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Controlling disease spread on networks with feedback mechanism*

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Many real-world networks have the ability to adapt themselves in response to the state of their nodes. This paper studies controlling disease spread on network with feedback mechanism, where the susceptible nodes are able to avoid contact with the infected ones by cutting their connections with probability when the density of infected nodes reaches a certain value in the network. Such feedback mechanism considers the networks' own adaptivity and the cost of immunization. The dynamical equations about immunization with feedback mechanism are solved and theoretical predictions are in agreement with the results of large scale simulations. It shows that when the lethality α increases, the prevalence decreases more greatly with the same immunization g. That is, with the same cost, a better controlling result can be obtained. This approach offers an effective and practical policy to control disease spread, and also may be relevant to other similar networks.

Keywords: complex network systems, disease spread, feedback mechanism
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1. Introduction

Complex networks, which have a more complicated topology than periodic lattices, have been observed in a large class of systems in different fields of science, technics, transport, social phenomena and so on.[1−3] In networks, nodes represent individuals or organizations, and links mimic the interactions among them. In view of wide occurrence of complex networks in nature, it becomes a very interesting issue to inspect the effect of their complex features on dynamic of epidemic and disease spreading,[4] and more in general on the nonequilibrium phase transitions that usually characterize these types of phenomena.[5] The characterization and understanding of epidemic dynamics on these networks can find immediate applications to a large number of problems, ranging from computer virus infections,[6] epidemiology[7] and the spreading of polluting agents.[8]

Most of these investigations demonstrate clear infection threshold effects on random and small-world networks[1] and the absence of infection threshold on scale-free network. So most methods have been designed to eradicate viruses and protect networks. Uniform immunization is effective for the homogeneous network; on the other hand, target immunization of the most highly connected individuals on scale-free network, while effective, requires global information about the network, rendering it impractical in many cases.[9]

However, the control of large outbreaks also raises the question of economic aspects of disease treatment.[10] Traditionally, successful control was understood in terms of reducing the number of individuals affected by the disease, regardless of cost. This might be justified when the treatment is cheap, but often the epidemic must be stopped at a manageable cost and with limited resources.[11] Furthermore, when controlling disease spread, the characteristic feature of many real-world networks is not taken into account, namely, the ability to adapt the networks in response to the dynamics in the network. Consider, for example, the spread of an infectious disease on a social network, people tend to respond to the emergence of an epidemic by avoiding contacts with infected individuals.

In view of this weakness, it becomes a task to find optimal immunization strategies oriented to minimize the risk of epidemic outbreaks on networks, a task with immediate practical and economical implications. In this paper, we study the susceptible–

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infected–susceptible (SIS) model[12] on the small-world network with feedback mechanism. We consider the network's adaptive ability and the cost of immunization in network. It is demonstrated that a simple feedback mechanism for the network has a profound impact on the immunization of the network. When the same number of nodes is immunized, the density of the infected nodes is decreased a lot with larger lethality α. In doing so, with the same limited cost, we can observe different epidemic results in network.

2. Model and analytical solutions

By investigating the SIS model, we estimate the effect of an increasing density of immunization individuals with feedback mechanism in complex networks. Each node of the network represents an individual and each link is a connection along that the infection can spread to other individuals. Within this description, individuals can only exist in two discrete states, susceptible, or 'healthy', and infected. Each susceptible node is infected with probability ν if it is connected to one or more infected nodes. At the same time, infected nodes are cured and become again susceptible with probability δ, defining an effective spreading rate $\lambda = \nu/\delta$. (Without lack of generality, we set $\delta = 1$.) Individuals run through the cycle susceptible $\rightarrow$ infected $\rightarrow$ susceptible. The updating can be performed with both parallel or sequential dynamics.[13] In an homogeneous network, the most significant result is the nonzero epidemic threshold λ_c. If the value of λ is above the threshold $\lambda \geq \lambda_c$ the infection spreads and becomes persistent in time. Below it, $\lambda \leq \lambda_c$, the infection dies out exponentially fast. An important observable is the prevalence ρ. It is the time average of the fraction of infected individuals reached after a transient from the initial condition.

