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Immunization and Epidemic Dynamics in Complex Networks

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Abstract. We study the behavior of epidemic spreading in networks, and, in particular, scale free networks. We use the Susceptible–Infected–Removed (SIR) epidemiological model. We give simulation results for the dynamics of epidemic spreading. By mapping the model into a static bond-percolation model we derive analytical results for the total number of infected individuals. We study this model with various immunization strategies, including random, targeted and acquaintance immunization.

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1 Introduction

The study of epidemic spreading is based upon the notion that a disease is conveyed by contact between an infected individual and an uninfected individual who is susceptible to the disease. An endemic stage is reached if a finite fraction of the population is infected. Similarly, this notion may describe the spreading of a computer virus through a network of computers. Recently, it has been shown that in a class of scale free networks an epidemic may spread regardless of how low is its rate of infection [1–3].

An extensive research was dedicated to the subject of attacks on networks [4–7], by targeting individuals either randomly or by intentionally. In particular, the Internet and the WWW were shown to be robust to random breakdowns and fragile to intentional attacks, due to their scale-free distribution of nodes. However, these studies focused on the results of damaging computers by an outside source, and did not take into account the possibility of a propagation of a problem throughout the connections amongst the computers themselves, in a way similar to a spreading of a disease. The application of network stability to immunization has been studied in [8], and the effect of partial information has been studied in [9].

Several models have been proposed for epidemic dynamics, differing in the disease stages, the dynamical parameters, and the underlying structure of contacts. The most common models for the disease dynamics are the SIR, SIS and SIRS models, which represent the development of each individual's disease in a network. In this paper we will focus on the SIR model, where an infected individual is infective for some period of time, and then is recovered and can no longer be infected by the disease. For a general introduction to epidemiology, see [10].

Another issue regarding the model used is the underlying network topology. The network of (possible) contacts between individuals determines which individuals can infect each other. In general, this network may also be dynamic and change during the spread of the disease. We will assume that the network is static during the epidemic outbreak. We will also assume that the network is sparse, *i.e.*, that the number of links is proportional to the number of individuals. We will focus on scale free network topologies.

Moreover, we will also allow a fraction of the individuals to be immunized, *i.e.*, these individuals can not be infected. When a vaccination for a disease exists, immunizing certain individuals against being infected by a disease as a preemptive method may be the most efficient way to prevent loss of time and funds (and, of course, suffering, when dealing with infected people) due to the disease. Obviously, immunization of the entire population will eradicate the disease entirely, but this is not always possible, or may involve high costs and effort. Therefore, the choice of which individuals to immune is an important step in the immunization process, and may increase the efficiency of the immunization strategy.

2 Epidemic dynamics

A contagious disease may turn into an epidemic, if the number of infected (sick) individuals is of the order of the number of individuals in the whole system. When there are no more sick individuals in the system (only susceptible left, and the others are removed), we may say that the disease is cured.

In many cases, an epidemic is indeed being cured. The main consideration therefore should be how much time is required for the system to reach such a stage, and how many individuals are being infected throughout the process. If both the time of the contagious period and the number of people exposed to it can be reduced, then the amount of suffering and loss of resources can be reduced as well.

2.1 The SIR model

The SIR model represents the development of a disease in a network of connected individuals. S stands for the susceptible stage, where the individual is healthy. I stands for the infected stage, where the individual is infected with the disease and can infect other individuals in contact with it. R is the removed stage, where the individual is either recovered and has acquired immunization to the disease or otherwise permanently removed from the system.

In our numerical simulations all the individuals (vertices) are at first susceptible, *i.e.*, they are all healthy and none of them is immuned to the disease. One vertex, chosen randomly, is being infected. At each time step, every susceptible neighbor of an infected vertex has a probability of becoming infected itself, and each infected vertex has a probability to be removed from the system. We assume here that both probabilities (infection and removal) are the same for each vertex and its neighbors (networks having different probabilities for each vertex are studied in [3]).

