Modeling the influence of information on the coevolution of contact networks and the dynamics of infectious diseases

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**ABSTRACT**

Outbreaks of infectious diseases may awaken the awareness of individuals, consequently, they may adjust their contact patterns according to the perceived risk from disease. In this paper, we assume that individuals make decisions on breaking or recovering links according to the information of diseases spreading which they have acquired. They will reduce some links when diseases are prevalent and have high risks; otherwise, they will recover some original links when the diseases are controlled or present minimal risk. Under such an assumption, we study the effects of information of diseases on the contact patterns within the population and on the dynamics of epidemics. By extensive simulations and theoretical analysis, we find that, due to the time-delayed information of diseases, both the density of the disease and the topology of the network vary with time in a periodic form. Our results indicate that the quality of information available to individuals can have an important effect on the spreading of infectious diseases and implications for related problems.

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**1. Introduction**

The problem of modeling disease spreading among individuals has been investigated intensively for many years. Although mathematical models have been applied to the study of infectious diseases for more than a century [1–3], due to the complexity of contact patterns of population, these traditional mathematical methods have been proven to be somewhat inadequate to reflect the real dynamics of diseases. Hence, the new field of complex network theory offers a better model (in some instances) to study the dynamics of infectious diseases [4–7]. In recent years, the spreading of epidemics on complex networks has been investigated in depth, yet, previous works mainly focused on static networks [8–14]. However, it is clear that the outbreak of an epidemic in human populations triggers individual and collective reactions that can substantially alter the social structure. Subsequently, much interest has grown for a new class of networks known as adaptive networks. In the adaptive networks, if healthy individuals know which of their neighbors is infected, they will break the links with the infected individuals and reconnect to other healthy individuals [15–24].

However, in the real world, on one hand, owing to the latent period of diseases, or the concealment of infected individuals (e.g., for sexually transmitted diseases, some infected individuals will be reticent in allowing their contacts to be aware of their illnesses), it is hard to know the status of individuals; on the other hand, with infectious disease outbreaks, individuals should reduce their connections/activities with the outside world, but not reconnect to others. For example, students do not go to school, workers do not go to work, and so on. Moreover, the links/relationships established by individuals denote their regular activities (e.g., go to school/office, business trip, etc.). If these links are broken, their normal life will be disrupted. So, individuals will recover the original links/relationships after the period of high disease prevalence. Based on the above facts, in this paper, we assume that individuals do not know the status of other persons, they adjust their connections according to the information concerning the disease that they have acquired. If the prevalence of disease is high, they will reduce a certain part of their links with their neighbors; once the density of disease becomes lower (and the expected risk is reduced), they will recover some of their original links again. Meanwhile, even though there are a variety...
of media around us, such as, newspapers, TV and the Internet, it may be impossible to obtain timely and accurate information of diseases. So, we also study the effects of time-delayed information on the decision-making of individuals and then on the dynamics of diseases spreading. Then the sufficient condition for the emergence of the Hopf bifurcation is analyzed. At last the conclusion is extended to general cases.

The layout of the paper is as follows. We introduce the model in Section 2 and present the simulation results in Section 3. Theoretical analysis is studied in Section 4. Finally, conclusions and discussions are presented in Section 5.

2. Model

We consider a susceptible-infected-susceptible (SIS) model on an adaptive network. In the SIS model, infectious (I) individuals contaminate their susceptible (S) neighbors along each link with transmission rate \( \beta \). Meanwhile, the infected individual recovers and returns to the susceptible state again with probability \( \mu \). To focus on the effects of the information of diseases and the response strength of the dynamics of diseases, we fix \( \beta = 0.03 \) and \( \mu = 0.1 \) throughout this paper. Furthermore, we use a random network (ER) [25] as the initial network for the convenience of analysis. Initially, the ER network with size \( N = 2000 \) and average degree of network \( \langle k \rangle = 10 \) (in Fig. 6, we show that other networks also have the same qualitative results as the ER network). Meanwhile, the initial density of infection \( I(0) = 0.01 \), i.e., 1% of nodes are randomly infected and others are susceptible nodes. Each data point presented below results from 30 independent runs in order to assure suitable accuracy.

