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Optimal vaccination and treatment of an epidemic network model

Lijuan Chen [a,b], Jitao Sun [a,*]

[a] Department of Mathematics, Tongji University, Shanghai 200092, PR China
[b] College of Mathematics and Computer Science, Fuzhou University, Fuzhou, Fujian 350002, PR China

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ABSTRACT

In this Letter, we firstly propose an epidemic network model incorporating two controls which are vaccination and treatment. For the constant controls, by using Lyapunov function, global stability of the disease-free equilibrium and the endemic equilibrium of the model is investigated. For the non-constant controls, by using the optimal control strategy, we discuss an optimal strategy to minimize the total number of the infected and the cost associated with vaccination and treatment. Table 1 and Figs. 1–5 are presented to show the global stability and the efficiency of this optimal control.

1. Introduction

In recent years, the main aim of researchers is to analyze and predict the consequences of the strategies designed to control infections disease. Mathematical models have become important tools in analyzing the spread and control of infectious disease. It's well known that vaccination and treatment are two important factors for preventing and controlling the epidemic outbreak. In order to study the role of these two controls, researchers have proposed various mathematical models such as the conventional models, delayed models, impulsive models and stochastic models (see [1–9] and the references therein). These models include SIS models [1,2], SIR models [3,4], SIRS models [5,6] and some other models [7–9].

However, we notice that most of the above mentioned literatures emphasize the qualitative analysis such as seeking the so-called basic reproduction number and discussing the existence and the stability of equilibria and periodic orbits. Actually, another important way to control epidemic outbreak is the optimal control theory, which pays attention to define a strategy to control the disease and obtain the best possible result. It's well known that there is a great variety of epidemic models and problems which can be treated with optimal control theory. For example, west Nile virus, tuberculosis, avian influenza, rabies, and so on. In [10], Zaman et al. found an optimal vaccination regime for the SIR model with the percentage of a vaccinated individuals. In 2011, Kar et al. [11] focused on the study of a nonlinear mathematical SIR epidemic model with a vaccination. The authors of Ref. [12] discussed an SVI (Susceptibles-Vaccinated-Infectious) epidemic model with treatment and found the optimal strategy to minimize both the disease burden and the intervention costs. In this Letter, we consider the following SIRS epidemic model with vaccination and treatment

$$\begin{cases} \dfrac{dS(t)}{dt} = -\lambda S(t)I(t) + \gamma R(t) - u_1 S(t) + \omega u_2 I(t), \\ \dfrac{dI(t)}{dt} = \lambda S(t)I(t) - \delta I(t) - u_2 I(t), \\ \dfrac{dR(t)}{dt} = \delta I(t) - \gamma R(t) + u_1 S(t) + (1-\omega)u_2 I(t), \end{cases} \tag{1.1}$$

* Corresponding author. Tel.: +86 21 65983241x1307; fax: +86 21 65981985.
E-mail address: sunjt@sh163.net (J. Sun).

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where the parameters λ, δ, γ and u_1, u_2 are positive constants in which λ is the transmission rate when susceptible individuals contact with infectious, δ is the natural recovered rate from infection and γ is the rate when recovered individuals move into the susceptible once more. Differently from the traditional SIRS epidemic model, (1.1) incorporates two kinds of controls described by $u_1 S(t)$ and $u_2 I(t)$. Here u_1 means the percentage of vaccination given to susceptible individuals at time t. We assume that vaccination is effective and the vaccinated become recovered. Also, u_2 is the percentage of treatment given to infectious at time t. We suppose that only $\omega u_2 I(t)$ would become susceptible at time t due to the limitation of treatment condition, whereas there are $(1-\omega)u_2 I(t)$ would become recovered at time t, and $\omega \in [0, 1]$.

As we know, most traditional epidemic models suppose that individuals mix uniformly and all individuals have the same rates of disease-causing contacts. This over-simplified assumption makes the analysis tractable but not realistic. Actually, the interpersonal contact patterns underlying disease transmission can be thought of expanding a complex network, where relations (edges) join individuals (nodes) who interact with each other. The connectivity k of a node is defined as the number of links connected to the node. The degree distribution of a network $p(k)$ is defined as the probability of a randomly chosen node to have a degree k. Based on the above, many epidemic models on complex networks (see [13–21]), were studied. For the mechanism of the spreading of epidemic on complex networks, different researcher gave different explanations. Many networks relevant to the epidemic spreading are heterogeneous, including the BA scale-free network [21], which the degree distribution follows a power law $p(k) \sim k^{-\sigma}$. However, to the best of the authors' knowledge, until this day, seldom did scholars consider the following SIRS epidemic studied (1.1) on complex networks. Motivated by the above, in this Letter, we firstly propose the following epidemic network model:

