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Epidemic dynamics on scale-free networks with piecewise linear infectivity and immunization

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We examine epidemic thresholds for disease spread using susceptible-infected-susceptible models on scale-free networks with variable infectivity. Infectivity between nodes is modeled as a piecewise linear function of the node degree (rather than the less realistic linear transformation considered previously). With this nonlinear infectivity, we derive conditions for the epidemic threshold to be positive. The effects of various immunization schemes including ring and targeted vaccination are studied and compared. We find that both targeted and ring immunization strategies compare favorably to a proportional scheme in terms of effectiveness.

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I. INTRODUCTION

When disease transmission [1] is modeled over networks [2–4], it is usual to model the infectivity (that is, the rate of transmission between infected and susceptible nodes) by assuming that transmission is equally likely over all links. For an idealized model this is the natural way to consider infectivity. However, when the underlying complex network is scale-free, the situation becomes unrealistic in the extreme tail of the distribution. While it has frequently been observed that real human social and disease transmission networks exhibit scale-free properties over several orders of magnitude, the tail of the distribution observed from data is always bounded. It is an open question whether these real networks are close to scale-free or only scale-free over a finite domain (note that any real network is of finite size so the degree is bounded) [5]. In [6] for example, the observation of a scale-free transmission mechanism for avian influenza is tempered by the fact that the finite available data necessarily limits inference to a bounded distribution. Moreover, when considering transmission of a disease in a finite time period, it is natural to suppose that there exists an upper bound on the infectivity of a highly connected individual. It is also quite reasonable to suppose that highly connected (and therefore highly visible) nodes in the network would be the focus of an immunization scheme (even for very limited control measures). Hence, in this paper we consider the case where the infectivity is a nondecreasing, but sublinear, function of the node degree.

The standard network susceptible-infected-susceptible (SIS) compartment model assumes that each infected node will contact every neighbor once within one time step [7], that is, the infectivity is equal to the connectivity, or the node degree. In [8], it is assumed that every individual has equal infectivity A, in which, at every time step, each infected individual will generate A contacts, where A is a constant. Joo and Lebowitz [9] examined cases where the transmission of infection between nodes depends on their connectivity, and a saturation function $C(k)$, which reduces the infection transmission rate across an edge going from a node with high connectivity k, was introduced.

Based on these results, in the present model, we take a more realistic approach. We assume the infectivity is piecewise linear: when the degree k of a node is relatively small, its infectivity is proportional to k, e.g., αk; when k is big, say, surpasses a constant A/α, then its infectivity is, say, A. We further discuss this model with respect to the effects of various immunization schemes.

Our motivation for this study is the observation that transmission of severe acute respiratory syndrome (SARS), most notably in Hong Kong during 2003, exhibits characteristics typical of a small-world or scale-free network [10–13]. During the SARS outbreak of 2003, several clusters of secondary infections were observed and traced back to a single primary infection. This can be explained either by assuming a highly infectious source or by assuming a highly connected source. The latter case leads naturally to a scale-free model of transmission, and the question of under which conditions a real disease transmitted on an apparently scale-free network will have a finite threshold. It has also recently been observed that the spatial-temporal distribution of avian influenza outbreaks naturally induces a scale-free network connectivity [6]. In this work, the available data exhibit a power law over three orders of magnitude, but, nonetheless, the tail of the distribution is bounded because the data are finite.

Of course, the SIS model used here was chosen because it is relatively simple, and also widely applicable. It may also be related to influenza vaccination problems [6] and strategies for dealing with computer viruses [14] among others. The remainder of this paper presents our model and results. In Sec. II we describe the general model, and Sec. III describes our analysis of this model. In Sec. IV we consider several models of immunization and Sec. V confirms our analysis with numerical simulations. In Sec. VI we conclude.

II. THE MODEL

Let $S_k(t)$ and $I_k(t)$ be the densities of susceptible and infected nodes with degree k at time t; then

$$S_k(t) + I_k(t) = 1,$$

and the mean-field equations for infected nodes with degree k can be written as

$$\frac{dI_k(t)}{dt} = \lambda k[1 - I_k(t)]\Theta(t) - I_k(t); \tag{1}$$

here we take a unit recovery rate, λ is the infection rate, and according to [4,15–18], $\Theta(t)$ can be written in general as

$$\Theta = \sum_{k'} \frac{\varphi(k')P(k'|k)I_{k'}}{k'}, \tag{2}$$

where $\varphi(k)$ denotes the infectivity of a node with degree k, and $P(k'|k)$ stands for the probability of a node with degree k pointing to a node with degree k'.

An epidemic threshold for (1) is the critical value λ_c of the infection rate λ, if λ is below λ_c, the disease will gradually die out, while if λ is above λ_c, the disease will spread on the network. What we are concerned with in this paper is to calculate the epidemic thresholds for the model (1) for various circumstances.

