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Effect of incubation period on epidemic spreading in complex networks

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We study the effect of incubation period on epidemic spreading in the Barabasi–Albert scale-free network and the Watts–Strogatz small world network by using a Suspectable-Incubated-Infected-Suspectable model. Our analytical investigations show that the epidemic threshold is independent of incubation period in both networks, which is verified by our large-scale simulation results. We also investigate the effect of incubation period on the epidemic dynamics in a supercritical regime. It is found that with the increase of incubation period Ω, a damped oscillation evolution of ρ_T (the ratio of persons in incubated state) appears and the time needed to reach a saturation value increases. Moreover, the steady value of ρ_T increases and approaches to an asymptotic constant with the value of Ω increasing. As a result, the infected ratio ρ_I decreases with the increase of Ω according to a power law.

Keywords: epidemic spreading; incubation period; complex network
PACC: 0250

1. Introduction

In recent years, network structure and dynamics have attracted much attention. Many social, biological, neural, and technological systems can be described by complex networks whose nodes represent individuals or units, and links refer to the interactions or relationships among them.[1] Because of their small average path lengths between any two nodes (small-world property) or a power law degree distribution (scale-free property, $P(k) \sim k^{-\gamma}$ with $2 \leq \gamma \leq 3$), the Watts–Strogatz (WS) small world network[2] and the Barabasi–Albert (BA) scale-free network[3] are often used as a framework to investigate the dynamic behaviours of many physical process, e.g. epidemic and rumour spreading, evolutionary game, synchronization behaviour, and packet traffic.[4]

Epidemic spreading behaviour is an interesting and important domain in the research field of complex networks.[5,6] Actually, the epidemiology has grown so rapidly since the middle of the 20th century that a variety of models have been formulated, analysed mathematically, and applied to this field research.[7−27] On the other hand, physicists are interested in these kinds of models because the epidemic process can be regarded as dynamic behaviour in complex networks. Some papers have given several valuable results: for the WS small-world network, there is found a critical threshold below which an infection dies out;[2] on the contrary, for the BA scale-free network, an infection even with a low spreading rate will prevail in the entire population.[5,6] These researches are of practical importance to guide disease prevention and immunization.

In reality, many diseases spread with an incubation period, and the incubation plays an important role in the spreading process.[17−27] In most studies concerning the incubation period, the models are usually presented as a system of discrete time delay differential equations.[17−23] Ahmed and Agiza have studied phase transitions in some modified versions of 1D suspectable-infected-recovered-suspectable (SIRS) model including incubation in Refs.[24, 25]. Agiza *et al*[26] have generalized this work to small world networks. Grabowski and Rosinska[27] have investigated the SIRS model with incubation time. Nevertheless, the effects of incubation period on the ratio of suspectable persons, the ratio of infected persons and the epidemic threshold have not been studied yet.

Studied in the present paper are the effects of incubation period on epidemic spread in the BA scale-free network and the WS small world network by using a suspectable-incubated-infected-suspectable (SIIS) model. The dependences of the ratio of suspectable persons and the ratio of infected persons on incubation period are studied. It is also found that the epidemic threshold is independent of incubation period in both networks.

The remainder of the present paper is organized as follows. In Section 2, the model is introduced. Our analytic investigations on epidemic threshold are presented in Section 3 and numerical simulations are shown in Section 4. Finally, the conclusions drawn from the present study are given in Section 5.

2. Model

The SIIS model is built based on the classic suspectable-infected-suspectable model.[5,6] In the epidemic process, individuals exist in three discrete states: susceptible (healthy) (S), infected (I) and incubated (T_ω, ω refers to the time that the epidemic has incubated). At each time step, each susceptible node is infected and changed to the incubation state with rate ν if it is connected to one or more infected nodes:

$$S(i) + T(j) \xrightarrow{v} T_{\omega=0}(i) + I(j), \tag{1}$$

where i and j denote two neighbours. All the nodes which have stayed in an incubation state increase their incubation time by one when the value of ω is smaller than incubation period Ω

$$T_\omega(i) \to T_{\omega+1}(i). \tag{2}$$

If ω equals Ω, the incubated node changes to an infected state, i.e.

$$T_{\omega=\Omega}(i) \to I(i). \tag{3}$$

Here we assume that the incubated epidemics do not infect the healthy persons. At the same time, infected nodes are cured and become susceptible again with rate Ω, i.e.

$$I(i) \to S(j). \tag{4}$$

The model is named SIIS because of the four dynamic processes from formula (1) to formula (4). Here, as usual, an effective spreading rate λ is defined as $\lambda = \nu/\delta$.

