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Immunization of susceptible–infected model on scale-free networks

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Abstract

In this paper, we investigate two major immunization strategies, random immunization and targeted immunization, of the susceptible–infected (SI) model on the Barabási–Albert (BA) networks. For the heterogeneous structure, the random strategy is quite ineffective if the vaccinated proportion is small, while the targeted one which prefers to vaccinate the individuals with the largest degree can sharply depress the epidemic spreading even only a tiny fraction of population are vaccinated. The analytical solution is also obtained, which can capture the trend of velocity change vs. the amount of vaccinated population.

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Keywords: Scale-free networks; Epidemic spreading; SI model; Immunization

1. Introduction

Epidemic dynamics, one of the attracting problems in both biological and physical communities, aims at explaining the dynamical processes of disease spreading, computer virus prevailing, and so on. The previous investigations based on the differential equations are always under the assumption of both homogeneous infectivity and homogeneous connectivity of each individual [1,2]. Denoting the possible contacts, along which the infection spreads, by edges, then the classical studies on epidemic dynamics can be considered as the disease spreads along complete or random networks. However, against the above assumption, the empirical data of real networks indicate the universal existence of heterogeneous topologies [3–5]. One intriguing finding is that many real networks have approximately power-law degree distributions, that is to say, the probability distribution function of degree, $P(k)$, approximately obeys the form $P(k)\sim k^{-\gamma}$, with $2<\gamma<3$. This kind of distribution implies an unexpected abundance of vertices with very large degrees, i.e. the so-called ''hubs'' or ''super-spreaders''. This series of networks, named scale-free (SF) networks, attract many researchers to investigate the corresponding epidemic behaviors (see the review paper [6] and the references therein).

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The most exemplary models in this field, having been extensively lucubrated, are the susceptible–infected–susceptible (SIS) and susceptible–infected–removed (SIR) models. The recent works about SIS [7,8] and SIR [9,10] models on SF networks present us with completely new epidemic propagation scenarios that a highly heterogeneous structure will lead to the absence of any epidemic threshold. However, many real epidemic processes cannot be properly described by the above two models. In this paper, we especially focus on the onset dynamics of epidemic outbreaks, whose behavior is governed by the pure prevalence without the natural recovery or removing. That is to say, a speeding time-scale is much smaller than the recovery time-scale. This process is usually used to mimic the case when the speed of the disease is so drastic that the effect of recovery and death can be ignored. In addition, in some broadcasting processes, each node is in the possession of two discrete state, *received* or *unreceived*. Different from the SIS or SIR model, the ones having received signals will not alter back to the state unreceived. Hence, it is more proper to utilize the so-called susceptible–infected (SI) model to describe those dynamic processes, in which the infected nodes stay infected and spread the infection to the susceptible neighbors with rate λ.

Very recently, Barthélemy et al. [11,12] studied the SI model in Barabási–Albert (BA) networks [13], and found that this epidemic process has an infinite spreading velocity in the limit of infinite population. Following a similar process on *random Apollonian networks* and weighted SF networks, Zhou et al. investigated the effects of clustering [14] and weight distribution [15] on SI epidemics. By using the theory of branching processes, Vázquez obtained a more accurate solution about the time behavior of SI model [16]. The SI model with identical infectivity, which leads to a slower spreading in SF networks than the standard model, has recently been investigated by Zhou et al. [17]. And the geographical effect on SI model is studied by Xu et al. [18,19]. Although these previous works are very helpful to the deeply understanding of SI epidemic, compared with the extensively studied SIR and SIS models, the SI model has not been carefully investigated thus far. Especially, the immunization effect on SI dynamics, which is very important for controlling the prevalence, has not yet been investigated. In this paper, we focus on the immunization effect of SI model on SF networks, which can be considered as a complementary work of the previous studies on the immunization of SIR and SIS models.

2. The model

In the standard network SI model, each individual is represented by a node of the network and the edges are the connections between individuals along which the infection may spread. Each individual can be in two discrete states, either susceptible or infected. The infection transmission is defined by the spreading rate λ at which each susceptible individual acquires the infection from an infected neighbor during one time step.

Using the mean-field theory, the reaction rate equations can be written as [11,12]

$$\frac{\mathrm{d}i_k(t)}{\mathrm{d}t} = \lambda k(1 - i_k(t))\Theta_k(t), \tag{1}$$

where $i_k(t)$ denotes the density of infected individuals with degree k, $\langle k\rangle$ the average degree, and Θ_k the density of the infected neighbors of a k-degree node. Neglecting terms of order $\mathbb{O}(i^2)$, the evolution behavior, $i(t) = \sum_k i_k(t)P(k)$, can be approximately solved as [11,12]

$$i(t) \sim \mathrm{e}^{ct}, \quad \text{with } c \sim \langle k^2\rangle / \langle k\rangle. \tag{2}$$

In an SF networks with a degree distribution exponent $2 < \gamma \leqslant 3$, the second-order moment $\langle k^2\rangle$ will approach infinity as the increase of network size N, indicating an infinite velocity in the onset of epidemic spreading.