In [4,15–17], $\varphi(k)=k$; then the epidemic threshold $\lambda_c=0$ for sufficiently large networks. If $\varphi(k)=\alpha k$, the threshold λ_c also vanishes. In [18], $\varphi(k)=A$, where A is a constant, which means that every node has the same infectivity, no matter its degree, small or large. In this case, $\lambda_c=\frac{1}{A}>0$, a positive threshold.

We suppose that the connectivity of nodes is uncorrelated (for more realistic correlated cases, the discussion is similar to that below, but the expressions are much more complicated), then $P(k'|k)=k'P(k')/\langle k\rangle$, where $\langle k\rangle=\Sigma_k kP(k)$. Then (2) becomes

$$\Theta = \frac{1}{\langle k\rangle}\sum_{k'} \varphi(k')P(k')I_{k'}, \tag{3}$$

where for scale-free node distribution $P(k)=C^{-1}k^{-2-\gamma}, 0<\gamma\leq 1$, where $C=\zeta(2+\gamma)$ is Riemann's zeta function, which provides the appropriate normalization constant for sufficiently large networks [6,19].

We remark here that $\Theta(t)$, depending on k in general, represents the probability that any given link points to an infected node. For simplified uncorrelated cases, $\Theta(t)$ does not depend on k.

III. EPIDEMIC THRESHOLD FOR THE SIS MODEL WITH PIECEWISE LINEAR INFECTIVITY

Rather than the piecewise constant infectivity used in [9], we here take a piecewise linear infectivity,

$$\varphi(k) = \min(\alpha k, A), \tag{4}$$

where α and A are positive constants, $0<\alpha\leq 1$. We will see that piecewise linear infectivity is more realistic than linear ones such as $\varphi(k)=\alpha k$ or $\varphi(k)=A$. We will also discuss briefly other cases, such as piecewise smooth and nonlinear infectivities.

Some results obtained here are analytical derivations for results obtained numerically in [9].

A. Piecewise linear infectivity

In this section, we discuss in detail the piecewise linear infectivity case. By imposing steady state $\frac{dI_k(t)}{dt}=0$, from (1) we have

$$I_k = \frac{\lambda k\Theta}{1+\lambda k\Theta}. \tag{5}$$

Substitute I_k in (3) by (5), we obtain a self-consistency equation as follows:

$$\Theta = \frac{\lambda\Theta}{\langle k\rangle}\sum_{k'} \frac{k'\varphi(k')P(k')}{1+\lambda k'\Theta} \equiv f(\Theta). \tag{6}$$

Obviously, $\Theta\equiv 0$ is a solution of (6), i.e., $f(0)=0$. Note that

$$f(1) = \frac{\lambda}{\langle k\rangle}\sum_{k'} \frac{k'\varphi(k')P(k')}{1+\lambda k'} < \frac{1}{\langle k\rangle}\sum_{k'} \varphi(k')P(k')$$

$$\leq \frac{1}{\langle k\rangle}\sum_{k'} k'P(k') = 1,$$

$$f'(\Theta) = \frac{\lambda}{\langle k\rangle}\sum_{k'} \frac{k'\varphi(k')P(k')}{(1+\lambda k'\Theta)^2} > 0,$$

$$f''(\Theta) = -\frac{2\lambda^2}{\langle k\rangle}\sum_{k'} \frac{k'^2\varphi(k')P(k')}{(1+\lambda k'\Theta)^3} < 0;$$

therefore, a nontrivial solution exists only if

$$\left.\frac{df(\Theta)}{d\Theta}\right|_{\Theta=0} > 1. \tag{7}$$

The value of λ yielding the inequality (7) defines the critical epidemic threshold λ_c:

$$\lambda_c = \frac{\langle k\rangle}{\langle k\varphi(k)\rangle} = \frac{\sum_k kP(k)}{\sum_k k\varphi(k)P(k)}. \tag{8}$$

Approximating the sum in (8) on discrete k by continuous integration, and supposing the size of the network is sufficiently large, we can calculate λ_c as

$$\lambda_c = \frac{\int_m^{+\infty} k^{-1-\gamma}dk}{\int_m^{A/\alpha} \alpha k^{-\gamma}dk + \int_{A/\alpha}^{+\infty} Ak^{-1-\gamma}dk}$$

$$= \begin{cases} \dfrac{\frac{1-\gamma}{\alpha m}}{\left(\frac{A}{\alpha m}\right)^{1-\gamma}-\gamma}, & 0<\gamma<1, \\ \dfrac{\frac{1}{\alpha m}}{1+\ln\frac{A}{\alpha m}}, & \gamma=1, \end{cases} \tag{9}$$

where m is the minimum connectivity of the network, and $\alpha m<A$.