3. Epidemic threshold

In this section, we investigate the effect of incubation period on epidemic threshold. We denote the ratio of susceptible persons as ρ_S, the ratio of infected persons as ρ_I, and the ratio of persons in incubated state as ρ_T. It is obvious that

$$\frac{\mathrm{d}\rho_I}{\mathrm{d}t} = -\rho_I\delta + \frac{\rho_T}{\Omega}, \tag{5}$$

where $-\rho_I\delta$ is the number of persons recovered from disease, and ρ_T/Ω is the number of persons that change to infected state from incubated state.

To start with, we consider the WS network. Owing to its exponential degree distribution, the fluctuations in the connectivity are very small in the WS network. Therefore, we assume that all nodes have the same number of the links, i.e. $k \approx \langle k\rangle$. Thus, we have

$$\frac{\mathrm{d}\rho_T}{\mathrm{d}t} = -\frac{\rho_T}{\Omega} + (1-\rho_I-\rho_T)\nu < k > \rho_I, \tag{6}$$

where $(1-\rho_I-\rho_T)\nu < k > \rho_I$ that is the number of persons that have changed to the incubated state from the suspectable state. In the steady state, $\mathrm{d}\rho_I/\mathrm{d}t = \mathrm{d}\rho_T/\mathrm{d}t = \mathrm{d}\rho_T/\mathrm{d}t = 0$. Therefore, we have

$$\rho_I = \frac{1}{\lambda(1+\delta\Omega)}(\lambda - \lambda_c). \tag{7}$$

Define the threshold λ_c as $\lambda_c = 1/\langle k\rangle$, then expression (7) will become

$$\rho_I = 0, \quad \text{if} \quad \lambda < \lambda_c; \tag{8}$$

$$\rho_I \approx \lambda - \lambda_c, \quad \text{if} \quad \lambda > \lambda_c. \tag{9}$$

This means that the threshold is independent of incubation period Ω.

Figure 1 shows the relations for ρ_I versus in log-log scale. One can see that at different values of Ω, the same epidemic threshold $\lambda_c = 0.1643 \pm 0.01$ is approached linearly by ρ_I.

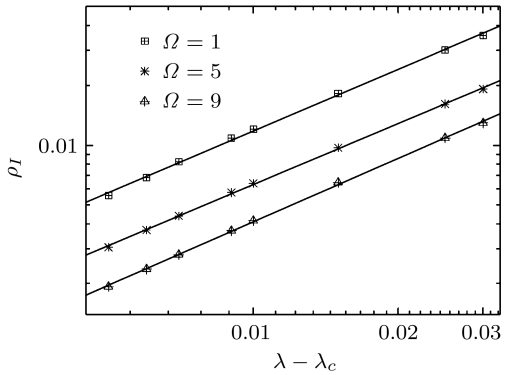


Fig.1. Log-log plots for density of infected nodes versus $\lambda-\lambda_c$ in WS network, where the WS network is generated by letting probability $p = 1$ and average degree $\langle k\rangle = 6$, the parameter $\delta = 0.9$, the network size is $N = 10^6$, and straight lines serve as the guide for eye viewing.

Now we come to consider the BA network. In the BA network, the strong fluctuations in the connectivity distribution cannot be neglected. In order to

take into account these fluctuations, we consider the relative density of infected nodes with a given connectivity, i.e. the probability with which a node with k links is infected. The dynamical equation of $\rho_{T,k}$ can be written as

$$\frac{\mathrm{d}\rho_{T,k}}{\mathrm{d}t} = -\frac{\rho_{T,k}}{\Omega} + (1-\rho_{I,k}-\rho_{T,k})\nu k\Theta. \quad (10)$$

The creation term considers the probability $(1-\rho_{I,k}-\rho_{T,k})$ with which a node with k links is susceptible and becomes infected via a connected node. The probability of this last event is proportional to the infection rate ν, the number of connections k, and the probability Θ with which any given link points to an infected node. As done in the previous studies,[5,6] we neglect the connectivity corrections, i.e. we consider that the probability with which a link pointing to an infected node does not depend on the connectivity of the emanating node and is only a function of the total density of infected nodes that the link points to. Similarly, we have the dynamical equation of $\rho_{I,k}$

$$\frac{\mathrm{d}\rho_{I,k}}{\mathrm{d}t} = -\rho_{I,k}\delta + \frac{\rho_{T,k}}{\Omega}. \quad (11)$$

