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Epidemic spreading on weighted networks with adaptive topology based on infective information☆

Yun Feng [a], Li Ding [a,b,*], Yun-Han Huang [a], Li Zhang [a]

[a] Department of Automation, School of Power and Mechanical Engineering, Wuhan University, Wuhan, 430072, PR China
[b] Department of Electrical and Computer Engineering, College of Engineering, University of Iowa, Iowa City, IA, 52242, USA

HIGHLIGHTS

- A new epidemic spreading model considering the interaction between network topology and epidemic spreading is established.
- The network topology varies according to the global and local infective information of individuals.
- The influences of the interaction on epidemic spreading and network topology are analyzed by discrete-time Monte-Carlo simulation.

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ABSTRACT

In this paper, we consider epidemic spreading on a weighted adaptive network in which the network topology varies according to the global and local infective information of individuals. We focus on the relationship between network topology and epidemic spreading. Interacting strength is defined to evaluate the level of how individuals' infective information taking effects on their connections. The model is analyzed by discrete-time Monte-Carlo simulations with an initial BA scale-free network. It is found that greater interacting strength leads to higher epidemic threshold, lower average disease density of steady-state and shorter epidemic prevalent decay time. Besides, the interaction tends to change the initial BA scale-free network to a fat-tail network while the scaling exponent almost keeps unchanged. In addition, individual who reacts with local infective information will significantly restrain the outbreak of disease comparing to the one with global infective information, and this phenomenon becomes more notable if the interacting strength becomes greater.

1. Introduction

In recent years, an increasing amount of attention has been focused on epidemic spreading over complex network. One of the well-studied problems is to investigate the transmission behavior of the disease over networks. In most literatures referring to the studying of epidemic spreading behavior, the topology structure of the underlying network is assumed to be static [1–8].

However, in real social networks, the relations between individuals are unlikely to keep unchanged all the time. Evolving network and adaptive network are networks whose topology structures are time-varying. Generally speaking, a network

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* Corresponding author at: Department of Automation, School of Power and Mechanical Engineering, Wuhan University, Wuhan, 430072, PR China.
E-mail address: liding@whu.edu.cn (L. Ding).

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whose topology structure varies independently from epidemic spreading is called an *evolving network* while an *adaptive network* refers to network whose topology varies according to disease spreading. A susceptible individual tends to avoid contacts with its infected neighbors on adaptive networks. Epidemic spreading on evolving networks [9–12] and adaptive networks [13–22] has been well studied and some remarkable results have been proposed. For example, a novel continuous-time adaptive susceptible–infectious–susceptible(ASIS) model is proposed to investigate the interaction between epidemic spreading and topology adapting in Ref. [23]. The epidemic dynamics with susceptible–infected–susceptible(SIS) model on a weighted adaptive network to emphasize that the contact strengths among people are diverse both in the duration time and the distance are studied in Ref. [24].

In these works, there is a basic assumption that the network topology varying probabilities keep unchanged during the epidemic spreading process. However, in reality, it is more natural that a susceptible individual tends to reduce contacts with its infected neighbors, while these contacts will be restored once the infected neighbors are cured, both of reduction and restoration may be determined by some probabilities related to current disease density. Motivated by the above considerations, we consider the problem that the network topology is time-varying with probability according to the current disease density. We define that individuals change weights between their neighbors according to disease density in the whole network as adopting global infective information while the ones according to neighborhood disease density as adopting local infective information. Based on these two topology adapting mechanisms, we construct a novel model considering interactions between epidemic spreading and topology adapting on a weighted network. The model is mainly analyzed by discrete-time Monte-Carlo simulation with an initial BA scale-free network.

The main contributions of this paper are given as follows. Firstly, it is found that greater interacting strength leads to higher epidemic threshold, lower average disease density of steady-state and shorter epidemic prevalent decay time. Secondly, the interaction tends to change the initial BA scale-free network to a fat-tail network while the scaling exponent almost keeps unchanged. Lastly, individuals who react with local infective information will significantly restrain the outbreak of disease comparing to the ones with global infective information, and this phenomenon becomes more notable if the interacting strength becomes greater.

The paper is organized as follows. In Section 2, a novel model considering the interaction is established. In Section 3, discrete-time Monte-Carlo simulation results of the proposed model are presented. Finally, we conclude this paper in Section 4.

2. Model description and formulation

2.1. Model description

We consider that epidemic spreads on an adaptive network whose topology structure varies adaptively according to the current disease density. The graph of the network is denoted by $G(N, L)$, which consists of N individuals and L pairs of links. It is assumed to be symmetric and undirected. Our model is based on the standard susceptible–infected–susceptible(SIS) model. Therefore, the whole population is divided into susceptible and infected individuals according to their current physical states. Susceptible individuals have the probability β to be infected by infected individuals through the links between them in a step. Meanwhile, infected individuals can be cured with the probability γ.

The weight between individuals i and j at step t is measured by $w_{ij}(t)$ and $w_{ji}(t)$. Due to the symmetry of the graph, $w_{ij}(t) \equiv w_{ji}(t)$. For convenience, it is assumed that $w_{ij}(t) \in [0, 1]$, $\forall i, j \in \{1, 2, 3, \ldots, N\}$. When $w_{ij}(t) = 0$, it means that the edge between them is broken. The initial weights in the whole network are assumed to be either 0 or 1. We define that a susceptible individual i has the probability $p_{ij}^d(t)$ to decrease the weight between an infected individual j from $w_{ij}(t)$ to $\alpha w_{ij}(t)$ in step t. Conversely, once individual j is being cured and individual i stays healthy, the weight that has been decreased will restore from $\alpha w_{ij}(t)$ to $w_{ij}(t)$ with probability $p_{ij}^r(t)$. We define weight restoring only happens to the weights that have been decreased. Here $\alpha \in [0, 1]$ is defined as the weight adapting parameter. $\alpha = 0$ indicates that the edge will be broken completely with probability $p_{ij}^d(t)$, which is similar to the model in Ref. [23]. Meanwhile, $\alpha = 1$ demonstrates that the weight keeps unchanged, in accordance with the situation of standard SIS model. α measures the interacting strength of epidemic spreading and weight adapting. Smaller α indicates greater interacting strength. An illustrative example of topology adapting is presented in Fig. 1. Blue circles represent healthy individuals while the red ones represent infected individuals. The width of lines between individuals represent the weight between them. Broader lines denote stronger links. Both weight decreasing process (green and purple edges) and weight restoring process (green edge) are illustrated in this figure.

2.2. Model formulation

In this part, the discrete-time mathematical model based on the microscopic Markov chain approach(MMCA) is proposed. In standard SIS model, the basic assumption is that each individual contacts with all its neighbors once in each step. The MMCA equations of proposed model are presented in Eq. (2.1).

$$\begin{cases} p_i^S(t+1) = q_i(t)p_i^S(t) + \gamma p_i^I(t), \\ p_i^I(t+1) = (1 - q_i(t))p_i^S(t) + (1-\gamma)p_i^I(t). \end{cases} \tag{2.1}$$