We remark that when $A \to +\infty$, from the above formula (9), $\lambda_c \to 0$; this is consistent with the fact that $\varphi(k)$ approaches the linear infectivity $\varphi(k)=\alpha k$; and when $\alpha m \geq A$, we can calculate that $\lambda_c = 1/A$; this is consistent with $\varphi(k) = A$ for all k.

From (9), we have a positive epidemic threshold λ_c if

$$\alpha m < \frac{1}{\gamma^{1/1-\gamma}} A (0 < \gamma < 1) \quad \text{or} \quad \alpha m < eA(\gamma = 1).$$

If $A \geq \gamma^{1/1-\gamma} m (0 < \gamma < 1)$ or $A \geq e^{-1} m (\gamma = 1)$, then λ_c is always positive.

B. Piecewise smooth and nonlinear infectivity

In some cases, infectivity may vary nonlinearly for small degrees and stay unchanged at a saturated value for large degrees, i.e., the infectivity may follow the following piecewise smooth function:

$$\varphi_1(k) = \min(\alpha k^\beta, A), \quad 0 \leq \beta \leq 1, \quad \alpha > 0.$$

In this case, the epidemic threshold

$$\lambda_c' = \begin{cases} \left[\dfrac{A\beta}{\beta - \gamma} \left(\dfrac{\alpha m^\beta}{A} \right)^{\gamma/\beta} - \dfrac{\alpha m^\beta}{\gamma(\beta - \gamma)} \right]^{-1}, & \beta \neq \gamma, \\ \left(\dfrac{m\alpha}{\beta} \ln \dfrac{A}{\alpha m^\beta} + \dfrac{m\alpha}{\beta} \right)^{-1}, & \beta = \gamma. \end{cases}$$

Thus we have positive λ_c' if $(\alpha m^\beta)^{\gamma/\beta - 1} > \frac{1}{\gamma(\beta - \gamma)} A^{\gamma/\beta}$ and $\beta > \gamma$ or $\alpha m^\beta < eA(\beta = \gamma)$.

Similar to Eq. (17) in [9], but with one more parameter, we can also use a smooth nonlinear infectivity, e.g.,

$$\varphi_2(k) = \frac{ak^\beta}{1 + bk^\beta}, \quad 0 \leq \beta \leq 1, \quad a > 0, \quad b \geq 0.$$

We can also discuss the epidemic threshold for this smooth nonlinear case for different parameters β, a, and b. The details will be discussed elsewhere [20]. We may also consider the effects of finite scale-free networks on the above discussions [5,20], and we may also consider epidemic thresholds for staged progression models [21].

IV. SIS MODEL WITH IMMUNIZATION

Vaccination is very helpful in controlling vaccine-preventable diseases. The SIS model is more appropriate than the susceptible-infected-recovered (or the susceptible-infected-removed) (SIR) model in the early stage of epidemic outbreaks when the effects of recovery and death can be ignored, and this is the optimal time period for immunization to be applied. In this section we discuss the SIS model on a scale-free network with piecewise linear infectivity and various immunization schemes [22,23].

A. Proportional immunization

Denote by δ the immunization rate, $0 < \delta < 1$; then Eq. (1) becomes

$$\frac{dI_k(t)}{dt} = \lambda k (1 - \delta)[1 - I_k(t)]\Theta(t) - I_k(t). \tag{10}$$

Let $\frac{dI_k(t)}{dt} = 0$; from (10) we have

$$I_k = \frac{\lambda(1 - \delta)k\Theta}{1 + \lambda(1 - \delta)k\Theta}. \tag{11}$$

Substitute I_k in (3) by (11), we obtain a self-consistency equation as follows:

$$\Theta = \frac{\lambda\Theta}{\langle k \rangle} \sum_{k'} \frac{(1 - \delta)k'}{1 + \lambda(1 - \delta)k'\Theta} \varphi(k') P(k') \equiv \tilde{f}(\Theta). \tag{12}$$

By arguments similar to those in Sec. III A, the epidemic threshold $\tilde{\lambda}_c$ is determined by the following inequality:

$$\left. \frac{d\tilde{f}(\Theta)}{d\Theta} \right|_{\Theta = 0} > 1;$$

therefore, it can be shown that

$$\tilde{\lambda}_c = \begin{cases} \dfrac{1}{1 - \delta} \dfrac{\frac{1-\gamma}{\alpha m}}{\left(\frac{A}{\alpha m}\right)^{1-\gamma} - \gamma}, & 0 < \gamma < 1, \\ \dfrac{1}{1 - \delta} \dfrac{\frac{1}{\alpha m}}{1 + \ln \frac{A}{\alpha m}}, & \gamma = 1, \end{cases}$$

that is,

$$\tilde{\lambda}_c = \frac{1}{1 - \delta} \lambda_c. \tag{13}$$

Note that in (13), when $\delta = 0$, i.e., if no immunization were done, then $\tilde{\lambda}_c = \lambda_c$; when $0 < \delta < 1$, $\tilde{\lambda}_c > \lambda_c$, that is, the immunization scheme is effective; while as $\delta \to 1$, $\tilde{\lambda}_c \to +\infty$, that is, in the case of a full immunization, it would be impossible for the epidemic to spread in the network.

