Epidemic spreading with time delay
in complex networks

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Abstract

We present a modified susceptible-infected-susceptible (SIS) model on complex networks, small-world and scale-free, to study epidemic spreading with the effect of time delay which is introduced to the infected phase. Considering topologies of the networks, both uniform and degree-dependent delays are studied during the contagion process. It is found that the existence of delay will enhance both outbreaks and prevalence of infectious diseases in the networks.

Keywords: Dynamics of social systems; Complex networks; Diseases; Critical point phenomena

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1 Introduction

Complex weblike structures describe a wide variety of systems of high technological and intellectual importance and have attracted an increasing interest recently [1, 2, 3]. The explosion of the general interest in the problem of the structure and evolution of most different networks is mainly connected with two characters, the small average path lengths among any two nodes (small-world property) [4] and a power law distribution (scale-free property), $P(k) \sim k^{-\gamma}$ with $2 \leq \gamma \leq 3$, for the probability that any node has $k$ connections to other nodes [5].

In the study of complex networks, a good example is to inspect the effect of their complex features on the dynamics of epidemic and disease spreading. It is easy to foresee that the characterization and understanding of epidemic dynamics on these networks can find immediate applications to a large number of problems, such as computer virus infections [6], epidemiology [7], and the spreading of polluting agents [8], etc. Recent papers [9, 10, 11, 12, 13, 14, 15] have given some valuable insights of that: for small-world networks, there is a critical threshold below which an infection with a spreading rate dies out; on the contrary, for scale-free networks, even an infection with a low spreading rate will prevalence the entire population.

In many social and biological systems, however, temporal delay is natural and the finite time interval required for the information transmission between two elements may be important [16, 17, 18, 19]. In this paper we will introduce time delay to the standard SIS model [7] on two prototype complex networks, the Watts-Strogatz (WS) model and the Barabási-Albert (BA) model. Which is motivated by the following questions: during the process of epidemic spreading, if an individual is infected there is always a period of time before he (or she) becomes recovery, including the time an infected individual is found and sent to a hospital, and the time a patient is being cured, etc.

The paper is organized as follows. In Sec. II we first define the model with
time delay on complex networks. Then we discuss the uniform delay in Sec. III and the degree-dependent delay in Sec. IV. Finally we draw our conclusions and perspectives in Sec. V.

2 The Model

In this section, we shall introduce the effect of time delay to the standard SIS model on complex networks, in which each node represents an individual of the population and the edges represent the physical interactions through which an infection spreads. The two prototype complex networks, WS graph and BA graph, can be constructed as follows.

WS graph: Starting with a ring of $N$ vertices, each connected to its $2K$ nearest neighbors by undirected edges, and then each local link is visited once with the rewiring probability $p$ it is removed and reconnected to a randomly chosen node. Duplicate and self-connected edges are forbidden. After the whole sweep of the entire network, a small-world graph is constructed with an average connectivity $\langle k \rangle = 2K$ (in the present work we will consider the parameters $N = 10^5$, $p = 0.1$ and $K = 5$).

BA graph: Starting from a small number $m_0$ of nodes, every time step a new vertex is added, with $m$ links that are connected to an old node $i$ with probability $\Pi_i = k_i / \sum_j k_j$, where $k_i$ is the connectivity of the $i$th node. After iterating this scheme a sufficient number of times, we obtain a network composed by $N$ nodes with connectivity distribution $P(k) \sim k^{-3}$ and average connectivity $\langle k \rangle = 2m$ (in the present work we will consider the parameters $N = 10^5$, $m_0 = 10$ and $m = 5$).

In our model, an individual is described by a single dynamical variable adopting one of the two stages: susceptible and infected. The two states completely neglect the details of the infection mechanism within each individual. The transmission of the disease is described in an effective way with the following rules: A susceptible
individual at time $t$ will pass to the infected state with the rate $\nu$ at time $t + \Delta t$ if it is connected to one or more infected individuals, where $\Delta t$ is the time step of Monte Carlo (MC) simulations. Infected individuals at time $t$ will pass to the susceptible state again with the rate $\delta$ at time $t + \Delta t + \tau_I$, where $\tau_I$ denotes the delay time in the infected phase. Here, an effective spreading rate $\lambda = \nu/\delta$ is defined. We can still keep the generality by setting $\delta = 1$. Individuals run stochastically through the cycle, susceptible $\rightarrow$ infected $\rightarrow$ susceptible.

In the present work, we have performed MC simulations of the model with synchronously updating in the network. Initially, the number of infected nodes is 5 percent of the size of the network. The total sampling times are 10000 MC time steps. After appropriate relaxation times, the systems stabilize in a steady state. Simulations were implemented on the networks averaging over 100 different realizations. Given a network, an important observable is the prevalence $\rho$, which is the time average of the fraction of infected individuals reached after a transient from the initial condition (averaging over 1000 time steps in this context). The information on the global spreading of infected diseases is contained in the function $\rho(\lambda, \tau_I)$.

3 Uniform delay

We firstly consider that all the individuals in the network have an uniform delay

$$\tau_I = \tau,$$  \hspace{1cm} (1)

that is to say, the details of the delay mechanism within individuals are independent of the connectivity fluctuations of the networks. In our simulations, the system stabilizes in dynamic equilibrium after appropriate relaxation times.