In the steady state, we can obtain

$$\rho_{I,k} = \frac{k\lambda\Theta}{1+k\lambda\Theta(1+\delta\Theta)} \quad (12)$$

from Eqs.(10) and (11). It is known that the probability with which a link points to a node with s links is proportional to $sP(S)$, i.e. a randomly chosen link is more likely to be connected to an infected node with a high connectivity. Thus,

$$\Theta = \sum_k \frac{kP(k)\rho_{I,k}}{\sum_s sP(S)}. \quad (13)$$

In the BA network, the degree distribution is given by $P(k) = 2m^2k^{-3}$ where m is the minimum degree of the nodes. Computing the average degree $\langle k\rangle = \int kP(k)\mathrm{d}k = 2m$, equation (13) together with Eq.(12) gives

$$\Theta = m\lambda\Theta\int_m^\infty \frac{1}{k[1+k\lambda\Theta(1+\delta\Omega)]}\mathrm{d}k. \quad (14)$$

This yields

$$\Theta = \frac{1}{(\mathrm{e}^{1/m\lambda}-1)(1+\delta\Omega)\lambda m}, \quad (15)$$

which can be reformulated as

$$\Theta = \frac{\mathrm{e}^{-1/m\lambda}}{m\lambda(1+\delta\Theta)(1-\mathrm{e}^{-1/m\lambda})} \approx \frac{\mathrm{e}^{-1/m\lambda}}{m\lambda(1+\delta\Omega)} \quad (16)$$

at lowest order in λ. The total density of the infected nodes can be written as

$$\rho_I = \sum_k P(k)\rho_{I,k}. \quad (17)$$

Substituting expressions (12) and (16) into expression (17) and solving the integral, we have

$$\rho_I \approx \frac{1}{1+\delta\Omega}\mathrm{e}^{-1/m\lambda}. \quad (18)$$

The result indicates that the epidemic threshold $\lambda_c = 0$ in the BA network, which is also independent of incubation period Ω. Figure 2 shows the plot for ρ_I versus $1/\lambda$ in a semi-log plane. The curves confirm that $\lambda_c = 0$, no matter what the value of Ω is.

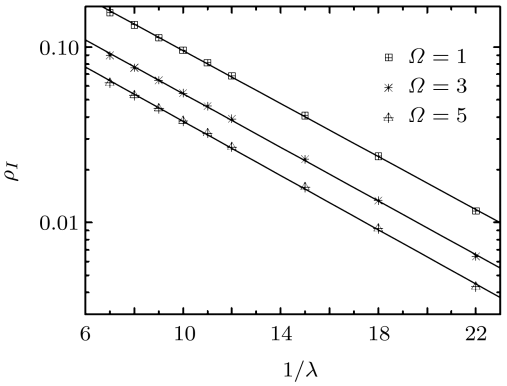


Fig.2. Semi-log plots for ρ_I versus $1/\lambda$ in BA network, where the BA network is generated with $m_0 = m = 5$, the parameter $\delta = 0.9$, the network size is $N = 10^6$, and the straight lines serve as the guide for eye viewing.

4. Simulations

In this section, simulations are performed to investigate the effect of incubation period on the epidemic dynamics in the supercritical regime $\lambda > \lambda_c$. Figure 3 shows the evolution of ρ_T with time at different values of Ω in the BA network. Initially 0.1% of nodes are in an infected state and the others are in a susceptible state. One can see that when the value of Ω is small (Fig.3(a)), the incubated ratio increases with time in an algebraic form, accompanied with a slight oscillation. With the value of Ω increasing, the oscillation is greatly strengthened (Fig.3(b)). The oscillation becomes damped with time until a saturation value is reached. When the value of Ω is large, a stepwise constant damped oscillation occurs (Fig.3(c)). We also note that with the increase of Ω, the time needed to reach the saturation value increases. In the WS network, similar results are observed except that the oscillation appears at a larger value of Ω and its amplitude is much smaller (see Fig.4).