B. Targeted immunization

We still use the piecewise linear infectivity $\phi(k)$ defined in (4). While proportional immunization schemes are effective, there may be more efficient schemes due to the heterogeneous nature of scale-free networks: they are robust to random attacks, but fragile to selective attacks. Accordingly, we can devise a targeted immunization scheme [23]. We introduce an upper threshold κ, such that all nodes with connectivity $k > \kappa$ are immunized, i.e., we define the immunization rate δ_k by

$$\delta_k = \begin{cases} 1, & k > \kappa, \\ c, & k = \kappa, \\ 0, & k < \kappa, \end{cases} \tag{14}$$

where $0 < c \leq 1$, and $\Sigma_k \delta_k P(k) = \bar{\delta}$, where $\bar{\delta}$ is the average immunization rate. The epidemic dynamics model is

$$\frac{dI_k(t)}{dt} = \lambda k (1 - \delta_k)[1 - I_k(t)]\Theta(t) - I_k(t);$$

this leads to

$$\Theta = \frac{1}{\langle k\rangle}\sum_{k'}\frac{\lambda(1-\delta_{k'})k'\Theta}{1+\lambda(1-\delta_{k'})k'\Theta}\varphi(k')P(k') \equiv \hat{f}(\Theta);$$

therefore, the epidemic threshold

$$\hat{\lambda}_c = \frac{\langle k\rangle}{\sum_{k'} k'\varphi(k')P(k')(1-\delta_{k'})} = \frac{\langle k\rangle}{\langle k\varphi(k)\rangle - \langle \delta_k k\varphi(k)\rangle}. \tag{15}$$

Note that $\langle \delta_k k\varphi(k)\rangle = \bar{\delta}\langle k\varphi(k)\rangle + \sigma'$, where $\sigma' = \langle (\delta_k - \bar{\delta}) \times [k\varphi(k) - \langle k\varphi(k)\rangle]\rangle$ is the covariance of δ_k and $k\varphi(k)$. There may be κ (usually big enough) where $\sigma' < 0$, but for appropriately small κ (between 5 and 25, say), $\delta_k - \bar{\delta}$ and $k\varphi(k) - \langle k\varphi(k)\rangle$ have the same signs except for some k's where $\delta_k - \bar{\delta}$ and/or $k\varphi(k) - \langle k\varphi(k)\rangle$ is zero; therefore $\sigma' > 0$ for appropriate κ. Then

$$\hat{\lambda}_c > \frac{1-\delta}{1-\bar{\delta}}\tilde{\lambda}_c.$$

If we set $\bar{\delta} = \delta$, then

$$\hat{\lambda}_c > \tilde{\lambda}_c \quad (0 < \delta < 1),$$

which means the targeted immunization scheme is more efficient than the proportional scheme discussed in Sec. IV A for the same average immunization rate.

C. Acquaintance immunization

As discussed in [18,22], a problem with the targeted immunization scheme is that it requires some global information about the degree of each node. In this section, we use another immunization strategy, acquaintance immunization [14], which calls for the immunization of random acquaintances of random nodes.

Choose a random fraction p of the N nodes; the probability that a particular node with k contacts is selected for immunization is $kP(k)/(N\langle k\rangle)$ [24,25]. Therefore, in (15) we may take $\delta_k = \frac{kP(k)}{N\langle k\rangle}pN = \frac{p}{\langle k\rangle}kP(k)$, so the epidemic threshold for this immunization scheme is

$$\check{\lambda}_c = \frac{(1-\bar{\delta})\langle k\varphi(k)\rangle - \sigma'}{\langle k\varphi(k)\rangle - \frac{p}{\zeta(2+\gamma)\langle k\rangle}\langle k^{-\gamma}\varphi(k)\rangle}\hat{\lambda}_c. \tag{16}$$

Note that

$$\begin{aligned}(1-\bar{\delta})\langle k\varphi(k)\rangle - \sigma' &= (1-\bar{\delta})\langle k\varphi(k)\rangle - \langle (\delta_k - \bar{\delta})[k\varphi(k) - \langle k\varphi(k)\rangle]\rangle \\ &> (1-\bar{\delta})\langle k\varphi(k)\rangle - \langle (1-\bar{\delta})[k\varphi(k) - \langle k\varphi(k)\rangle]\rangle \\ &> (1-\bar{\delta})\langle k\varphi(k)\rangle - \langle (1-\bar{\delta})k\varphi(k)\rangle = 0,\end{aligned}$$

$$\langle k\varphi(k)\rangle - \frac{p}{\zeta(2+\gamma)\langle k\rangle}\langle k^{-\gamma}\varphi(k)\rangle > \langle k\varphi(k)\rangle - \langle k^{-\gamma}\varphi(k)\rangle > 0.$$