The feedback mechanism can be introduced in the following.[14] Here we use $\rho(t)$ to describe the density of infected individuals at time step t. We suppose that all individuals have perceived the density of the infected individuals $\rho(t-1)$ at time step t. Each link of the whole network will disappear with probability $\alpha\rho(t-1)$, and preserve with probability $[1-\alpha\rho(t-1)]$. Here α is a parameter and ranges from 0 to 1 according to lethality of disease. Thus we can regard α as a constant of lethality. Obviously we can obtain that the larger the density of infected individuals $\rho(t)$ and the larger the constant of lethality α, the greater the connections will be cut off.

To illustrate the effect of feedback mechanism on disease spread of networks, we choose small-world networks of Watts–Strogatz (WS) model to study. The WS network is constructed as follows.[1] The starting point is a ring with N nodes, in which each node is symmetrically connected with its 2000 nearest neighbours. Then, for every node each link connected to a clockwise neighbour is rewired to a randomly chosen node with probability p, and preserved with probability $1-p$. This procedure generates a random graph with a connectivity distributed exponentially for large k, and an average connectivity $< k >= 2k$. The graph has small average shortest-path lengths and large clustering coefficients. The WS networks show the so-called small-world effect obviously for the rewiring probability $0 < p < 0.2$.[15,16]

For the class of exponentially bounded networks, one can generally consider that each node has roughly the same number of links, $k \approx < k >$, and, therefore, each node is homogeneous in its connectivity properties. We approach the analytical study of the behaviour of the SIS model with feedback mechanism in WS network. A single mean-field (MF) reaction equation for the density of infected nodes ρ(t) is considered,

$$\partial_t \rho(t) = -\rho(t) + \lambda < k > [1 - \alpha\rho(t-1)]\rho(t)[1-\rho(t)], \quad (1)$$

where $\rho(t-1)$ is the density of infected nodes at time $t-1$. In Eq.(1) we have neglected the density correlations among the different nodes, which are independent of their respective connectivities, and ignored all higher order correction in $\rho(t)$. The first term on the right-hand side in Eq.(1) considers infected nodes becoming healthy with unit rate. The second term represents the average density of newly infected nodes generated by each active node. This is proportional to the infection spreading rate λ, the number of links

from each node $k \simeq < k >$, the mean probability that given link points to a healthy node $[1-\rho(t)]$, and the probability of two nodes still connected with each other $[1-\alpha\rho(t-1)]$ at time step t. When $t \to 0$ the value of $\rho(t)$ equals a constant approximately, so we can achieve $\rho(t) \simeq \rho(t-1)$. After imposing the stationary condition $\partial_t \rho(t) = 0$, we obtain an epidemic threshold $\lambda_c = 1/ < k >$, such that

$$\rho = 0 \qquad \text{if} \qquad \lambda < \lambda_c, \tag{2}$$

$$\rho \sim (\lambda - \lambda_c)/(\alpha + 1) \qquad \text{if} \qquad \lambda \geq \lambda_c. \tag{3}$$

We also apply this feedback mechanism to immunization procedures. The simplest immunization procedure one can consider consists of random introduction of immune nodes in the network,[17] in order to get a uniform immunization density. Immune nodes cannot become infected and, thus, do not transmit the infection to their neighbours. In this case, for a fixed spreading rate λ, the relevant control parameter is the immunity g, defined as the fraction of immune nodes presented in the network. We also apply feedback mechanism to the network. At the MF level, the presence of uniform immunity will effectively reduce the spreading rate λ by a factor $(1-g)$; i.e. the probability of finding and infecting a susceptible and nonimmune node. By substituting $\lambda \to \lambda(1-g)$ in Eq.(1) we obtain

$$\partial_t \rho(t) = -\rho(t) + \lambda(1-g) < k > [1-\alpha\rho(t-1)]\rho(t)[1-\rho(t)]. \tag{4}$$

In the case of a constant λ, with the same method as in Eq.(1), the stationary prevalence obtained from Eq.(4) is given by

$$\rho = 0 \quad \text{if} \quad g > g_c, \tag{5}$$

$$\rho \sim (g_c - g)/(\alpha + 1) \quad \text{if} \quad g \leq g_c. \tag{6}$$

Here, g_c is the critical immunization value above which the density of infected nodes in the stationary state is null and dependent on λ as

$$g_c = \frac{\lambda - \lambda_c}{\lambda}. \tag{7}$$

Thus, the critical immunization that achieves eradication is related to the spreading rate and the epidemic threshold of the infection. Equation (7) is obviously valid only for $\lambda > \lambda_c$, and it implies that the critical immunization allowing the complete protection of the network null prevalence is increasing with the spreading rate λ. Equations (3) and (6) indicate that when the value of lethality α increases, the prevalence reduces whether the network is immunized or not.

