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Optimal treatment of an SIR epidemic model with time delay

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1. Introduction

Mathematical modeling of population dynamics is a fast growing research area, which has been playing important roles in discovering the relation between species and their interaction. The basic and important concern for mathematical models in epidemiology is qualitative analysis; the persistence, permanence, asymptotic stability, and the existence and uniqueness for the models. Many influential results related in these topics have been established and can be found in many articles and books. An epidemic model for the spread of infectious disease was first introduced by Kermack and Mckendrick (1927). In their model the populations are subdivided into three classes; the susceptible, infected, and recovered populations. They assumed that susceptible populations in a fixed total population become infected by contact with infected individuals, infected individuals either die or recover at a constant rate. The model consists of three ordinary differential equations (ODEs) which represent the rate of change in their respective population.

Extensions of the Kermack–McKendrick model to populations in which the individuals have mobility in an environment have also

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ABSTRACT

In this paper the optimal control strategies of an *SIR* (susceptible–infected–recovered) epidemic model with time delay are introduced. In order to do this, we consider an optimally controlled *SIR* epidemic model with time delay where a control means treatment for infectious hosts. We use optimal control approach to minimize the probability that the infected individuals spread and to maximize the total number of susceptible and recovered individuals. We first derive the basic reproduction number and investigate the dynamical behavior of the controlled *SIR* epidemic model. We also show the existence of an optimal control for the control system and present numerical simulations on real data regarding the course of Ebola virus in Congo. Our results indicate that a small contact rate(probability of infection) is suitable for eradication of the disease (Ebola virus) and this is one way of optimal treatment strategies for infectious hosts.

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been studied. In the classical epidemiological model (Brauer and Castillo-Chavez, 2001), a population of total size (*N*) was divided into susceptible individuals (*S*), infected individuals (*I*), and recovered individuals (*R*). The relation between these three categories leads to the classical *SIR* model. Several epidemic models on theoretical developments are given in Milner and Pugliese (1999), Linda and Amy (2000), Tuckwell and Toubiana (2007) and Zaman et al. (2007, 2008).

In recent years, some mathematical models incorporating delayed effects have been studied. Smith in (1994) and Smith and Thieme in (1990) derived a scalar delayed differential equation for the population with immature and mature age classes. The maturation period was regarded as a time delay. Using the same idea, a system of delayed differential equations for mature population in a patchy environment has been proposed in So et al. (2001). More recent studies consider an epidemic model with density dependence to describe disease transmission in variable population size, which can be found in (see, Cooke et al., 1999; Hethcote and van den Driessche, 2000; Ma et al., 2003; Bachar and Dorfmayr, 2004). Takeuchi et al. (2000) and Ma et al. (2002) studied the SIR infectious disease model in which an infectious disease is transmitted by a vector after an incubation time. In their models, they assumed that the birth and the death rate are all constant so the dynamics of the total population may be simple. In order to investigate population dynamics for the model with more biological meanings, it should be considered that birth and death rates are density dependent. In



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this point, an *SIR* epidemic model with density dependent birth and death rates with the incubation time was formulated by Yoshida and Hara (2007). They analyzed transmission dynamics for the epidemic *SIR* model with time delay and studied the global stability of the *SIR* model. On the other hand, recently, Zaman et al. (2008) studied the stability and optimal vaccination of a controlled *SIR* epidemic model without time delays. In this project we are interested in combining and improving on the results in Zaman et al. (2008) and Yoshida and Hara (2007).

In this paper, we consider a controlled SIR epidemic model with time delay to prevent the spread of diseases by using optimal treatment strategies. In order to do this, we first introduce a control variable representing the optimal treatment for infectious hosts and set an optimal control system for the SIR epidemic model. Then we derive the basic reproduction number and investigate the dynamical behavior of the controlled SIR epidemic model. Moreover, we show the existence of an optimal control for this control problem and the infection in a community dies out by using the possible optimal control treatment. We also analyze the optimal control and optimality system using optimal control techniques. For numerical simulation, we fit data from Ebola a hemorrhagic fever outbreaks Congo (1995), where we show that the basic reproduction number is less than unity, so the infection in the community dies out by using control treatment strategy. Furthermore, our optimal control strategies reduce infected individuals and increase the total number of susceptible and recovered individuals. From these results, our optimal control system can be used by epidemic researchers to realistically simulate the stochastic dynamics of Ebola epidemics in order to study the effect of control intervention.

The paper is organized as follows. In Section 2, a description of an *SIR* epidemic model and the corresponding objective functional are given. We derive the basic reproduction number for the control system and show the existence. Then, we introduce the optimal control techniques to find the optimal solution of the dynamics system. We report our numerical results obtain from real data and analyze in detail the dynamical behaviors of the control processes in Section 3. Finally, we describe some conclusions in Section 4.