$$\begin{cases} \dfrac{dS_k(t)}{dt} = -\lambda k S_k(t)\Theta(t) + \gamma R_k(t) - u_1 S_k(t) + \omega u_2 I_k(t), \\ \dfrac{dI_k(t)}{dt} = \lambda k S_k(t)\Theta(t) - \delta I_k(t) - u_2 I_k(t), \\ \dfrac{dR_k(t)}{dt} = \delta I_k(t) - \gamma R_k(t) + u_1 S_k(t) + (1-\omega)u_2 I_k(t), \quad k = 1, 2, \ldots, n, \end{cases} \tag{1.2}$$

where the parameters λ, γ, δ, u_1, u_2 and ω have the same biological meaning as those in (1.1). k describes the degree of the complex networks. In this section, it is assumed that the connectivity of nodes on the network is uncorrelated, thus, we have $\Theta(t) = \frac{1}{\langle k\rangle}\sum_{k=1}^{n} kp(k)I_k(t)$ where $\langle k\rangle = \sum_{k=1}^{n} kp(k)$ is the average degree of the network and $p(k)$ is the connectivity distribution.

On the other hand, nowadays people pay much attention to prevent or reduce the spread of infectious diseases on networks. In [22], the authors studied an epidemic model including susceptible, infected and imperfectly vaccinated compartments on several kinds of networks. The epidemic threshold and prevalence are analyzed. In [23], the authors considered a variant of SIS model defined on scale-free metapopulation networks, wherein the curing rate in a node with degree k is proportional to k^{α}. An optimal control strategy to suppress epidemic explosion is studied. Both of [22] and [23] only present the epidemic threshold and don't show the stability of the epidemic-free equilibrium and the unique positive equilibrium. In [24], the authors study the global stability and optimal control of an SIRS epidemic model on heterogeneous networks. But we notice that they discuss only one kind of control, i.e., vaccination, and neglect the other kind of control: treatment. Motivated by the above, in this paper, we shall study the global stability of the system (1.2). Also we shall show that an optimal control exists for the control problem when the vaccinated percentage u_1 and the treated percentage u_2 are considered as two continuous functions on time t. Because of incorporating two controls, the proposed model in this Letter is more reasonable than that of [24]. The results we obtained improve and supplement those of [24].

From the view point of biology, we only need to focus our discussion on the positive solution of system (1.2). So it is assumed that the initial conditions of (1.2) are of the form

$$S_k(0) > 0, \qquad I_k(0) > 0, \qquad R_k(0) > 0, \quad k = 1, 2, \ldots, n. \tag{1.3}$$

One can easily show that the solution of (1.2) with the initial condition (1.3) are defined for all $t > 0$.

The organization of this Letter is as follows. In Section 2, for (1.2), by using Lyapunov function, we obtain the sufficient conditions which ensure the global attractivity of the epidemic-free equilibrium and the unique positive equilibrium of the system. In Section 3, the analysis of optimization problem is presented. In Section 4, numerical simulations are presented to illustrate the feasibility of our main results. In the last section, we give a brief discussion.

2. Global stability of (1.2)

In this section, we shall study the global stability of (1.2) with the initial conditions (1.3). From (1.2), we have $\frac{d}{dt}(S_k(t) + I_k(t) + R_k(t)) = 0$, $k = 1, 2, \ldots, n$. By simple probabilistic reasoning, we suppose that

$$S_k(t) + I_k(t) + R_k(t) = 1, \quad k = 1, 2, \ldots, n. \tag{2.1}$$

In other words, we only consider a network of identical populations in this Letter. In order to investigate the global stability of (1.2), we only need to study the global stability of the following system:

$$\begin{cases} \dfrac{dS_k(t)}{dt} = -\lambda k S_k(t)\Theta(t) + \gamma\big(1 - S_k(t) - I_k(t)\big) - u_1 S_k(t) + \omega u_2 I_k(t), \\ \dfrac{dI_k(t)}{dt} = \lambda k S_k(t)\Theta(t) - \delta I_k(t) - u_2 I_k(t), \quad k = 1, 2, \ldots, n. \end{cases} \tag{2.2}$$