Suppose that individuals can adjust their links according to the information about diseases they have acquired. If one individual \( i \) has \( k_i(0) \) neighbors initially, then the number of links \( k_i(t) \) at time step \( t \) is given as:

\[
k_i(t) = \text{ceil} \left( \frac{k_i(0)}{1 + \alpha I(t - \tau)} \right), \quad i = 1, 2, \ldots, N; \quad \alpha > 0.
\]

The \( \text{ceil}(\cdot) \) function returns the value of a number rounded upwards to the nearest integer. Where \( \alpha \) is the response strength of individuals to diseases, and \( I(t - \tau) \) is the density of infection at time step \( t - \tau \). Namely, individuals obtain information with a time lag \( \tau \). Time lag \( \tau \) also implies that individuals begin to decide to cut or not some links only after time step \( \tau \), since no one can obtain the epidemic information before \( \tau \). The Eq. (1) means that the higher perceived risk of disease, the fewer the links that remain.

At each time step, individual \( i \) decides to reduce or recover links by comparing \( k_i(t) \) and \( k_i(t - 1) \). If \( k_i(t) < k_i(t - 1) \), then s/he randomly breaks \( k_i(t - 1) - k_i(t) \) links at time \( t \); if \( k_i(t) > k_i(t - 1) \), then s/he randomly recovers \( k_i(t) - k_i(t - 1) \) links; if \( k_i(t) = k_i(t - 1) \), do nothing.

3. Simulation results

At first, the effects of the time delay \( \tau \) and the response strength \( \alpha \) on the density of infection are studied in Fig. 1. As one can expect, with the increase of the value of \( \tau \) and the value of \( \alpha \), the time evolutions of the density of infection will pass from a steady state to a periodic state. Meanwhile, the condition for the emergence of periodic oscillation for the larger value of \( \alpha \) is easier than the smaller value of \( \alpha \). Moreover, for the case of \( \alpha = 2 \) [black links in Fig. 1(a)-(d)], one can find that the density of infection always evolves to a steady state regardless of the values of \( \tau \). From these phenomena we can conclude that there exist critical values of \( \tau_c \) for

![Fig. 1.](image-url)
different $\alpha$ which induces a Hopf bifurcation. In the next section, the sufficient condition for the emergence of this Hopf bifurcation is given by theoretical analysis.

Meanwhile, the impact of the response strength $\alpha$ on final epidemic size with $\tau = 0$ is shown in Fig. 2. As expected, one can find that the final epidemic size decreases with the increase of the value of $\alpha$.

Because individuals adjust their degrees according to Eq. (1), the structure of networks also switches frequently with time steps. As an example, in Fig. 3, we plot the time evolutions of average degree of the network versus $\tau$ for different $\tau$ with $\alpha = 15$. Clearly, the $(k)_1$ also oscillates periodically with time. What’s more, a larger time delay induces a more severe oscillation.

4. Theoretical analysis

In this section, we give some theoretical analysis to obtain the critical values of $\tau_c$ for different values of $\alpha$. By using the mean-field method on a homogeneous network (e.g., random network, small-world network), we can approximately give the equation

$$\frac{dI}{dt} = \beta(k)_1 I(t)(1 - I(t)) - \mu I(t),$$

(2)

where $(k)_1 = \frac{k_i}{\langle k \rangle}$ describes the time evolution of the average degree of network.

The first term of RHS (right-hand side) of Eq. (2) represents the average density of newly infected nodes generated by each active node. This is proportional to the transmission rate $\beta$, the number of links emanating from each node $(k)_1$, and the probability that a given link points to a healthy node, $1 - I(t)$. The second term considers infected nodes become healthy with recovery rate $\mu$ [4].

To obtain the equilibrium of Eq. (2), we set $I(t) = I(t - \tau) = I^*$ and let the RHS of Eq. (2) be zero, so

$$\beta^*(1 - I^*) (k) = \mu I^*(1 + a I^*).$$

(3)

It is easy to show that, if the basic reproductive number $R_0 = \frac{\beta(k)}{\mu} > 1$, then the dynamical Eq. (2) has a unique positive equilibrium

$$I^* = \frac{\beta(k) - \mu}{\mu \alpha + \beta(k)}.$$  

(4)

By setting

$$I = I^* + x$$

and substituting Eq. (5) into Eq. (2) then we have

$$\frac{dx}{dt} - \frac{\beta(k)(x + I^*)(1 - I^* - x)}{1 + a I^* + \alpha x(t - \tau)} - \mu I^* + x.$$  

(6)

By using the first approximation, we have

$$\frac{1}{1 + a I^* + \alpha x(t - \tau)} \approx \frac{1}{1 + a I^*} \left(1 - \frac{x(t - \tau)}{1 + a I^*}\right).$$  

(7)

And then substitute Eqs. (3) and (7) into Eq. (6), then Eq. (6) can be rewritten as

$$\frac{dx}{dt} = \frac{-\beta(k)I^*}{1 + a I^*} x(t) - \frac{\alpha \mu I^*}{1 + a I^*} x(t - \tau).$$  