So $\check{\lambda}_c = \Lambda\hat{\lambda}_c$, where Λ is a positive constant. This means the acquaintance immunization scheme is comparable in effectiveness to the targeted immunization scheme. Figure 2(a) below shows that $\hat{\lambda}_c > \check{\lambda}_c$.

We remark here that it can be further calculated that

$$\langle k\varphi(k)\rangle = \frac{\alpha m}{\gamma(1-\gamma)m^{\gamma}\zeta(2+\gamma)}\left[\left(\frac{A}{\alpha m}\right)^{1-\gamma} - \gamma\right],$$

$$\langle k^{-\gamma}\varphi(k)\rangle = \frac{\alpha}{2\gamma\zeta(2+\gamma)}\left[\frac{1}{m^{2\gamma}} - \frac{1}{2\gamma+1}\left(\frac{\alpha}{A}\right)^{2\gamma}\right].$$

D. Active immunization

In this section we propose a different immunization scheme: choose an infected node and immunize its neighbors whose degree $\geq\kappa$. That is, the epidemic dynamics model is

$$\frac{dI_k(t)}{dt} = \lambda k[1 - I_k(t)]\Theta(t) - (1+\bar{\delta}_k)I_k(t), \tag{17}$$

where

$$\bar{\delta}_k = \sum_{k'}\frac{k'P(k')}{\langle k\rangle}\delta_{k'},$$

and $\delta_{k'}$ is defined in (14).

After the early stage of a disease epidemic, there may be quite a lot of infected individuals; therefore this immunization scheme may be more appropriate. We show this rigorously below.

By letting $\frac{dI_k(t)}{dt} = 0$, model (17) leads to

$$\Theta = \frac{\lambda\Theta}{\langle k\rangle}\sum_{k'}\frac{k'\varphi(k')P(k')}{1+\bar{\delta}_{k'}+\lambda k'\Theta} \equiv \bar{f}(\Theta);$$

therefore, the epidemic threshold

$$\bar{\lambda}_c = \frac{\langle k\rangle}{\sum_{k'}(1+\bar{\delta}_{k'})^{-1}k'\varphi(k')P(k')}.$$

Note that

$$\bar{\delta}_k = \sum_{k'}\frac{k'P(k')}{\langle k\rangle}\delta_{k'} = \frac{\langle k\delta_k\rangle}{\langle k\rangle};$$

we have

$$\bar{\lambda}_c = \frac{\langle k\rangle + \langle k\delta_k\rangle}{\langle k\varphi(k)\rangle}. \tag{18}$$

Compare (18) with (8), we have

$$\bar{\lambda}_c = \lambda_c + \frac{\langle k\delta_k \rangle}{\langle k\varphi(k) \rangle} > \lambda_c. \tag{19}$$

That is to say, the immunization scheme we propose here is indeed effective, and the lower κ, the greater the term $\langle k\delta_k \rangle$ is and the more effective the scheme.

E. A brief summary

In previous sections we have discussed proportional, targeted, acquaintance, and active immunization schemes, and estimated the thresholds for each scheme. By comparing the thresholds for different immunization schemes, we have concluded that the targeted immunization scheme is more efficient than the proportional scheme; the acquaintance immunization scheme is comparable to the targeted immunization scheme; and the effectiveness of the active immunization scheme is also discussed.

In [22] a probability approach is used to calculate epidemic thresholds for random, targeted, and acquaintance immunization schemes, which are critical probability values and can be used to evaluate the fraction of immunized individuals. While in [23] proportional and targeted immunization schemes are discussed, epidemic thresholds are not considered directly; instead, as in [22], the critical fractions of immunized individuals are discussed.

Here, we give a direct characterization of epidemic thresholds for more immunization schemes, including the scheme of active immunization, so the thresholds are easier to apply practically.

V. NUMERICAL SIMULATIONS

In this section we present the results of numerical experiments investigating the (practical) effectiveness of some of the aforementioned immunization schemes. We simulate stochastic realizations of a SIS model on a scale-free network. We use the preferential attachment algorithm of Barabási and Albert (see [26]) to generate a network with theoretical scale-free exponent 3. (This corresponds to $\gamma=1$ in our notation.)