In Fig. 1, we report the simulation results about the time evolution of infected density with one randomly selected node to be infected initially. All the simulations are implemented on the BA networks [13], which can be constructed by continuously adding one node with m edges connected to the existing nodes relying on the probability proportional to their degrees. The advantage with the BA model is that it is the mostly studied and lacks structural-biases such as none-zero degree–degree correlations. Clearly, the epidemic spreading if very fast, and in the early stage, $i(t)$ follows an exponential form.

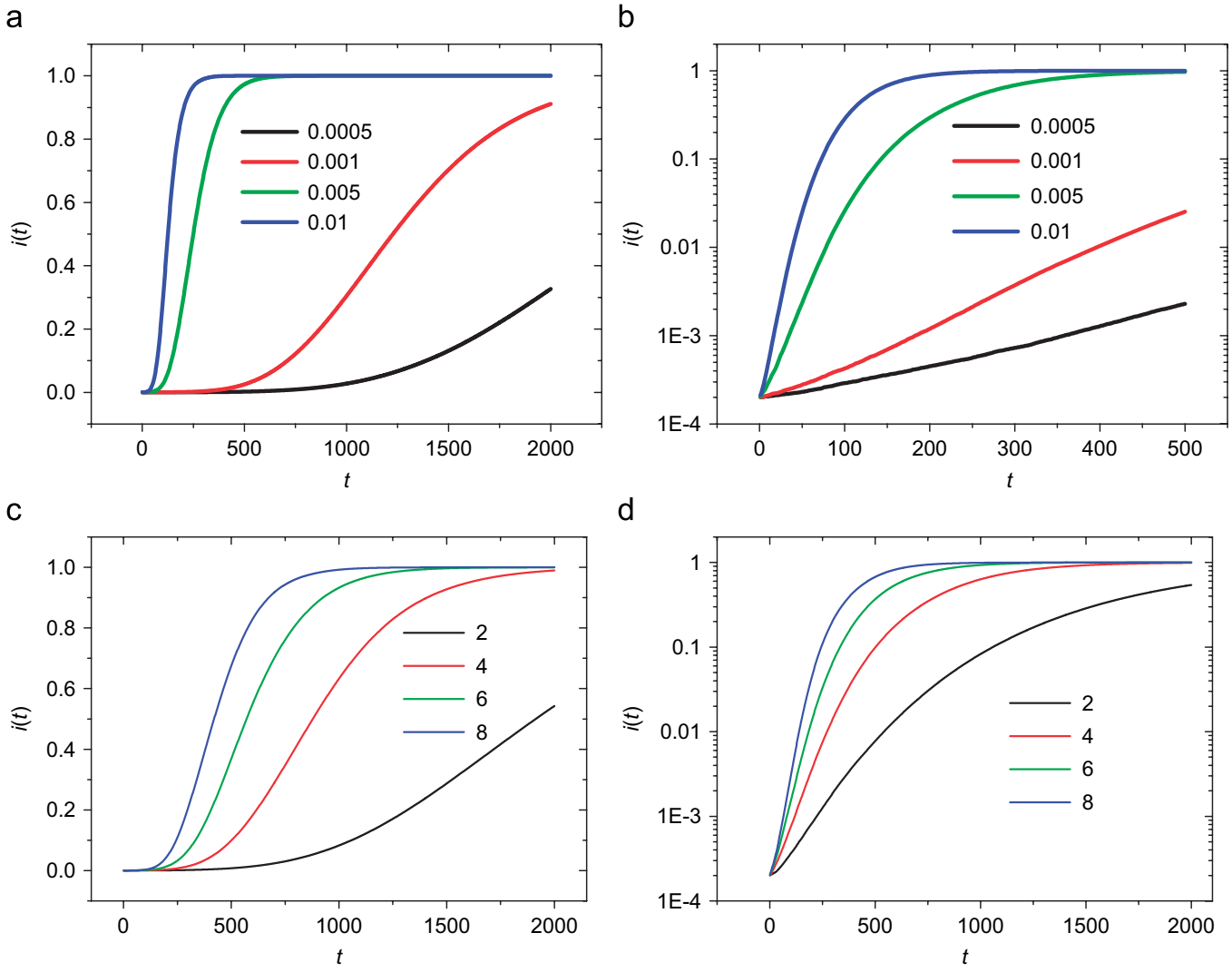

Fig. 1. (Color online) The infected density $i(t)$ vs. time. The four plots exhibit the time evolution of $i(t)$ for different spreading rates λ in normal (a) and single-log (b) coordinates, and for different minimal degree m in normal (c) and single-log (d) coordinates, respectively. The numerical simulations are implemented based on the BA network of size $N = 5000$. In plots (a) and (b), the average degree of BA networks is fixed as $\langle k \rangle = 6$, and in the plots (c) and (d), the spreading rate is fixed as $\lambda = 0.001$. The legends in panels (a) and (b) denote the different spreading rates, and the legends in panels (c) and (d) denote the different minimal degrees. All the data are averaged over 1000 independent runs.

3. Immunization effect

