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Discussion Dynamics of an epidemic model with quarantine on scale-free networks

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A R T I C L E I N F O

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ABSTRACT

Quarantine strategies are frequently used to control or reduce the transmission risks of epidemic diseases such as SARS, tuberculosis and cholera. In this paper, we formulate a susceptible-exposed-infectedquarantined-recovered model on a scale-free network incorporating the births and deaths of individuals. Considering that the infectivity is related to the degrees of infectious nodes, we introduce quarantined rate as a function of degree into the model, and quantify the basic reproduction number, which is shown to be dependent on some parameters, such as quarantined rate, infectivity and network structures. A theoretical result further indicates the heterogeneity of networks and higher infectivity will raise the disease transmission risk while quarantine measure will contribute to the prevention of epidemic spreading. Meanwhile, the contact assumption between susceptibles and infectives may impact the disease transmission. Furthermore, we prove that the basic reproduction number serves as a threshold value for the global stability of the disease-free and endemic equilibria and the uniform persistence of the disease on the network by constructing appropriate Lyapunov functions. Finally, some numerical simulations are illustrated to perform and complement our analytical results.

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1. Introduction

Mathematical models have shown to be an important tool to investigate dynamical behavior of epidemic diseases. In the early studies, these models were presented on homogeneous networks by assuming that transmission from an infectious individual to any susceptible individual is equally likely. Obviously, this assumption is unrealistic in some sense because physical contacts between individuals vary with each individual.

Since a new modeling framework on scale-free networks was introduced by Barabási and Albert in 1999 [1], many real complex networks, such as contact networks, have been shown to exhibit scale-free property. In a scale-free network, the probability P(k) that a node is connected to k other nodes follows a power-law, that is $P(k) \sim k^{-r}$ [1], where r is a characteristic exponent with value in the range $2 < r \le 3$. Consequently, the study of epidemic models based on scale-free networks has gradually attracted the attention of the researchers in recent decades, and many constructive results have been obtained. Pastor-Satorras and Vespignani first pointed out that the epidemic threshold is absent or

https://doi.org/10.1016/j.physleta.2017.09.040 0375-9601/© 2017 Elsevier B.V. All rights reserved. infinitesimal with a large number of links and nodes by investigating a susceptible-infected-susceptible (SIS) epidemiological model on scale-free networks [2]. Since then, a large body of researches observed similar conclusions in various scenarios [3-5]. Many researchers investigated the effect of scale-free network structures on epidemic spreading from different aspects including quenched networks [6], inclusion of households (clusters) [7], adaptive and weighted contact networks [8], and networks with rewiring edges [9,10]. In terms of theoretical analysis, the authors of Ref. [11] established mathematical proof about global stability of the well known SIS model built by Pastor-Satorras and Vespignani. Besides that, Olinky and Stone showed that connectivity-dependent infection schemes have the same impact on epidemic propagation as network structures [12]. The authors of Ref. [13] further revealed that effectively heterogeneous contacts play a significant role in epidemic dynamics by introducing an effective contact function. Meanwhile, the authors of Ref. [14] investigated the combinational effects of the connectivity structure and functional factors such as transmissions of edges and infectivities of nodes. Apart from network structure and infection schemes, epidemic transmission with delay [15-17] and the effects of various immunization schemes including ring and targeted vaccinations [18,19] were studied.







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Quarantine, as an effective measure, has been frequently used to control the spreading of a large number of human and animal infectious diseases, such as SARS in 2003 [20-22] and the 2009 swine influenza pandemic [23]. Li et al. proposed an SIORS epidemic model on the scale-free networks [24] and discussed the effects of guarantine on the spreading of infectious diseases and local asymptotic stability of the disease-free equilibrium. Huang et al, also presented an SIORS epidemic model with demographics and vaccination on complex heterogeneous networks and studied the global asymptotic stability of disease-free equilibrium and global attractivity of endemic equilibrium [25]. But the guarantined rate in [24] and [25] are both constants. In this paper, we shall investigate the influence of quarantine depending on degree of nodes on disease transmission on scale-free networks through a new deterministic model. Deterministic models have been presented to describe epidemic spread based on two basic compartmental frameworks. One is the susceptible-infected-susceptible (SIS) framework. In this model, there are only two states, susceptible or infected. The susceptible individuals may get infected by contacting with an infected individual, and the infected individuals will be cured by treatment and become susceptible again. This kind of models are appropriate for diseases conferring no immunity [26], such as salmonella and influenza. The other one is the susceptibleinfected-recovered (SIR) framework, in which the infected individuals may become recovered and remain immune to further infection, such as measles [27] and varicella [28]. Alternatively, SIR model is called the susceptible-infected-removed model because the recovered individuals can be removed from the population. The purpose of the current paper is to investigate the dynamics of diseases conferring temporary or waning immunity [26] with quarantine on scale-free networks. Moreover, many diseases in real world have a latent period during which the infected individuals can not transmit the disease to susceptible individuals. Therefore, it is more realistic to introduce an exposed compartment in the SIR model, which leads to an susceptible-exposed-infected-recovered (SEIR) model. As a matter of fact, plenty of SEIR models have been formulated and applied to different infectious diseases, such as tuberculosis [29] and dengue fever [30], and edge-based SEIR dynamics [31] are also investigated. In addition, we introduced an quarantined compartment in order to investigate the effect of quarantine strategy on scale-free networks. It is widely assumed that the larger the infectious nodes degree, the more the infected individuals by these nodes. Thus, we view the quarantined rate as a function of degree to rapidly and effectively control the epidemic spreading. The quarantined rate in our model is more appropriate than constant one in the traditional epidemiology and in [24] and [25]. The resulting model is an susceptible-exposed-infectedquarantined-recovered (SEIQR) model. Besides that, the birth and death of individuals are taken into account to make the models more reasonable.