(8)

The characteristic equation of Eq. (2) at the positive equilibrium $I^*$ is obtained by substituting $x = Ce^{\lambda \tau}$ ($C$ is a constant) into Eq. (6),

$$\lambda = -\frac{\beta(k)I^*}{1 + a I^*} - \frac{\alpha \mu I^*}{1 + a I^*} e^{-\lambda \tau}.$$  

(9)

So when $\tau = 0$, we have

$$\lambda = -\frac{\beta(k)I^*}{1 + a I^*} - \frac{\alpha \mu I^*}{1 + a I^*} < 0.$$  

(10)

So we can conclude that the positive equilibrium $I^*$ is always locally stable when time delay $\tau = 0$.

In the complex plane, eigenvalue $\lambda$ gets closer to the imaginary axis with the increase of time delay $\tau$. If $\lambda$ passes the imaginary axis, a Hopf bifurcation will emerge, i.e., the Eq. (2) transfers from stable state to oscillation state [26]. Thus, by setting $\lambda = iy$, and substituting it into Eq. (9), we have

$$\begin{align*}
\beta(k)I^* & = -\alpha \mu I^* \cos \gamma \tau, \\
\alpha \mu I^* & = \alpha \mu I^* \sin \gamma \tau.
\end{align*}$$  

(11)

From Eq. (11)(a), one have

$$y = \frac{1}{\tau} \left(2n\pi \pm \arccos \frac{-\beta(k)}{\alpha \mu}\right), \quad n = 0, \pm 1, \pm 2, \ldots.$$  

(12)

Combing Eq. (11)(a) and (b) with Eq. (12), and we have

$$\tau_c(n) = \frac{1 + a I^* \left|2n\pi \pm \arccos \frac{-\beta(k)}{\alpha \mu}\right|}{\sqrt{(\alpha \mu)^2 - (\beta(k))^2 I^*}},$$

$$n = 0, \pm 1, \pm 2, \ldots.$$  

(13)
no matter evolutions of the density of infection will reach to steady states analysis is verified by case of Fig. 4


\[ \tau_c = \frac{(1 + \alpha^3)}{\sqrt{(\alpha \mu)^2 - (\beta(k))^2}}. \]  

One can find that the necessary condition for the validity of Eqs. (12)-(14) is \( \alpha \mu > \beta(k). \)

Next we turn to show

\[ \frac{d\Lambda}{dt} \bigg|_{\tau = \tau_c} \neq 0. \]  

This signifies that the eigenvalue \( \Lambda \) has positive real part for \( \tau > \tau_c \). Namely, the smallest critical value \( \tau_c \) can guarantee the emergence of Hopf bifurcation.

From Eq. (9) we have

\[ \frac{d\Lambda}{dt} \bigg|_{\tau = \tau_c} = \frac{\alpha \mu A^*}{1 + \alpha l^*} e^{-\Lambda \tau_c} \neq 0. \]

So Eq. (16) is verified.

According to Eq. (14), the possible behavior of our model can be summarized in a phase diagram on the parameter plane (\( \alpha, \tau \)). As shown in Fig. 4, the phase plane is divided into two regions, i.e., stable region and oscillation region. From Fig. 4, one can find that, if \( \alpha \leq 3 \) the equilibrium \( I^* \) is always stable for any time delay \( \tau \) (the left of the dash line in Fig. 4). It is because that when \( \alpha \leq 3 \) the inequality (15) cannot be satisfied. Moreover, a larger response strength \( \alpha \) induces smaller \( \tau_c \).

To verify the validity of our analysis, Fig. 5 shows that the time evolutions of the density of infection \( I(t) \) for different \( \tau \) and \( \alpha \) according to Eq. (2). As predicted by Fig. 4, periodic oscillation cannot emerge when response strength \( \alpha \leq 3 \) (see the black lines in Fig. 5(a)-(d)). Yet, there exists a critical threshold \( \tau_c \) when \( \alpha > 3 \), namely, the phenomenon of periodic oscillation emerges if \( \tau > \tau_c \); otherwise, the system reaches a steady state. Taking \( \alpha = 10 \) and \( \alpha = 15 \) as examples (see A and B points in Fig. 4.), as given in Fig. 4, for the case of \( \alpha = 10 \) the periodic oscillation will emerge if \( \tau > 31 \); otherwise, the system reaches to a steady state. For the case of \( \alpha = 15 \), the periodic oscillation will emerge if \( \tau > 27 \). The analysis is verified by Fig. 5(b)-(d), as shown in Fig. 5(b), the time evolutions of the density of infection will reach to steady states no matter \( \alpha = 2, 10 \) or 15 when the time delay \( \tau = 20 \). But, when \( \tau = 35 \), the periodic oscillations emerge for the cases of \( \alpha = 10 \) and 15 (see red and blue lines in Fig. 5(d)). Moreover, by comparing Fig. 5 with Fig. 1, one can find that both of them have similar properties, so the simulation results in the above section are supported by theoretical analysis to some extent.