2.2 The SIR model as bond percolation

One of the nicest features of the SIR model is that despite it being a dynamic model it can be mapped into a completely static one [3,11,12]. Consider a network where each node transmits the epidemic to each of its neighbors with rate r, and is removed with average recovery time τ. The infection can be, therefore, considered as a Poisson process, with average $r\tau$. Thus, the probability for each neighbor *not* to be infected is $e^{-r\tau}$.

The outcome of this process is therefore the same as bond percolation, in which each directed link is occupied with probability $p_b = 1 - e^{-r\tau}$. If r and τ have the same value for each node, the network can be taken to be non-directed. While information on the epidemic dynamics is lost by this description, the critical threshold for the dynamic model can be deduced from the bond percolation problem. Furthermore, the total fraction of infected individuals in the endemic state is the same as the size of the giant component of the percolation model, and the probability of a single disease event to decay before reaching the endemic state equals the fraction of finite components in the percolation networks.

To find the threshold for bond percolation in networks one should consider the average number of links outgoing from a site which is, in itself, reached by following a link. This is similar to the course of the epidemic. If an infected individual infects, on average, at least one other individual, then the epidemic can reach an endemic state. Since a site can be reached by one of its k links, its probability of being reached is $kP(k)/(N\langle k\rangle)$, where N is the number of nodes, $P(k)$ is the fraction of nodes having degree (number of links) k, and $\langle k\rangle = \sum_k kP(k)$ denotes the average degree of nodes in the network. The probability of each of its $k-1$ outgoing links of infecting its neighbor is p_b. Since the network is randomly connected, as long as the epidemic is not spread yet, the average number of influenced neighbors is:

$$\langle n_i\rangle = p_b \sum_k \frac{P(k)k(k-1)}{\langle k\rangle} \ . \tag{1}$$

Therefore, an endemic state can be reached only if $\langle n_i\rangle > 1$, leading to [5,6]

$$\left(\frac{\langle k^2\rangle}{\langle k\rangle} - 1\right) > p_b^{-1} \ . \tag{2}$$

From this expression it can easily be seen that scale free networks, with degree distribution $P(k) \sim k^{-\gamma}$, with $\gamma \leq 3$, having a divergent second moment, undergo the transition only at $p_b \to 0$ [5,6]. That is, an epidemic can spread in this network regardless of how small the infection probability and how quick is the recovery process [1,2].

3 Immunization

In general immunization can be seen as a site percolation problem. Each immunized individual can be regarded as a site which is removed from the network. The goal of the immunization process is to pass (or at least approach) the percolation threshold, leading to minimization of the number of infected individuals. The complete model of SIR and immunization can be considered as a site–bond percolation model, and the immunization is considered successful if the network is below the percolation threshold.

It is well established that immunization of randomly selected individuals requires immunizing a very large fraction f of the population, in order to arrest epidemics that spread upon contact between infected individuals [1,4, 5,10,13–15]. Many diseases require 80%-100% immunization. For example, Measles requires 95% of the population to be immunized [10]. The same is correct for the Internet, where stopping computer viruses requires almost 100% immunization [1,4,5,16]. On the other hand, targeted immunization of the most highly connected individuals [4,6–8,10,17], while effective, requires global information about the network in question, rendering it impractical in many cases. Here, we develop a mathematical model and propose an effective strategy, based on the immunization of a small fraction of *random acquaintances* of randomly selected nodes. In this way, the most highly connected nodes are immunized, and the process prevents epidemics with a small finite immunization threshold and without requiring specific knowledge of the network.

3.1 Random immunization

Social networks are known to possess a broad distribution of the number of links (contacts), k, emanating from a node (an individual) [18–22]. Examples are the web of sexual contacts [23], movie-actor networks, science citations and cooperation networks [24,25] etc. Computer networks, both physical (such as the Internet [26]) and logical (such as the WWW [27], e-mail [28] and trust networks [29]) are also known to possess wide, scale-free, distributions. Studies of percolation on broad-scale networks show that a large fraction f_c of the nodes need to be removed (immunized) before the integrity of the network is compromised. This is particularly true for scale-free networks, $P(k) = ck^{-\gamma}$ $(k \geq m)$, where $2 < \gamma < 3$ — the case of most known networks [18–20] — where the percolation threshold $f_c \to 1$, and the network remains connected (contagious) even after removal of most of its nodes [5]. In other words, with a random immunization strategy almost all of the nodes need to be immunized before an epidemic is arrested (see Fig. 1).