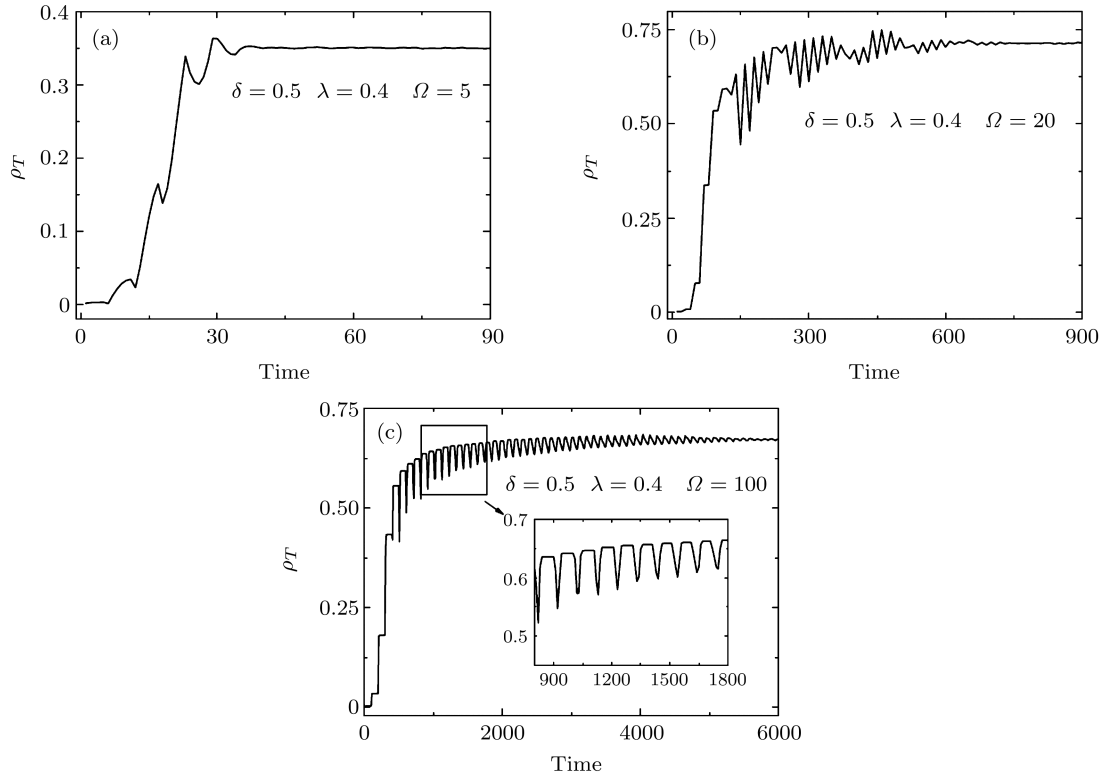


Fig.3. Curves for evolutions of ρ_T with time in BA network with $\Omega = 5$ (a), 20 (b), 100 (c) and the network size is $N = 10^6$.