The rest of the paper is organized as follows. Section 2 presents an SEIQR model on scale-free networks. Section 3 calculates the basic reproduction number and investigates theoretically the stability of disease-free and endemic equilibria using characteristic equations and Lyapunov functions. Section 4 presents some simulations and a brief discussion. Finally, Section 5 concludes the paper with a summary of main results.

2. The model

We assume that there are five states of the nodes on a scalefree network, and let $S_k(t)$, $E_k(t)$, $I_k(t)$, $Q_k(t)$ and $R_k(t)$ denote the densities of the susceptible, exposed, infectious, quarantined and recovered individuals with degree k at time t, respectively. Then, $S_k(t) + E_k(t) + I_k(t) + Q_k(t) + R_k(t) = 1$. Based on the meanfield theory, we formulate the following model on a scale-free network:

$$\begin{cases} \frac{dS_k(t)}{dt} = d - \lambda k S_k(t) \Theta(t) - dS_k(t), \\ \frac{dE_k(t)}{dt} = \lambda k S_k(t) \Theta(t) - (\mu + d) E_k(t), \\ \frac{dI_k(t)}{dt} = \mu E_k(t) - (\delta(k) + \gamma + d) I_k(t), \\ \frac{dQ_k(t)}{dt} = \delta(k) I_k(t) - (\sigma + d) Q_k(t), \\ \frac{dR_k(t)}{dt} = \gamma I_k(t) + \sigma Q_k(t) - dR_k(t), \end{cases}$$
(2.1)

where k = 1, 2, ..., n, with *n* being the maximum degree number in this network. The parameter λ represents the infection rate, μ is the rate at which the exposed individuals become infectious, $\delta(k)$ describes the rate at which the infectious individuals become quarantined, which is related to degree k, γ (σ) denotes the recovery rate at which nodes recover from infected (quarantined resp.) compartment. Suppose that the newborns are all susceptible and deaths (without taking disease-related death into account) are balanced by birth, then, the birth rate is assumed to be equal to the per capita natural death rate denoted by d. Based on the epidemiological meaning, the parameters μ , $\delta(k)$, γ and σ are all nonnegative while *d* and λ are positive. $\Theta(t)$ is the probability that a randomly chosen neighbor is infective at time *t*, and on the uncorrelated network, the probability that a link points to a node of connectivity i is independent of the connectivity k of the node from which the link is emanating. Throughout this paper, we assume that complex networks are uncorrelated. Therefore, $\Theta(t)$ can be written as

$$\Theta(t) = \frac{1}{\langle k \rangle} \sum_{i=1}^{n} \varphi(i) P(i) I_i(t), \qquad (2.2)$$

where $\langle k \rangle$ is the average degree of the network, i.e., $\langle k \rangle = \sum_{i=1}^{n} iP(i)$, P(k) is the probability that a node is connected to k other nodes and follows a power-law, and $\varphi(k)$ is the infectivity of nodes with degree k, i.e., $\varphi(k)$ denotes the average number of edges from which a node with degree k can transmit the disease [12]. For notational convenience, we define $\langle m(k) \rangle = \sum_{i=1}^{n} m(i)P(i)$ in which m(k) is a function of the variable k.

3. Threshold and stability analysis

It is noted that

$$\Gamma = \left\{ (S_k, E_k, I_k, Q_k, R_k) \in R_{5n}^+ : S_k, E_k, I_k, Q_k, R_k \ge 0, \\ S_k + E_k + I_k + Q_k + R_k \le 1, k = 1, 2, \cdots, n \right\}$$

is a positively invariant set for system (2.1) based on Proposition 2.2 in [32]. The interior and boundary of Γ are denoted by $\stackrel{o}{\Gamma}$ and $\partial\Gamma$ respectively. The system (2.1) always admits a diseasefree equilibrium $P_0 = \{1, 0, 0, 0, 0, \dots, 1, 0, 0, 0, 0\} \in \partial\Gamma$, namely, $S_k = 1$, $E_k = I_k = Q_k = R_k = 0$, $k = 1, \dots, n$. Next, we will derive an epidemic threshold, which is an important epidemiological index in the study of epidemic transmission.

According to [33,34], only the compartments E_k and I_k , $1 \le k \le n$, are involved in the calculation of epidemic threshold. Around the disease-free equilibrium P_0 , the rate matrix F of appearance of new infections and the transfer rate matrix V of individuals between any two compartments are denoted respectively by

$$F = \left(\begin{array}{cc} 0 & A \\ 0 & 0 \end{array}\right),$$

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