3. Numerical simulation results

In order to provide further support to the present MF description, by means of numerical simulations, we show the disease spread behaviours of the SIS model on the WS network with feedback mechanism. We consider the parameter $k = 3$, $p = 0.1$ and $N = 5000$ for the WS network. By extensive simulations, the prevalence $\rho(\lambda)$ of network is shown. We start with an initial density of infected nodes $\rho_0 = 5.0 \times 10^{-4}$, and iterate the rules of the SIS model with parallel updating. Each result of the simulations is obtained by averaging over 100 different starting configurations. In Fig.1 we plot the fraction of infected nodes in the stationary state, ρ, for different values of lethality α. Only when λ increases above a value λ_c, a significant prevalence is observed. The effect of the feedback mechanism becomes clear when we compare the shape of the prevalence curves with those obtained without feedback mechanism ($\alpha = 0$). The prevalence ρ is smaller with the larger lethality α when the spreading rate λ has the same value. Just as our prediction, the prevalence density decreases due

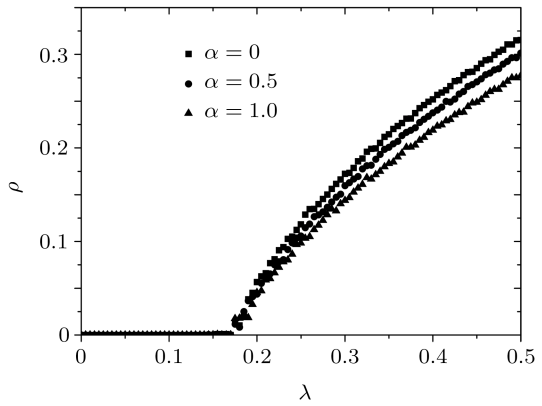


Fig.1. Prevalence ρ (fraction of infected individuals in the stationary state) as a function of the spreading rate λ for the WS network with different constant of lethality α, and the constant of lethality α range from 0 to 1 (from top to bottom).

to increasing the lethality α. Further insight into the time evolution of the behaviour of epidemic is gained from Fig.2. For values λ above λ_c the density approaches a nonzero plateau value. The effect of feedback mechanism is apparent for halting disease spread in the network. In Fig.3, we plot ρ as a function of $(\lambda - \lambda_c)$ in log–log scale. A linear fit to the form $\rho \sim [(\lambda - \lambda_c)/(1+\alpha)]^{\beta}$ provides a constant exponent $\beta = 0.935 \pm 0.002$ which is in excellent agreement with the analytical finding of Eq.(3).

In the presence of uniform immunization with feedback mechanism, we also investigate the system by looking at the infection's prevalence in the stationary regime (endemic state) as a function of the immunity g and lethality α. The uniform immunization is actualized by randomly selecting and immunizing gN nodes on a network of fixed size N. Our simulations are implemented at a fixed spreading rate $\lambda = 0.25$ and $\lambda = 0.4$. In Fig.4, we show the behaviour of reduced prevalence ρ_g as a function of the uniform immunization g and lethality α. In particular, we obtain the threshold immunization $g_c = 0.385$, which is in good agreement with the estimate from Ref.[17]. Here the value of g_c cannot yield from Eq.(7) when $\lambda = 0.25$ and $\lambda_c = 0.16$, we think because each pair of nodes are not connected absolutely randomly. However, just as our prediction, for a certain value of immunity g, the lethality α increases, and the prevalence ρ_g decreases. And comparing Fig.4(a) with Fig.4(b), we find that the effect of feedback mechanism is more apparent for larger spreading rate. For example, for the same value of $g = 0.05$, the prevalence is reduced about 2.0×10^{-2} ($\lambda = 0.25$) and 3.1×10^{-2} ($\lambda = 0.4$) when the lethality $\alpha = 1.0$.

