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An epidemic spreading model on adaptive scale-free networks with feedback mechanism

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HIGHLIGHTS

- Present a new SIRS epidemic model with feedback mechanism on scale-free networks.
- Obtain and analyze the basic reproductive number and the epidemic threshold.
- Study the stability of disease-free equilibrium and the permanence of the disease.
- The feedback parameters can affect the epidemic spreading and the endemic level.

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ABSTRACT

A *SIRS* epidemic model with feedback mechanism on adaptive scale-free networks is presented. Using the mean field theory the spreading dynamics of the epidemic is studied in detail. The basic reproductive number and equilibriums are derived. Theoretical results indicate that the basic reproductive number is significantly dependent on the topology of the underlying networks. The existence of equilibriums is determined by the basic reproductive number. The global stability of disease-free equilibrium and the epidemic permanence are proved in detail. The feedback mechanism cannot change the basic reproductive number, but it can reduce the endemic level and weaken the epidemic spreading. Numerical simulations confirmed the analytical results.

1. Introduction

Public security and individual health are still at the risk of the outbreak of epidemic diseases even after the development of modern medicine in recent years [1–4]. What's worse, owing to the development of global transportation network, more and more frequent contacts among people make the new emerging outbreak rapidly spread worldwide before adequate supplies of vaccine could be made and distributed [5]. Therefore, the study of epidemic models is of dramatic significance, which plays a significant role in predicting and controlling the spread of disease [6,7]. According to individuals' disease status, the majority of traditional epidemic models are based on a compartmentalization of individuals, which generates two representative models: the *SIS* (susceptible–infected–susceptible) model and the *SIR* (susceptible–infected–recovered) model. In the *SIS* model the infected nodes will not obtain immunity and can return to the susceptible state again after

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recovering, such as the gonorrhea and influenza, etc. In the *SIR* model the recovered nodes have permanent immunity and people are never infected by that epidemic disease again, such as the measles, parotitis and SARS, etc. Early classical representations of epidemic disease dynamics assumed that all individuals were large and mixed homogeneously such that deterministic equations with simple frequency-dependent transmission were appropriate. Obviously these simple models cannot completely reflect the realistic feature of the spread of diseases, which are subsequently extended in ways towards making them more realistic in recent years. Some such extensions are combining with spatial structure that the geographical embedding could affect the topology of disease transmission network [8,9].

Another generalization of the initial simple deterministic epidemic model is to focus on the complex topology of social interactions. As shown in the field of complex networks, the scale-free property is a fundamental discovery in social networks [10,11]. For further understanding of the epidemic spreading dynamics in real world, the scale-free property of social networks has been taken into account by many epidemic models. In networks, nodes represent individual people and edges represent the relationship of people. As for epidemic spreading dynamics, that relationship is contact to transmit the disease. Recently, starting with the works by Pastor-Satorras and Vespignani [12,13], there has been a burst of activity on researching the impacts of the network topology on epidemic spreading [14–26]. Refs. [14,15] studied the *SIS* epidemic dynamics on scale-free networks with degree correlations. Barthélemy et al. observed that epidemic spreading follows a precise hierarchical dynamics on scale-free networks [23]. Loecher and Kadtke studied the details of the hierarchical propagation and achieved an unusual enhanced predictability for the order of infected nodes [24].

However, in most of the research work mentioned above, the initiative response of individual is not considered when epidemic diseases prevail. In fact, as soon as an epidemic outbreaks, people will be more cautious and will reduce contacts with other people consciously. Obviously, the feedback mechanism can change the contacts among people, i.e. network topology structure. Although Refs. [25,26] proposed a *SIS* model with the feedback mechanism, the recovered nodes have not been considered and the strict proof of the endemic equilibrium permanence under the feedback mechanism has not been given. In this paper, considering the recovered nodes we focus on a new *SIRS* epidemic model with feedback mechanism on heterogeneous networks and comprehensively proved the permanence of the disease in detail.

The rest of this paper is organized as follows. In Section 2, we present a new *SIRS* epidemic model with feedback mechanism on adaptive scale-free networks. In Section 3, two equilibriums are obtained at first. Then we analyze the globally asymptotic stability of disease-free equilibrium and the permanence of the disease in detail. In Section 4, some numerical simulations of the proposed model are shown. Finally, we conclude the paper in Section 5.

2. Model formulation

One of the most effective interventions to contain the spread of epidemic diseases is the feedback mechanism as discussed above. In order to investigate the efficiency of feedback mechanism policy, we consider the new *SIRS* model with feedback mechanism on scale-free networks. On the scale-free networks, each individual is represented by a node of the network and the edges are the connections between individuals along which the infection may spread. Taking into account the heterogeneity induced by the presence of nodes with different connectivities, let $S_k(t)$, $I_k(t)$ and $R_k(t)$ be the relative densities of susceptible, infected and recovered nodes of degree k at time t respectively. In the course of disease transmission, a susceptible individual is infected with probability α if it connects to an infected one. The rate constant of recovery for infected individuals is denoted by β. The rate constant of losing immunity for recovered individuals is γ. Here, we assume that the birth rate equals the death rate, and the rate constant is l. Thus, the dynamic mean-field reaction rate equations can be written as

$$\begin{cases} \dfrac{dS_k(t)}{dt} = l - lS_k(t) - \alpha k(1 - \mu\theta(t))\theta(t)S_k(t) + \gamma R_k(t) \\ \dfrac{dI_k(t)}{dt} = \alpha k(1 - \mu\theta(t))\theta(t)S_k(t) - \beta I_k(t) - lI_k(t) \\ \dfrac{dR_k(t)}{dt} = \beta I_k(t) - \gamma R_k(t) - lR_k(t) \end{cases} \tag{1}$$

where the probability $\theta(t)$ describes a link pointing to an infected individual, which satisfies

$$\theta(t) = \frac{\sum_k kP(k)I_k(t)}{\sum_s sP(s)} = \frac{1}{\langle k\rangle}\sum_k kP(k)I_k(t), \tag{2}$$

where $\langle k\rangle$ is the average degree within the network, $P(k)$ is the degree distribution. $I(t) = \sum_k P(k)I_k(t)$ is the total density of infected individuals in the network. Clearly, these variables obey the normalization condition:

$$S_k(t) + I_k(t) + R_k(t) = 1.$$

The initial conditions for system (1) can be given as follows

$$S_k(0) = 1 - I_k(0) - R_k(0) \geq 0, \quad I_k(0) \geq 0,\ R_k(0) \geq 0.$$