Immunity is a practical controlling strategy to the prevalence of the disease. The most extensively investigated approaches is the so-called *mass vaccination* [1,20] (or called *random immunization*). In random immunization, a fraction f of the whole population is randomly selected to be vaccinated in advance. The most significant problem is that whether it is effective for the highly heterogeneous networks? In the previous works, by using the mean-field theory and branch process theory, Callaway et al. [21] and Cohen et al. [22], separately but almost at the same time, both proved that the random immunization is of less effectivity for SIR model on SF networks.

In Fig. 2, we plot the time evolution of infected density $i(t)$ for different immunization range f, which is defined as the fraction of population being selected to be vaccinated. From Fig. 2, one can find that the spreading velocity has almost no change if only a very few individuals are selected to be vaccinated. Therefore, similar to the situations for SIR model, the random immunization is of less effectivity for SI model on SF networks.

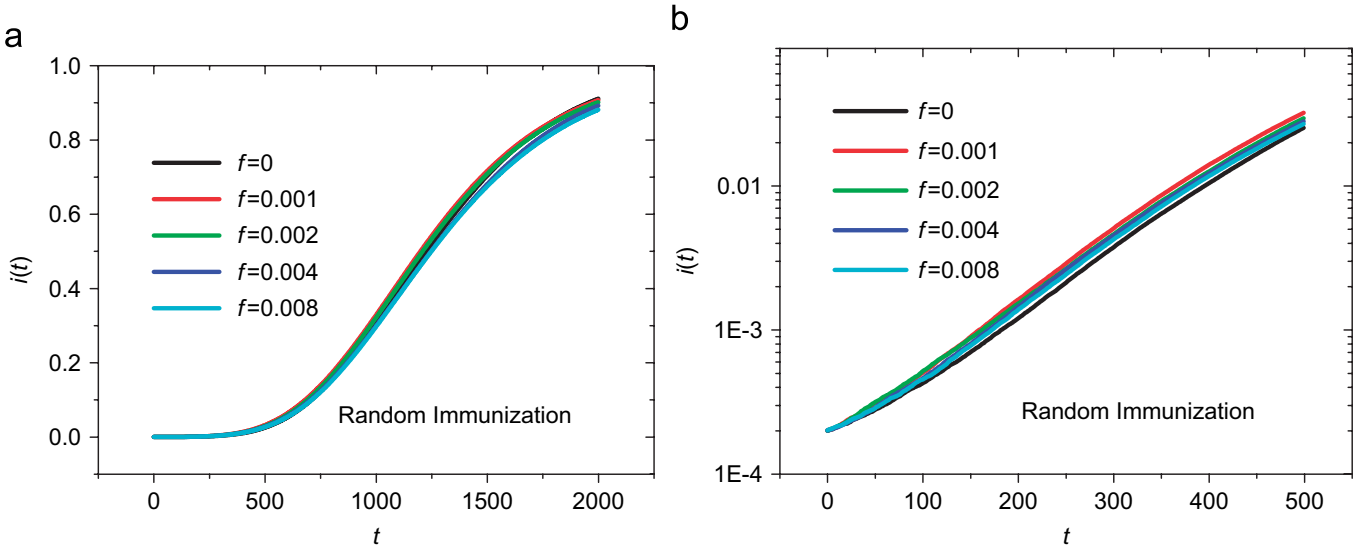

Fig. 2. (Color online) The infected density $i(t)$ vs. time under random immunization for the SI model based on BA networks in normal (a) and single-log (b) plots. The network size $N = 5000$, the minimal degree $m = 3$, and the spreading rate $\lambda = 0.001$ are fixed. The black, red, green, blue and sky-blue curves, from top to bottom, represent the cases of $f = 0$, 0.001, 0.002, 0.004 and 0.008, respectively. All the data are averaged over 1000 independent runs.