We consider a population of 1000 individuals of whom 1 is infected. We set the infectivity parameter $\alpha=0.02$ with $A=0.2$, so an individual with ten contacts has maximum infectivity. The recovery rate ($I \to S$ transition) is given by $\nu=0.01$. These parameters were chosen so that the quasiequilibrium state of around 990 infected individuals is typically reached after 50 time steps, taken to be days.

In Figs. 1(a) and 1(b) we show a typical realization of the SIS model after 1000 days and a histogram of the number of infected individuals in the population at day 1000 across 500 realizations. We see that very few realizations result in extinction of the disease over the time frame considered.

We repeated the simulation above when the immunization schemes—targeted, acquaintance and active—are implemented. For the targeted immunization scheme we choose $c=1$ and $\kappa=7$. The choice of κ is one more than $\langle k \rangle \approx 6$ of the generated network. A value of $\kappa=7$ is also used in the active immunization scheme. In the acquaintance immunization method, p, the proportion of the population selected for (possible) immunization each day, was chosen to be 1, so all of the population is a candidate for immunization each day. The results are summarized in Figs. 1(c)–1(h).

We see in Figs. 1(c) and 1(d) that (globally) targeted immunization reduces the pool of susceptible individuals in the population. This has the effect of the size of any epidemic being smaller, but more interestingly, for many realizations, the spread of the disease is stopped, becoming extinct within the period of simulation. Figure 1(c) shows an example of persistence of the disease but with a much lower pool of susceptible individuals. It is very similar to the time series demonstrating the active scheme operating [Fig. 1(g)].

The acquaintance immunization scheme has the property that, for any given p, eventually the entire population will be immunized. In practice, however, this asymptotic result has a long waiting time. We see in Figs. 1(e) and 1(f) that even with $p=1$ it can take a long time for the pool of susceptible individuals to decrease, and to perhaps obtain a reasonable rate of decrease the cost of immunization will be high. That is, to improve the performance, δ_k needs to be increased and the total number of immunizations will be high. Clearly, targeted immunization is more effective.

In Figs. 1(g) and 1(h) the result of the active immunization scheme is shown. We see that the results are comparable with those of the targeted scheme and indeed, for the same κ, if the spread of the disease is not caught then in terms of number of individuals immunized active is equivalent to targeted immunization. The advantage of the active method over targeted is that it will in general be less costly, as individuals are only locally immunized, whereas in targeted immunization all individuals with a certain number of contacts are immunized. Figure 1(g) shows a realization when the disease persists among a lower pool of individuals.

The above numerical experiment demonstrated the effectiveness of the various immunization schemes in a stochastic setting across different scale-free network realizations. It is instructive to see how the differential equations (10) behave subject to different immunization strategies and how they relate to the theoretical epidemic thresholds derived earlier. We consider this comparison in the plots of Fig. 2 for a particular realization of a Barabási and Albert scale-free network.

In Fig. 2(a), we compare the thresholds among no immunization and the other four immunization schemes; it is shown that all four immunization schemes are effective compared to the case without any immunization; and we can verify the conclusion in Sec. IV B that the targeted immunization scheme is more efficient than the proportional scheme discussed in Sec. IV A for the same average immunization rate. This is shown in Fig. 2(b), where $\delta=\bar{\delta}=0.218$; we can also see the effects of κ on the threshold λ for the targeted immunization scheme: the smaller κ is, the greater the threshold becomes, as is shown in Fig. 2(c).

Figure 2(d) further shows the theoretical thresholds of λ for the immunization schemes targeted, active, and acquaintance with respect to κ. Comparing with Figs. 2(a) and 2(b) and in particular Fig. 2(c) we can see that the theoretical thresholds calculated for a given κ are consistent with the