In Fig. 1 under the different values of delay time, the plots of $\rho$ versus $\lambda$ in the WS and $\rho$ versus $1/\lambda$ in the BA networks are shown. In the case of $\tau = 0$, the model becomes the standard SIS model and gives an epidemic threshold $\lambda_c \sim 1/\langle k \rangle$.
in the WS network and $\rho \sim \exp(-1/m\lambda)$ in the BA network, which were firstly introduced by Pastor-Satorras and Vespignani [11] by using mean-field theory. In the presence of time delay ($\tau = 2, 4, 6$), one can easily find that the epidemic prevalence in steady state increases greatly, which induce the epidemic threshold $\lambda_c$ to becomes smaller in the WS network and the scaling effect to become weaker in the BA network.

In Fig. 2 we show the linear-log plots of $\rho$ versus $\tau$ in the WS and BA networks under different values of the effective spreading rate, $\lambda = 0.10$ (full squares), 0.15 (full circles), and 0.20 (full triangles), respectively. Consistently, the epidemic prevalence in steady state increases with the enhancement of the effect of time delay. In addition, both WS and BA networks present a linear relation between $\rho$ and $\ln \tau$, that is, $\rho \sim A + B \ln \tau$, for given effective spreading rate. Simulations indicate that the parameter $B$ is identical for both networks.

To find the relation between the epidemic threshold and time delay in the WS network, we plot $\lambda_c$ as a function of $\tau$ in Fig. 3. Closed squares represent the
Figure 2: Linear-log plots of densities of infected nodes $\rho$ vs $\tau$ in the WS (a) and BA (b) networks under different values of the effective spreading rate (from bottom to top) $\lambda = 0.10, 0.15, \text{and} 0.20$, respectively.

Figure 3: The plot of the epidemic threshold $\lambda_c$ as a function of uniform delay time $\tau$ in the WS network. The solid line is a fit to the form $\lambda_c = C + D e^{-\tau/\tau_0}$. 
numerical results and the solid line is a fit to the form \( \lambda_c(\tau) = C + De^{-\tau/\tau_0} \), which implies there is a relation of the first order exponential decay between \( \lambda_c \) and \( \tau \).

Parameters values (given by simulations) \( C = 0.0113 \pm 0.0007 \), \( D = 0.0713 \pm 0.0002 \), and \( \tau_0 = 2.14 \pm 0.02 \).

## 4 Degree-dependent delay

In all the simulations above, we take homogeneous individual activities in the networks, i.e., the delay is identical for each individual during the evolution of the system. However, considering the heterogeneousness of networks, the distribution of the connectivity, we suggest a degree-dependent delay form

\[
\tau_i = \frac{k_i^{1-\alpha}}{\alpha},
\]

where \( \alpha \) is a tunable parameter. In Eq. (2), the delay time is inversely proportional to \( k_i \), namely, the larger degree a node has, the smaller the delay time the node takes. In language of sociology, \( k_i \) represents the degree of the activity of an individual. So active individuals are easier to be found if they are infected and it will take less time for them to become susceptible again. Here, the details of the recovery mechanism within each individual are completely neglected.

We perform simulations of the model with the same rule and the system reaches a dynamic equilibrium after an initial transient. In Fig. 4 under the different values of \( \alpha \), the plots of \( \rho \) versus \( \lambda \) in the WS and \( \rho \) versus \( 1/\lambda \) in the BA networks are shown. With the reducing of the tunable parameter (from 0.45 to 0.15), the epidemic threshold \( \lambda_c \) becomes smaller in the WS network and the scaling effect become weaker in the BA network. The results are qualitatively consistent with the case of uniform delay since the value of delay time is inverse proportional to the tunable parameter \( \alpha \) (see Eq. (2)).

At the end, the plot of epidemic threshold \( \lambda_c \) as a function of \( \alpha \) in the WS net-
Figure 4: Plots of $\rho$ vs $\lambda$ in the WS (a) and $\rho$ vs $1/\lambda$ in the BA (b) networks under different values of the degree-dependent delay time. Parameter values (from bottom to top) $\alpha = 0.15$, $0.25$, $0.35$, and $0.45$, respectively.

Figure 5: The plot of the epidemic threshold $\lambda_c$ as a function of the tunable parameter $\alpha$ in the WS network. The solid line is a fit to the form $\lambda_c(\alpha) \sim E + F\alpha$. 

work is shown in Fig. 5. Closed squares represent the numerical results and the solid line is a fit to the form $\lambda_c(\alpha) = E + F\alpha$, which predicts a linear relation between $\lambda_c$ and $\alpha$. Parameters values (given by simulations) $E = -0.010 \pm 0.002$, $F = 0.37 \pm 0.01$.

5 Conclusions

We have investigated the spread of infectious diseases with time delay in complex networks. The effect is presented in the infected phase of the standard SIS model. Both the uniform and degree-dependent delays are considered during the contagion process. It was found that the existence of delay will enhance both outbreaks and prevalence of infectious diseases in the networks. However, the results are based on numerical simulations. It deserves to make further study on the theoretical side and explore the connection to real data of diseases. In reality, there always exists a mean incubation period during the spread of epidemics [20, 21]. Our model may provide an explanation for this spreading phenomenon in social systems.

Since time delay arises naturally from the kinetic theory [22, 23], physicists can contribute to topics related to that, such as the explanation of mutant virus strains [24], the modelling of the front shapes in virus infections [25], and the characterization of the speed of virus infections [26], etc.

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