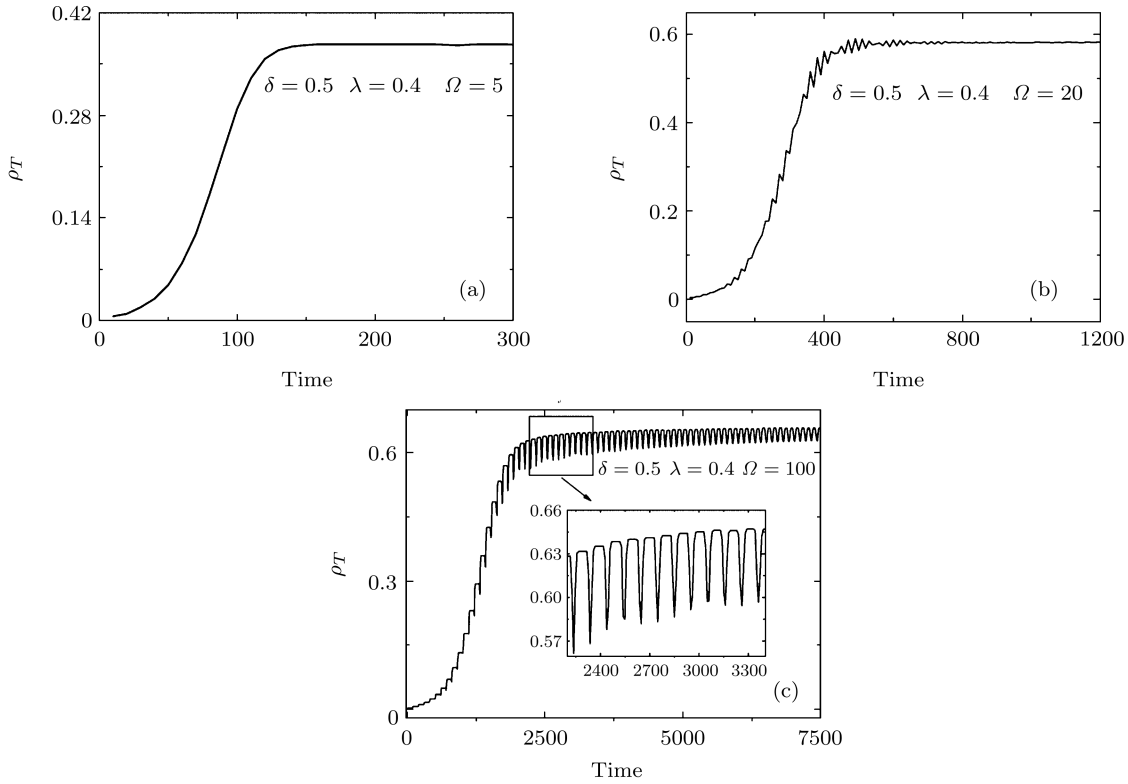


Fig.4. Evolution of ρ_T with time in WS network at $\Omega = 5$ (a), 20 (b), 100 (c), and network size $N = 10^6$.

Figure 5(a) shows the dependence of the steady value of ρ_T on the incubation period Ω in the BA network. With the increase of Ω, the steady value of ρ_T increases and approaches to an asymptotic constant. As a result, when the value of Ω is large, we have

$$\rho_I = \frac{\rho_T}{\delta}\frac{1}{\Omega} \tag{19}$$

from Eq.(5), i.e. ρ_I is inversely proportional to Ω. Figure 5(b) shows the log-log plot for the steady value of ρ_I versus the value of Ω. A linear fitting to the form $\rho_I \sim \Omega^{\beta}$ provides exponent $\beta = -1 \pm 0.04$, which is in good agreement with that obtained from expression (19).

We have also performed simulations within the WS network and similar results are obtained (not shown).

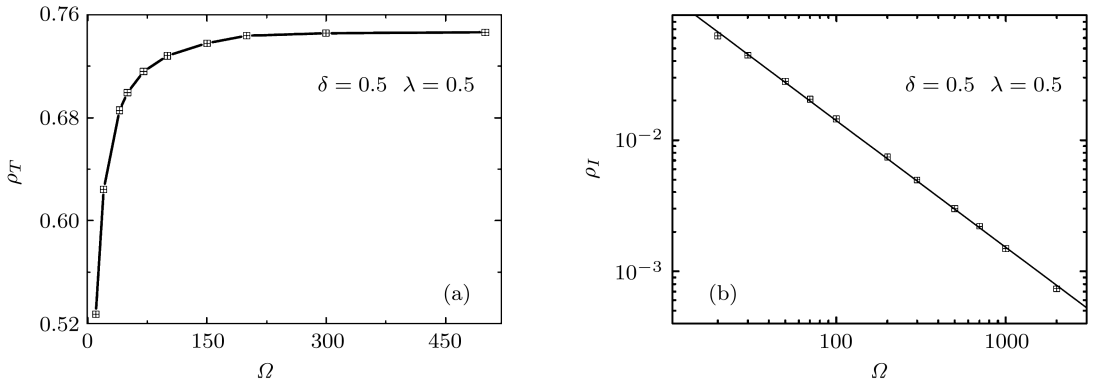


Fig.5. Dependences of (a) ρ_T and (b) ρ_I on incubation period Ω in BA network, with network size $N = 10^6$.

5. Conclusions

In reality, many diseases spread with an incubation period, and the incubation plays an important role in the spreading process. Nevertheless, the effects of incubation period on the ratio of suspectable persons, the ratio of infected persons, and the epidemic threshold have not been studied yet.

We carry out analytical investigations on the epidemic threshold in the BA network and the WS network by using an SIIS model. It is found that the epidemic threshold is independent of incubation period in both networks, which is verified by our large scale simulation results.

We also investigate the effect of incubation period on the epidemic dynamics in the supercritical regime ($\lambda > \lambda_c$). It is found that with the increase of incubation period, a damped oscillation of ρ_T with time appears and the time needed to reach a saturation value increases. Moreover, it is also found that the steady value of ρ_T increases and approaches to an asymptotic constant with the increase of Ω. As a result, ρ_I decreases with the increase of Ω according to a power law.

In the present paper, a constant incubation period is considered. The effect of heterogeneous distribution of incubation period needs studying further in our future work.

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