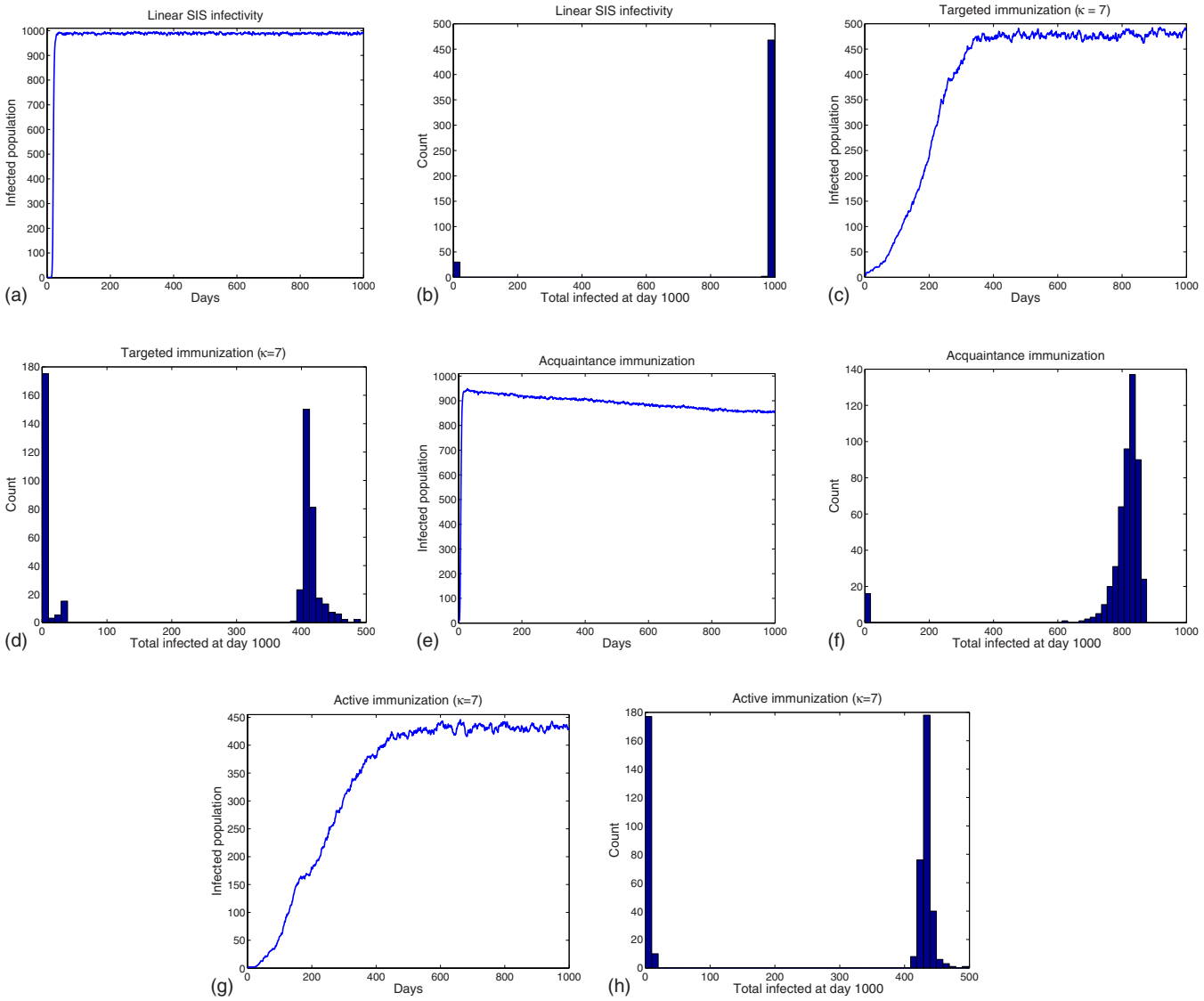

FIG. 1. (Color online) Typical realizations of the spread of a SIS disease model on a scale-free network: (a) no, (c) targeted, (e) acquaintance, and (g) active immunization. (b), (d), (f), and (h) are summary histograms of realizations after day 1000 for the piecewise linear infectivity SIS model with and without immunization.

simulated differential equations. That is, the theoretical epidemic thresholds are always lower than the infectivity λ resulting in an outbreak of the disease. This clearly demonstrates that our analytical expressions for epidemic thresholds are consistent and can usefully be calculated for practical immunization schemes.

VI. REMARKS AND DISCUSSION

The SARS outbreak of 2003 and its effect are still recent memories. Moreover, the threat of future outbreaks of other emerging diseases or of a human-transmissible version of the H5N1 avian influenza remain. The complex network model approach to SARS has been extensively studied in [10–13] and elsewhere. If we consider transmission of agents such as SARS or H5N1 on scale-free networks, the conclusions are dire [6]: the disease threshold is effectively zero. Nonetheless, we have shown that under rather limited, but realistic, constraints on the extent of the scale-free quality of the network, the threshold becomes positive.

The problem of how best to respond to disease transmission on a network currently remains unaddressed. In particular, as research on the SARS virus continues, it is likely that SARS will become a vaccine-preventable disease in the near future, so the discussions above about the effectiveness of various immunization schemes may be helpful for us to control the SARS disease transmission in the early stage of an outbreak. Moreover, apart from immunization, a variety of epidemic models including general contact rates, quarantine, and isolation, etc. may be used in controlling SARS [27]. These may be further studied by using the method presented in this paper.

