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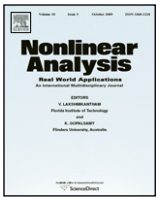

Stability analysis of a stage structured SIS model with general incidence rate☆

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ABSTRACT

A stage structured SIS model with general incidence rate is considered in this paper. The basic reproductive number R_0 for determining whether a disease is extinct or not is found. Furthermore, we obtain the global asymptotic stability of the disease-free equilibrium and the endemic equilibrium. A numerical simulation is given to illustrate the application of the results and the epidemiological significance.

1. Introduction

Every year, millions of people around the world suffer from or die of various infectious diseases. Accordingly, many attempts have been made to develop realistic mathematical models for investigating the transmission dynamics and the asymptotic behaviors of epidemiological models. How to control or eliminate disease spreading is a subject of continuing interest to both theoreticians and empiricists.

Some infections such as measles are caused by a virus. A person who suffers a viral infection either dies or develops antibodies and recovers, and is then usually immune to the disease. Even so, for many diseases an infected individual can recover and return to the susceptible state and later become reinfected. The common cold is one of the diseases that most of us get repeatedly. A SIS model is of significance in describing the dynamic properties of some kinds of epidemics and has been studied extensively (see [1–5]). In many epidemiological models, it is assumed that the infection rate is invariant in all stages. But in fact, some epidemics are age-associated, with their affects and prevalence increasing with advancing age, such as Alzheimer's disease. Since the properties of many epidemics may be variable, their transmission abilities change gradually in different stages; their infectivity usually depends on the parasite or viral loads in infected individuals or vectors. For example, measles and varicella always occur in immaturity, while typhus, schistosomiasis cutaneous, diphtheria and sexually transmitted diseases always break out in maturity. Fatal dengue haemorrhagic fever (DHF) and dengue shock syndrome (DSS) occur in adults and in primary dengue infection. Some diseases such as hepatitis B and schistosomiasis usually have a longer period of infection; their infecting force may be different for various stages in progression and most of the individuals may be infected repeatedly. Consequently, incorporating stage structure into SIS models seems to be important and more realistic in population dynamics [6,7,1].

The incidence of a disease is the number density of new cases per unit time. Standard epidemiological models use a double-bilinear incidence rate βSI based on the law of mass action [1,8]. Since the number of susceptible individuals is

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large, it is unreasonable to consider the bilinear incidence rate within a certain limited time. If the population is saturated with infective, a nonlinear incidence rate $\beta I^p S^q$ (see [9–11]) and a saturated incidence rate $\frac{\beta SI}{1+\alpha S}$ (see [1,12]) are always used in epidemiological models.

Motivated by the recent work of Aiello, Freedman and Wu [7], in the present article, we consider an age stage structured SIS model with general incidence rate $aU(S)I$, where a is the probability in unit time of transmitting infection between two individuals taking apart in every contact. Note that the incidence rates $\beta I^p S^q$ for $p = 1$ and $\frac{\beta SI}{1+\alpha S}$ mentioned in the last paragraph can be included in the general form $aU(S)I$. We believe that the introduction of the incidence rate $aU(S)I$ is efficient for including more situations with different incidence rates. To the best of our knowledge, this is the first time that this model has been investigated, and also this is the reason for the novelty of our results.

This article is organized as follows. In Section 2, we give a basic model (2.1) and some preliminary lemmas. We discuss the basic reproductive number and the dynamic properties of the model in Section 3. In Section 4, we give a numerical simulation. We end the paper with a brief discussion in Section 5.

2. The model and preliminaries

Let $x_1(t), x_2(t)$ and $y(t)$ denote the number densities of juvenile, mature and infectious individuals at time t respectively. We consider the following model:

$$\begin{cases} \dot{x}_1(t) = \alpha x_2(t) - \gamma x_1(t) - \alpha e^{-\gamma\tau} x_2(t-\tau), \\ \dot{x}_2(t) = \alpha e^{-\gamma\tau} x_2(t-\tau) - \beta x_2^2(t) - aU(x_2(t))y(t) + ry(t), \\ \dot{y}(t) = aU(x_2(t))y(t) - dy(t) - ry(t), \end{cases} \tag{2.1}$$

where $\alpha, \gamma, \tau, \beta, a, r$ and d are positive constants. It is assumed that only the mature individuals are susceptible; the birth rate at any time t is proportional to the number of adults at that time via a constant coefficient α. The delay τ is the time taken from birth to maturity for an individual. $\alpha e^{-\gamma\tau} x_2(t-\tau)$ represents the rate of recruitment into the adult individuals. This rate is essentially the birth rate τ time units ago, discounted by the factor $e^{-\gamma\tau}$ which accounts for mortality during the juvenile phase. γ measures the juvenile death rate while β measures the adult death rate. $aU(x_2(t))$ is the function of infection of susceptible population by a single infectious individual. d is the rate of death from infections and r is the cure rate.

For ecological reasons, we shall discuss the solutions of (2.1) with the following initial conditions:

$$\begin{aligned} &x_1(\theta) = \varphi_1(\theta), \quad x_2(\theta) = \varphi_2(\theta), \quad y(\theta) = \varphi_3(\theta), \quad \theta \in [-\tau, 0]; \\ &(\varphi_1, \varphi_2, \varphi_3) \in C([-\tau, 0], R_+^3), \quad R_+^3 =: \{(x_1, x_2, y) : x_1 \geq 0, x_2 \geq 0, y \geq 0\}; \\ &\varphi_i(0) > 0, \quad i = 2, 3, \quad \varphi_1(0) = \int_{-\tau}^{0} \alpha e^{\gamma s} \varphi_2(s) ds. \end{aligned} \tag{2.2}$$

The following assumption is needed in what follows: $U(S)$ is continuous and nonnegative on $[0, \infty)$ and

$$U(0) = 0, \quad \frac{dU(S)}{dS} > 0 \quad \text{for } S \in \left(0, \frac{\alpha}{\beta} e^{-\gamma\tau}\right), \quad \lim_{s\to\infty} U(S) = U(\infty). \tag{2.3}$$

We also note that the last two equations in (2.1) are in fact decoupled from the first equation, and the behavior of $x_1(t)$ is determined by a single linear equation with a non-homogeneous term once $x_2(t)$ is known. Consequently, for some discussion in the next section, our focus will be on the last two equations of (2.1):

$$\begin{cases} \dot{x}_2(t) = \alpha e^{-\gamma\tau} x_2(t-\tau) - \beta x_2^2(t) - aU(x_2(t))y(t) + ry(t), \\ \dot{y}(t) = aU(x_2(t))y(t) - dy(t) - ry(t). \end{cases} \tag{2.4}$$

Lemma 2.1. *The solutions of (2.1)–(2.2) are positive and eventually bounded.*

Proof. Firstly, we obtain, for $t \geq 0$, $y(t) = \varphi_3(0) e^{\int_0^t [aU(x_2(s)) - d - r] ds} > 0$. We shall prove that $x_2(t) > 0$ for $t \geq 0$. Suppose that there exists a $t_0 > 0$ such that $x_2(t_0) = 0$ and $\dot{x}_2(t_0) \leq 0$. Then from (2.4), it follows that

$$\dot{x}_2(t_0) = \alpha e^{-\gamma\tau} x_2(t_0 - \tau) + cy(t_0) > 0.$$

This is a contradiction. Thus, $x_2(t) > 0$ for $t \geq 0$.

Consider

$$\dot{z}(t) = -\gamma z(t) - \alpha e^{-\gamma\tau} \varphi_2(t-\tau), \quad z(0) = \varphi_1(0). \tag{2.5}$$