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Global dynamics of a network epidemic model for waterborne diseases spread

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ABSTRACT

A network epidemic model for waterborne diseases spread is formulated, which incorporates both indirect environment-to-human and direct human-to-human transmission routes. We consider direct human contacts as a heterogeneous network and assume homogeneous mixing between the environment and human population. The basic reproduction number R_0 is derived through the local stability of disease-free equilibrium and established as a sharp threshold that governs the disease dynamics. In particular, we have shown that the disease-free equilibrium is globally asymptotically stable if $R_0 < 1$; while if $R_0 > 1$, in case of permanent immunity the endemic equilibrium is globally asymptotically stable based on Kirchhoff's matrix tree theorem, and in case of temporal immunity sufficient conditions are given to guarantee global stability of the endemic equilibrium. Moreover, various immunization strategies are investigated and compared. The results obtained are informative for us to further understand the disease propagation in multiple transmission routes and devise some effective intervening measures to fight against the diseases spread.

1. Introduction

Since the spread of a disease is not amenable to direct experimental study on humans, mathematical modeling for the spread of infectious diseases is becoming increasingly an important tool to shed some light on the disease dynamical processes. Due to the seminal work of Ross [1], who first proposed a kinetic model to investigate the spread of malaria and employed a "threshold" concept, disease dynamical modeling has become a crucial approach in understanding the transmission and interventions of communicable disease qualitatively and quantitatively. The following influential work was that of Kermack and McKendrick's [2], who proposed a systematic 'threshold theory' that determines whether a disease will become prevalent or not.

In recent years, epidemiology models have been studied by a number of researchers. For example, Mollison detailed the structure of epidemic models and their relation to data [3]. De la Sen et al. discussed a generalized time-varying *SEIR* propagation disease model subject to delays which potentially involved mixed regular and impulsive vaccination rules [4]. Kuniya and Inaba formulated a normalized system for an infected population as an initial boundary value problem of a partial differential equation, and then established a threshold value for the existence and uniqueness of a nontrivial endemic periodic solution of an age-structured *SIS* epidemic model with periodic parameters [5]. Nistal et al. investigated the stability, periodic solutions and (regular or adaptive) impulsive vaccination design in a generalized *SVEIR* epidemic model

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[6]. Ackleh and Salceanu considered a nonautonomous version of the *SIR* epidemic model, and gave some sufficient conditions for the robust uniform persistence of the total population, as well as of the susceptible and infected subpopulations [7]. The aim of these models is to both understand observed epidemiological patterns and predict the consequences of the introduction of public health interventions to control the spread of diseases (see e.g., [8] for an overview).

Nowadays, waterborne diseases still remain a serious public health concern, rendering more than 3.5 million deaths per year according to WHO estimates [9]. For example, cholera is one of the serious waterborne diseases, which causes diarrhea and dehydration and could lead rapidly to death, especially in populations that are impoverished or have limited access to health care [10]. Historically, six out of the seven cholera pandemics have swept the globe since 1816. Most recently, the seventh pandemic started from Indonesia in 1961, spread into Europe, South Pacific and Japan in the late 1970s, reached South America in 1990s, and has continued (though much diminished) to occur, such as the Zimbabwe cholera epidemic from August 2008 to July 2009 which caused more than 98 thousand cases and 4,200 deaths [11]. Worldwide, about 3–5 million people have been infected with cholera and an estimated 100–130 thousand persons die in the year of 2010 alone [12]. Therefore, cholera poses a significant public health burden to developing countries and continue to draw worldwide attention.

There are many mathematical models being proposed in the literature to investigate the complex epidemic and endemic behavior of cholera. For example, Capasso and Paveri-Fontana suggested the earliest mathematical model to study the 1973 cholera epidemic in the Mediterranean region [13]. The model consisted of two components, the concentration of the pathogen in water, x_1, and the population of the infected people, x_2. In 2001, Codeco [14] extended the work of [13] to explicitly account for the role of the aquatic reservoir in cholera dynamics and incorporate the susceptible population into the model. Following the similar idea, Hartley et al. [15] in 2006 included a hyper-infective stage of *V*. cholerae (i.e., freshly shed vibrios) into their model, which tried to implicitly highlight the importance of direct human-to-human interaction in cholera epidemics. However, all of the previous models considered only one transmission route. Actually, many different specific transmission pathways are possible, and there has been evidence showing that the direct human-to-human interaction also plays a important role in cholera transmission [16,17]. To this end, Tien and Earn [18] proposed a waterborne pathogen model, allowing bilinear incidence rates for both the environment-to-human and human-to-human transmission routes. Mukandavire et al. [19] presented a model with saturation effect to estimate the reproduction number for the 2008–2009 cholera outbreak in Zimbabwe. In addition, there are many other factors being considered in attempting to understand waterborne disease dynamics, including water treatment efforts [20], climatological factors such as rainfall [21,22], or pathogen ecology outside of human hosts [23,24]. Unraveling how these factors interact to affect disease dynamics is remaining a challenging issue.

So far, most of the above models often assume that the population is mixed homogeneously; in other words, each individual in the population has the equal probability of contacting any other individual. This hypothesis is obviously not realistic. In fact, the connections among individuals allowing for the spread of infectious diseases naturally shape a network, while the generated network provides us insights into the epidemic dynamics. Under such framework, each individual corresponds to a node of the network, and potential interactions among individuals correspond to the links; two individuals connected by a link are neighbors of each other. For instance, the spreading of diseases can be considered as occurring over a network of human contacts [25] and the spreading of computer viruses as occurring over the internet [26,27]. The intimate relationship between epidemiology and network theory dates back to the mid-1980s [28,29]. Hence, one may argue that epidemiology has embraced the potential of network theory more than any other discipline.

In 2001, Pastor-Satorras and Vespignani [30,31] pioneered this field to model disease spread on a contact network with the following *SIS* model (i.e., infected nodes go back to the susceptible state once recovered, without any immunity) based on the mean-field theory,

$$\begin{cases} \frac{dS_k(t)}{dt} = \gamma I_k(t) - \beta k S_k(t)\Theta(I_k(t)), \\ \frac{dI_k(t)}{dt} = \beta k S_k(t)\Theta(I_k(t)) - \gamma I_k(t), \end{cases} \tag{1}$$

where γ is the cure rate of infected nodes, β is the infected rate of each susceptible (healthy) node if it is connected to at least one infected nodes; N_k is the total number of nodes with degree k, i.e., $N_k = S_k + I_k$, S_k and I_k are respectively the number of susceptible and infectious nodes with degree k, and n is the maximum degree of all nodes. At each time step, the first term in first equation of (1) represents the recovery of infected nodes in degree k, while the second term describes the probability that a node with k links is healthy and then acquires the infection via a connected infectious node. In uncorrelated networks the quantity $\Theta(I_k(t))$ [31] is defined as

$$\Theta(I_k(t)) = \frac{\sum_{k=1}^{n} kI_k}{\sum_{k=1}^{n} kN_k},$$

which represents the probability that a randomly chosen edge emanating from a node of degree k points to an infected node. Pastor-Satorras and Vespignani [32] demonstrated that the infection can invade the whole population if the following condition is satisfied,

$$R_0 = \frac{\beta}{\gamma}\frac{\langle k^2\rangle}{\langle k\rangle} = \frac{\beta}{\gamma}\left(\langle k\rangle + \frac{Var[k]}{\langle k\rangle}\right) > 1, \tag{2}$$