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Spreading dynamics and synchronization behavior of periodic diseases on complex networks

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HIGHLIGHTS

- A new discrete-susceptible–infected–recovered–susceptible (DSIRS) model is introduced.
- The disease spreading dynamics on complex networks is analyzed.
- Synchronization behavior of epidemic spreading is investigated.
- The cycle duration and the topological network structure for the immune.
- Immune strategies are compared and analyzed for the new model to prevent the disease propagating.

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ABSTRACT

A new discrete-susceptible–infected–recovered–susceptible (DSIRS) model is introduced in this paper to investigate the disease spreading dynamics and synchronization behavior on complex networks. In the model, every node is considered independently rather than as a part of one group that has a common node state in complex networks. The synchronization phenomenon of epidemic spreading based on the model in random networks and scale-free networks is analyzed. Synchronization is affected by the infection duration, the complete cycle duration and the topological network structure, which affects the immune strategy. Accordingly, immune strategies including the maximum degree immune strategy and the nearest immune strategy are proposed to prevent disease propagating.

1. Introduction

The outbreak of infectious disease (i.e., The Black Death in the 14th century, SARS in 2003, avian influenza in 2004, etc.) brings great loss and damage to human society. Therefore, research on the propagation behavior of epidemics is enormously important to prevent and control its spread. The main way infectious diseases spread in humans is by contact, and the contacting relationship among the people who spread infectious diseases naturally forms a complex social relation network [1]. In recent years, the development of the complex network dynamics theory provides a new way of thinking about the study of disease outbreaks [2,3]. Complex networks theory is an important tool when researching natural social problems with a large number of nodes, and the theory plays a vital role in various scientific research fields (i.e., transmission dynamics [4], network cascading failures [5], etc.).

In order to better understand the process of infectious diseases spreading, it is very important to study the dynamics of infectious diseases combined with the social relation network. In fact, the transmission of infectious diseases in society networks can be summarized as follows: the individuals who are infected with the virus in-network will affect the

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susceptible individuals contacted within the same network and cause healthy individuals, with a certain probability, to be infectious. With the continuous infection of the individuals, the infectious disease would become an outbreak [6]. The spreading mechanism can be described well with a complex network model [7,8]. A node can denote the individual, and the relationship between the individual can be denoted by an edge. An infectious disease transmission dynamics model has been widely examined recently, and a number of different network transmission models have been proposed. However, the most representative model is the mathematical model based on differential equations [9–13]. Zhu et al. [14] provided a typical mean-field modeling framework to describe the time-evolution dynamics and offer some mathematical skills to study the spreading threshold and the global stability of the model. In Ref. [15], a general susceptible–infected–susceptible (SIS) model with infective vectors on complex networks is studied, and a new technique based on the basic reproduction matrix is introduced. Wang et al. [16] reviewed two node-based SIR models incorporating degree correlations and an edge-based SIR model without considering the degree correlation to predict the disease evolution on correlated networks. The predictions were then compared to these models with stochastic SIR simulations.

However, the mathematical models based on differential equation typically did not consider the differences between the individual and the impact of the network topology structure, which is closely related to the discrete model [17,18] for the spread of infectious diseases. Most of these models macroscopically describe the transmission dynamic behaviors on the complex network [9–16] and are rarely discussed in the microscopic view. In Ref. [19], Liu et al. proposed a novel and successful targeted immunization strategy based on percolation transition. Ababou studied the epidemic spreading in a finite-precision BA model in Ref. [20] and numerically investigated the SIRS epidemic model on an exponential network generated by a preferential attachment procedure [21], in which the synchronization behavior was discussed.

Synchronicity is a striking feature in the process of disease spreading and one of the central phenomena representing the emergence of collective behavior in natural and complex systems. In Ref. [22], the issue of exponential synchronization for a class of stochastic coupled networks with Markovian switching is investigated. S.P. Ansari et al. [23] presented the synchronization between a pair of identical SIR epidemic chaotic systems and fractional-order time derivatives using an active control method. A framework is proposed to infer unobserved epidemic subpopulations by exploiting the synchronization properties of multi-strain epidemic models in Ref. [24]. The synchronization process is ubiquitous in nature and plays a very important role in many different contexts, such as biology, ecology, climatology, sociology, technology, or even in the arts [25,26].

Previous studies have shown that immunization is an effective way to inhibit traditional epidemic spreading in which infections are transmitted as a reaction process from nodes to all neighbors [27–31]. There are some main immune strategies for the protection of complex networks including random immunization [32], targeted immunization [27], local immunization [33,34], etc. Liu et al. [35] analyzed the successful and unsuccessful probabilities of the acquaintance immunization strategy by utilizing a simple example and noted the possible invalidation reasons of this method. Samanta [36] considered a dynamic model of hand–foot–mouth disease with a varying total population size, saturation incidence rate and discrete time delay to become infectious. Yang et al. [37] studied the control of traffic-driven epidemic spreading by immunization strategy and considered the random, degree-based and betweenness-based immunization strategies.

The contributions of this paper can be summarized as follows:

(1) A discrete SIRS model is proposed to study epidemic spreading dynamics from the micro-cosmic point of view. The main point of this model compared with the classical differential equation model is that every node is considered independently rather than as part of one group that has a common node state in complex networks.
(2) The synchronization of an epidemic based on the model discussed above in random and scale-free networks is analyzed. For the two networks, a positive correlation relationship is presented between the synchronization parameters and the infection time for the same infection cycle.
(3) The proposed immune strategies effectively prevent and control the spread of an epidemic. Some new immune strategies (such as the maximum degree immune strategy and the nearest immune strategy) are introduced to control disease propagation. The immune effect is remarkable compared with a random immune strategy in random and scale-free networks.

The remainder of this paper is organized as follows. Section 2 introduces the DSIRS model, which is used to simulate the disease propagation mechanism in a complex network. Section 3 presents the numerical results for the epidemic dynamics on random and scale-free networks for different values of infection parameters and investigates the synchronization phenomena. Conclusions are drawn in Section 4.

2. The DSIRS model

In social networks, random networks and scale-free networks are two types of common networks. Therefore, this paper studies a discrete SIRS model based on the above two types of networks. Node states in a network follow the progression of susceptible (S), infectious (I) and then recovered (R). A return to the initial state was due to the loss of immunity ability. Each node i is described at time t by an integer variable $\tau_i(t)$, which runs from 0 to $\tau_e(\tau_e = \tau_I + \tau_R)$, being the natural period of the disease cycle. This period is the combination of the infection time τ_I and the refractory time τ_R. The range of $\tau_i(t)$ is $[0, \tau_e]$.

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