Remark 1. Although our analytical results are obtained on random networks, these results are also valid on more general networks as well. For instance, in Fig. 6(a), we plot the time evolutions of the density of infection on BA network [27] for different values of \( \tau \) with \( \alpha = 15 \). Similar to Fig. 1, with the increase of time delay \( \tau \), the density of infection also passes from steady state to periodic state. At the same time, similar to Fig. 3, the time evolutions of the average degree of BA network also oscillate periodically with time steps.

Remark 2. To simplify the analysis, our simulation results and the theoretical analysis are based on the assumption that each individual has same time lag of information. In reality, different persons have different capabilities for obtaining information. So, we want to know whether the periodic oscillation will occur with heterogeneous time delays. In Fig. 7, we assume the time delay of individuals satisfies uniform distribution \( \tau_i \in [1, 11] \) in Fig. 8. As shown in Fig. 8, there still exist periodic oscillations for the time evolution of the density of infection with \( \alpha = 15 \) and \( \tau = 35 \).

Remark 3. In our model, we assume that the time scales of diffusion of the epidemic and the evolution of network structure are the same, namely, individuals make decision on whether to break or to recover links every time step. Usually, individuals may make decisions after a certain time interval. So we assume the time intervals of individuals satisfies a uniform distribution \( \tau_i \in [1, 11] \) in Fig. 8. As shown in Fig. 8, there still exist periodic oscillations for the time evolution of the density of infection with \( \alpha = 15 \) and \( \tau = 35 \).

Remark 4. Many mechanisms have been suggested by researchers to explain the causes of the periodic oscillation in many diseases, including periodic transmission rate [28], imitation behavior among voluntary vaccination [29], spatial migration in different patches [30], and so on. Here, we proposed a simple coevolutionary mechanism based on time-delayed information of diseases, where individuals make decisions on breaking or recovering links according to the information of diseases spreading which they have acquired. And we have shown that this model, when applied to complex network propagation structures does give rise to the posited periodic oscillations.

5. Conclusions and discussions

In this paper, we have studied a model for an SIS epidemic process in a population of individuals on a complex network, where transmission of diseases can occur along the network links. Owing to the informed and rational response of individuals, the number of neighbors/degrees of individuals varies with the estimated risk of infection. If the current estimated risks are higher than the risks obtained from previous time steps, then individuals will randomly break some of their links; otherwise, some links which have been broken previously will be recovered again if current risks are lower than the risks obtained from previous time steps.

Due to such an adaptive mechanism and the time lag with which information pertaining to the disease spread is obtained, the system passes from steady state to periodic state via a Hopf bifurcation with the increase of the value of \( \alpha \) and the time delay \( \tau \). Then the sufficient condition of Hopf bifurcation is given by theoretical analysis.
Fig. 5. According to Eq. (2), the time evolutions of the density of infection for different response strengths $\alpha$ and different time delays $\tau$ are investigated. (a): $\tau = 0$; (b): $\tau = 20$; (c): $\tau = 30$; (d): $\tau = 35$. Bigger oscillations in (c) and (d) correspond to larger $\tau$.

Fig. 6. The time evolutions of infection (a) and the average degree of network (b) on BA network for different values of $\tau$ investigated. Here $\alpha = 15$.

To simplify our analysis, some stricter conditions are required. For instance, the considered networks are random networks, the time delay is the same for all individuals, and the time intervals of making decision are also the same, and so on. But, of course, in the real world, these assumptions are unrealistic, so we further check our results under more general conditions, e.g., considered networks are scale-free networks, the time delays and the time intervals of making decisions of individuals follow uniform distribution. Encouragingly, we find that our results are rather robust to these conditions. Our work is expected to provide valuable information for understanding the spreading dynamics of epidemics and offering some instructive decision-making.

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The time evolution of infection versus time step \( t \) with time delay \( \tau \) satisfies uniform case \( \tau \in [30, 40] \). Here \( \alpha = 15 \).

The time evolution of the density of infection versus time step \( t \) under the condition where the time interval of making decisions of individuals satisfies uniform case \( T_i \in [1, 11] \). Here \( \tau = 35 \) and \( \alpha = 15 \).

References