In [28] a method is developed for determining minimal vaccine allocations to prevent an epidemic in a population with m heterogeneous subgroups; while in [27] various

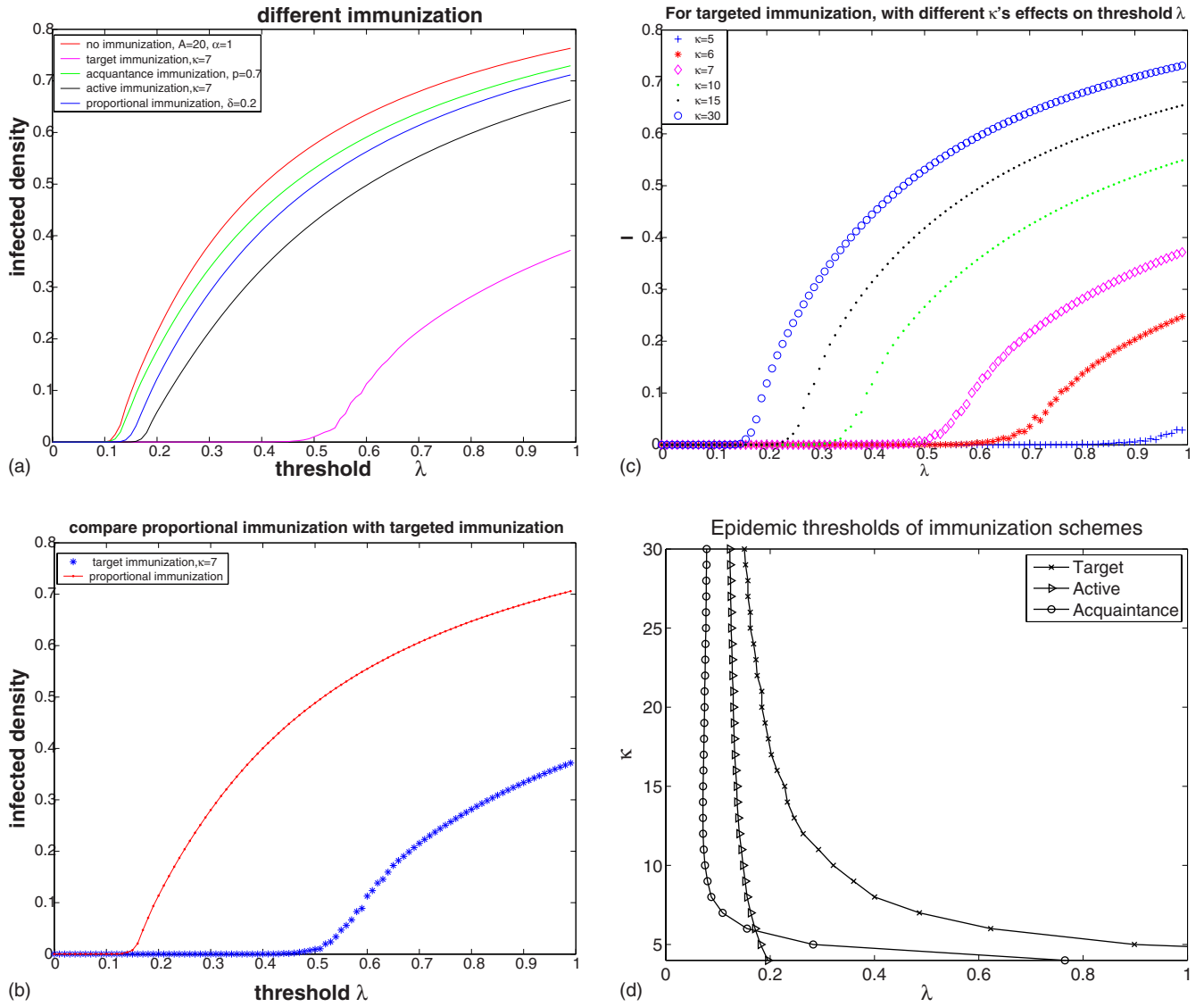

FIG. 2. (Color online) Comparison of the effectiveness of different immunization schemes. (a) The thresholds among no immunization and the other four immunization schemes, where $A=20$, $\alpha=1$, and we take $c=1$, $\kappa=7$ in (14); (b) shows that the targeted immunization scheme is much more efficient than the proportional scheme for the same average immunization rate, here $\delta=\bar{\delta}=0.218$. The theoretical thresholds are $\tilde{\lambda}_c=0.1471$ for proportional immunization and $\hat{\lambda}_c=0.4862$ for targeted ($\kappa=7$). (c) The effects of κ on the threshold λ for the targeted immunization scheme. (d) shows the theoretical thresholds of λ for the targeted, active, and acquaintance immunization schemes with respect to κ.

SARS models based on the Kermack-McKendrick model are discussed, and they share the basic properties that there is a threshold between disappearance of the disease and an epidemic outbreak, and that an epidemic will die out without infecting the entire population.

On a directed network we may need to distinguish degree distribution between in-degrees and out-degrees, as the infectivity $\varphi(k)$ and immunization scheme choice will depend on these quantities. More precisely, in a directed network, the infectivity will depend on out-degree distribution, while the choice of immunization scheme will depend on in-degree distribution.

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