) always has a disease-free equilibrium $E_1(A/d, 0, 0)$. Further, if $A\beta > d(d+\gamma)$, system (1.2) admits a unique endemic equilibrium $E^*(S^*, I^*, R^*)$, where

$$S^* = \frac{(d+\gamma)[A\alpha(d+\delta) + d(d+\gamma+\delta)]}{d[\alpha(d+\gamma)(d+\delta) + \beta(d+\gamma+\delta)]}, \quad I^* = \frac{\beta S^* - d - \gamma}{\alpha(d+\gamma)}, \quad R^* = \frac{\gamma}{d+\delta} I^*. \tag{2.1}$$

Let

$$R_0 = \frac{A\beta}{d(d+\gamma)}.$$

R_0 is called the basic reproductive number of system (1.2). It is easy to show that if $R_0 > 1$, the endemic equilibrium E^* exists; if $R_0 < 1$, E^* is not feasible.

The characteristic equation of system (1.2) at the endemic equilibrium E^* is of the form

$$(\lambda + d)[\lambda^2 + p_1\lambda + p_0 + (q_1\lambda + q_0)e^{-\lambda\tau}] = 0, \tag{2.2}$$

where

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