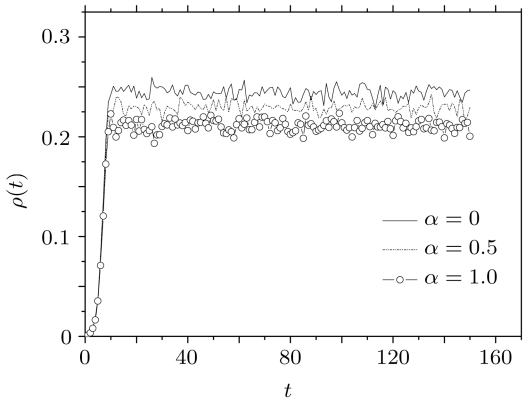


Fig.2. Density of infected nodes $\rho(t)$ as a function of t in WS model with different constants of lethality α when spreading rate $\lambda = 0.25$.

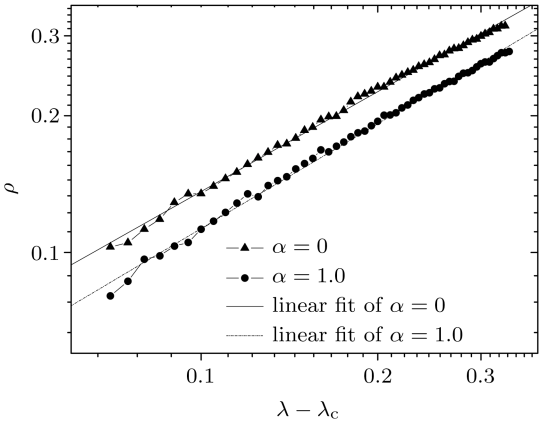


Fig.3. Density of infected nodes ρ in logarithm as a function of $\lambda - \lambda_c$ in WS network with different constant of lethality α. A linear fit to the condition of $\alpha = 0$ and $\alpha = 1$.

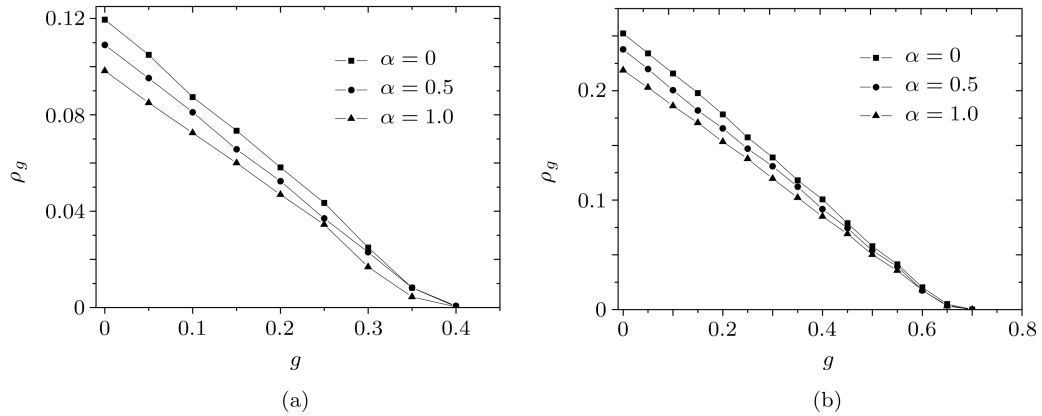


Fig.4. Reduced prevalence ρ_g from computer simulations of the SIS model in the WS network with uniform immunization and feedback mechanism at a fixed spreading rate $\lambda = 0.25$ (a) and $\lambda = 0.4$ (b). Extrapolation of the linear behaviour of ρ_g for the largest immunization values yields an estimate of the critical immunity $g_c \simeq 0.385$ (a) and $g_c \simeq 0.675$ (b).

4. Summary and discussions

Considering the cost for immunization, we find that these results have important practical and economic implications. If the resource is limited, it is only enough to immune a fraction of individuals g ($g < g_c$). Applying feedback mechanism can reduce the prevalence density variously by varying the lethality α. In particular, when spreading rate λ is larger, the effect of this feedback mechanism is more obvious with respect to controlling disease spread.

In summary, we have proposed a novel efficient feedback strategy for immunization. This strategy consider the networks' own adaptivity and the cost for immunization. We have presented analytical and numerical results for the prevalence in the WS network with feedback mechanism. These results confirm that increasing the value of lethality α, we can get reduced density prevalence. Such mechanism does not change the critical immunization. Besides epidemic dynamics, our findings also have strong implications for the spreading of information, opinions and beliefs in a population, which can be described in a similar way.

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