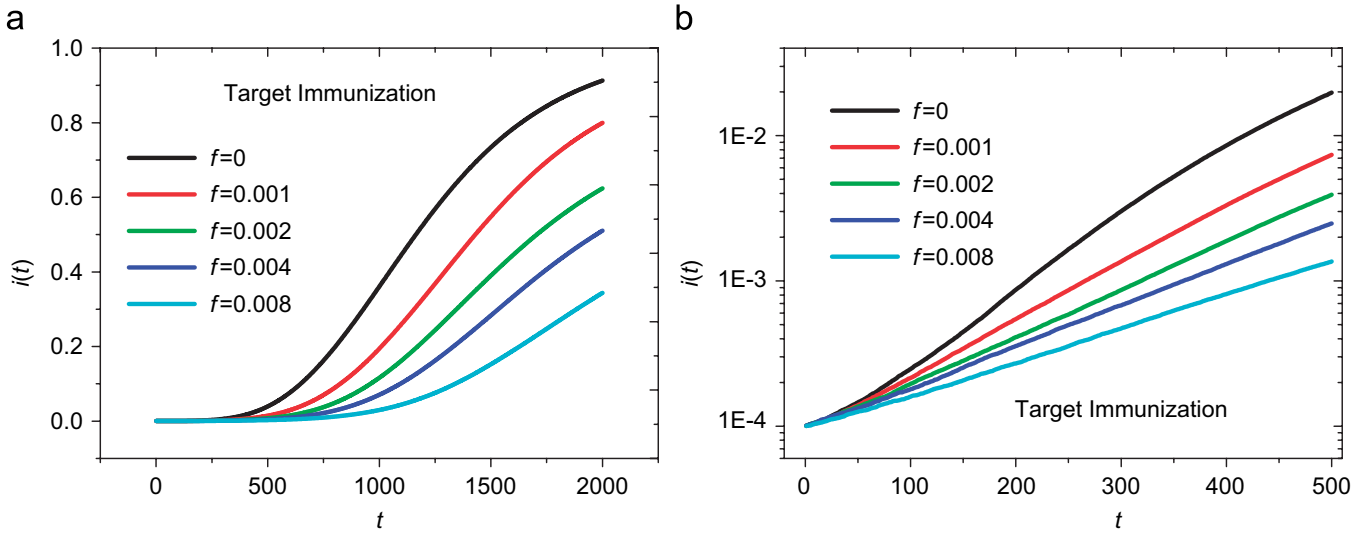

Fig. 3. (Color online) The infected density $i(t)$ vs. time under targeted immunization for the SI model based on BA networks in normal (a) and single-log (b) plots. The network size $N = 5000$, the minimal degree $m = 3$, and the spreading rate $\lambda = 0.001$ are fixed. The black, red, green, blue and sky-blue curves, from top to bottom, represent the cases of $f = 0$, 0.001, 0.002, 0.004 and 0.008, respectively. All the data are averaged over 1000 independent runs.

Other than the random immunization, if the degree of each node is known, one recently proposed efficient immunization strategy is the so-called *targeted immunization* [23–25], which means to vaccinate the nodes with the largest degrees first. Fig. 3 shows the effect of targeted immunization for different f. The spreading velocity remarkably decreases even only a small fraction, $f = 0.001$, of population get vaccinated, which strongly indicates the efficiency of the targeted immunization. From Fig. 3(b), it is observed that the time scale governing the epidemic behavior in the early stage sharply changes even only 10^{-3} fraction of population (i.e., five nodes) are vaccinated.

Consider an SF network of size N, the degree distribution, $P(k) = Ak^{-\gamma}$, obeys the following normalized condition:

$$\int_m^M Ak^{-\gamma}\,\mathrm{d}k = 1, \tag{3}$$

where A is a normalized constant, M the maximal degree and m the minimal degree. We assume after the fraction f of population with largest degrees having been vaccinated, the maximal degree decreases to $k_c(f)$, and if f is sufficiently small so that the degree distribution still obeys a power-law form with exponent γ almost unchanged, then

$$\int_m^{k_c(f)} Ak^{-\gamma}\,\mathrm{d}k = 1 - f. \tag{4}$$

Following the mean-field theory [11,12], the time evolution of $i(t)$ in the early stage approximately obeys an exponential form $i(t) = i(0)\mathrm{e}^{\tau(f)t}$, where $i(0) = 1/N$ is the initial infected density. The time scale $\tau(f)$ can be obtained as