2. Optimal Control Techniques in Delay Model

To begin the optimal control procedure, it is necessary to have a model which describes the population dynamics. Yoshida and Hara (2007) considered an *SIR* model with time delay. We use this epidemic model to set our control problem. We may have a population which lacks social structures and where individuals may switch between the Susceptible, Infected, and Recovered (or immune) states according to $S \rightarrow I \rightarrow R$. We assume that all newborns are susceptible and the number of the total population is denoted by N(t) = S(t) + I(t) + R(t). Then the delayed *SIR* epidemic model which is proposed in Yoshida and Hara (2007) becomes a system of differential equations with time delay:

$$\frac{dS(t)}{dt} = \left(b - \mu \frac{rN(t)}{K}\right) N(t) - \frac{\beta S(t)I(t-h)}{N(t-h)} - \left(d + (1-\mu)\frac{rN(t)}{K}\right) S(t),$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t-h)}{N(t-h)} - \left(d + (1-\mu)\frac{rN(t)}{K}\right) I(t) - \alpha I(t),$$

$$\frac{dR(t)}{dt} = \alpha I(t) - \left(d + (1-\mu)\frac{rN(t)}{K}\right) R(t),$$
(1)

where b > 0, d > 0, $\alpha > 0$ and $\beta > 0$ are the birth, death, recovery and contact rate, respectively. r = b - d is the intrinsic growth rate, μ is the convex combination constant, K is the carrying capacity of the population, and h is a non-negative constant which represents a time delay on the infected individuals I and the total individuals N during the spread of diseases. Susceptible individuals acquire infection at a per capita rate $\beta I(t - h)/N(t - h)$. In this model, the incidence rate is $\beta S(t)I(t - h)/N(t - h)$. This incidence rate seems more reasonable than $\beta I(t)/N(t)$ because the force of infection is proportional to I(t - h)/N(t - h) with time delay. Note that in some epidemic models, bilinear incidence rate $\beta S(t)I(t)$ and standard incidence rate $\beta S(t)I(t)/N(t)$ are frequently used. These incidences imply that the contact rate or contact number is constant. Actually, the infection probability per contact is likely influenced by the number of infected individual because more infected individuals can increase infection risk. For instance, during SARS outbreak in 2003, Chinese government did a lot of protection measures and control polices: closing schools, closing restaurants, postponing conferences, isolating infectious etc. These actions greatly reduced the contact number per unit time. The dynamics of the total population *N* are governed by the following logistic equation:

$$\frac{dN(t)}{dt} = \left(b - \mu \frac{rN(t)}{K}\right)N(t) - \left(d + (1 - \mu)\frac{rN(t)}{K}\right)N(t).$$
(2)

The birth rate decreases and the death rate increases to its carrying capacity *K* for $0 < \mu < 1$. The birth and death rate are density independent for $\mu = 0$ and 1, respectively.

Now using the delayed *SIR* epidemic model (1), we will derive an optimal control model to fit our control strategy. The theoretical foundation of optimal control models with underlying dynamics given by ordinary differential equations was developed by Pontryagin and his co-worker in Moscow about 1950 (Kamien and Schwartz, 2000). So by Pontryagin's Maximum Principle, its extension and appropriate numerical methods, we will set an optimal control problem in the time delayed *SIR* epidemic model to control the spread of diseases. The main goal of this problem is to investigate an effective treatment strategy to control infection diseases, which means that we can make an *SIR* epidemic control model which satisfies that the maximum numbers of infected individuals are not larger than that of susceptible individuals and more individuals are recovered after infection.

In order to set an optimal control problem, first, we make the following notational conventions. Let Λ , T > 0 be given constant and define the control set:

$$U = \{u(t) \in L^{2}(0, T) : 0 \le u(t) \le \Lambda, 0 \le t \le T\},$$
(3)

where u(t) is Lebesgue measurable and called a control variable. In this problem, the biological meaning of the control variable is that low levels of the number of infected individuals build by no contact to the susceptible individuals. In case of high contact rate the number of infected individuals increases while the number of susceptible and recovered individuals decreases. Better treatment and low contact rate bring the number of infected individuals to a small level, susceptible individuals begin to build again and more individuals are recovered from infection. Therefore, the probability of infected individuals I(t)/N(t) that an infected individual spread is made by an infectious individual and this is controlled by an optimal control treatment u(t) so that a fraction u(t)I(t)/N(t) of infected individuals are moved from *I* class to *R* and *S* classes. From these facts, our optimal control problem is given by the following.

Find a control u(t) and a triple individual (S(t), I(t), R(t)) to minimize the objective functional

$$J_{\epsilon}(u) = \int_0^T \left[I(t) + \frac{\epsilon u^2(t)}{2} \right] dt$$
(4)

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