$$\tau(f) = \lambda\left(\frac{\int_m^{k_c(f)} k^2 P'(k)\mathrm{d}k}{\int_m^{k_c(f)} kP'(k)\mathrm{d}k} - 1\right), \tag{5}$$

where $P'(k)$ is the degree distribution of the network after vaccination (i.e. after the removal of Nf nodes of largest degrees), which reads

$$P'(k) = \frac{1}{1-f}P(k), \quad k = m, m+1, \ldots, k_c(f). \tag{6}$$

In the large-limit of N, the maximal degree in the original network, $M \sim N^{1/(\gamma-1)}$, approaches to infinite. Combine Eqs. (2)–(5), the time-scale $\tau(f)$ after targeted immunization in the $N \to \infty$ limit can be analytically obtained, for any $\gamma \in (2, 3)$, as

$$\tau(f) = \lambda\left(m \times \frac{2-\gamma}{3-\gamma} \times \frac{f^{(3-\gamma)/(1-\gamma)} - 1}{f^{(2-\gamma)/(1-\gamma)} - 1} - 1\right). \tag{7}$$

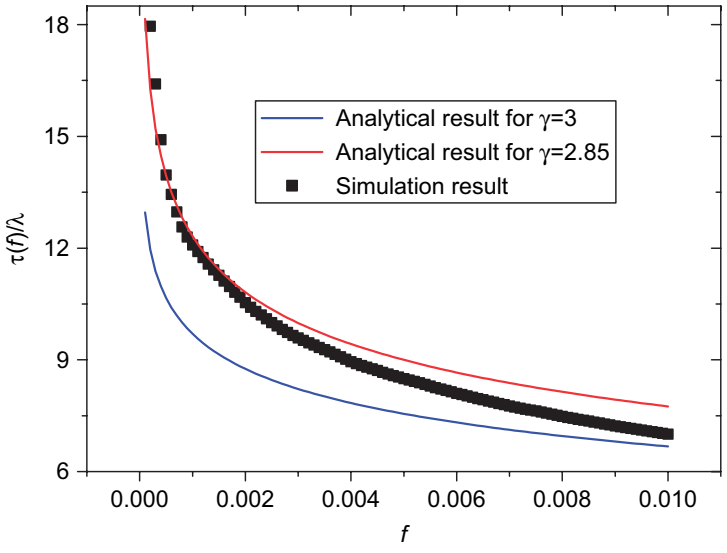

Fig. 4. (Color online) The rescaled time scale τ/λ vs. immunization fraction f. The black squares represent the simulation result based on BA networks. The network size $N = 10\,000$, the minimal degree $m = 3$, and the spreading rate $\lambda = 0.001$ are fixed. The blue and red curves denote the analytical results for $\gamma = 3$ and $\gamma = 2.85$, respectively. All the data are averaged over 1000 independent runs.

Especially for the BA networks with $\gamma = 3$, the analytical result is

$$\tau(f) = \lambda\left(\frac{m \ln f}{2(\sqrt{f} - 1)} - 1\right). \tag{8}$$

In Fig. 4, we report the numerical and analytical results for BA networks with $N = 10\,000$ and $m = 3$. Although the analytical result for BA networks (e.g. Eq. (8), shown as the blue curve) can capture the trend of $\tau(f)$, the quantitative departure is very obvious. Note that, the fitting value of γ in finite size BA networks is smaller than 3.0, which will lead to an even broader distribution than $P(k) \sim k^{-3}$ thus a faster spreading than the theoretical prediction. We have obtained the average fitting value of γ as $\overline{\gamma} \approx 2.85$, over 100 independent configurations of BA networks with $N = 10\,000$ and $m = 3$. The red curve in Fig. 4 represents the analytical result for the modified exponent 2.85 following Eq. (7), one can see clearly that it agrees well with the simulation for small f and can capture the trend of $\tau(f)$. For larger f, the assumption that the degree distribution still obeys a power-law form with same exponent after the removal of Nf hub nodes will not be valid, resulting in the observed departure.

4. Conclusion

As an important branch of the studies on epidemic spreading, immunity never loses its attraction. Some striking conclusion somewhat changes our opinions about epidemic. However, despite of the well-studied SIS and SIR model, the immunization effect on the outbreaks of epidemic spreading, of significantly practical value, has not been carefully investigated thus far. The purpose of this paper is to provide a complementary work of the previous studies on the immunization of SIR and SIS models.

Two major immunity strategies are investigated based on the BA networks. The random immunization is of less effectivity while the targeted immunization can sharply depress the spreading velocity even only a very few hundreds of nodes are vaccinated. Furthermore, the analytical results is obtained which agree with the simulation well for sufficiently small immunization fraction.

Acknowledgments

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