To calculate the immunization threshold, one should consider the site–bond percolation model. The considerations are the same as for the epidemic threshold [Eq. (2)], with the exception that a site may also be immunized, in which case it can not propagate the disease. This adds another factor of $p_s = 1 - f$, the probability that a site is not immunized, to the calculation. Leading to

$$\left(\frac{\langle k^2\rangle}{\langle k\rangle} - 1\right) > (p_s p_b)^{-1} \ . \tag{3}$$

As can be seen, in this case as well, the epidemic will only be arrested if $p_b p_s \to 0$, meaning that for every epidemic almost the entire population must be immunized in order to prevent the epidemic spreading.

3.2 Targeted immunization

When the most highly connected nodes are targeted first, removal of just a small fraction of the nodes results in the network's disintegration [4,6,7]. This has led to the suggestion of targeted immunization of the HUBs (the most highly connected nodes in the network) [8,9].

The simplest targeted immunization strategy calls for the immunization of the most highly connected individuals. To use this approach, the number of connections of each individual should be known (at least approximately – see [9]). In this case, the probability that a site is not immunized, when the immunization rate is f, is $\theta_f(k)$, where,

$$\theta_f(k) = \begin{cases} 1, & k < k^*, \\ c, & k = k^*, \\ 0, & k > k^*, \end{cases} \tag{4}$$

and k^* and $0 < c \leq 1$ are determined by the condition

$$\sum_k P(k)\theta_f(k) = 1 - f \ . \tag{5}$$

To find the critical immunization threshold using this strategy, one can again find the fraction giving, on average, one outgoing infective link per infected individual. this amounts to the demand:

$$\sum_k \frac{k(k-1)P(k)\theta_{f_c}(k)}{\langle k\rangle} = p_b^{-1} \ . \tag{6}$$

Solving Eq. (6) in conjunction with Eq. (5) allows the calculation of the exact immunization threshold. The implications of partial knowledge of the node degrees, leading to functions other than $\theta_f(k)$, were studied in [9].

3.3 Acquaintance immunization

3.3.1 Description

One problem with the targeted immunization approach is that it requires a complete, or at least fairly good knowledge of the degree of each node in the network. Such global information often proves hard to gather, and may not even be well-defined (as in social networks, where the number of social relations depends on subjective judging). The acquaintance immunization strategy proposed herein works at low immunization rates, f, and obviates the need for global information.

In our approach [30], we choose a random fraction p of the N nodes and look for a random acquaintance with whom they are in contact (thus, the strategy is purely local, requiring minimal information about randomly selected nodes and their immediate environments). The acquaintances, rather than the originally chosen nodes, are the ones immunized. The fraction p may be larger than 1, for a node might be queried more than once, on average, while the fraction of nodes immunized f is always less than or equal to 1.

3.3.2 Analysis

Suppose we apply the acquaintance strategy on a random fraction p of the network. The critical fractions, p_c and f_c, needed to stop the epidemic can be analytically calculated. In each event, the probability that a particular node with k contacts is selected for immunization is $kP(k)/(N\langle k\rangle)$ [5, 6]. This quantifies the known fact that randomly selected acquaintances have, on average, a higher degree than randomly selected nodes [31,32].

Suppose we follow some possible branch in the course of the epidemic, starting from a random link of the spanning cluster. That is, we study the possible spread of the epidemic by considering nodes that are not immunized, and therefore are susceptible to the epidemic that may become infected. In some layer (hop-distance from the starting point), l, we have $n_l(k')$ nodes of degree k'. In the next layer $(l+1)$ each of those nodes has $k'-1$ new neighbors (excluding the one through which we arrived). Let us denote the event that a node of degree k is susceptible to the disease (not immunized, and therefore may

be infected through the course of epidemic spreading) by s_k. To find out the number of nodes, $n_{l+1}(k)$, of degree k that are susceptible and are reached in the course of the epidemic, we multiply the number of links going out of the lth layer by the probability of reaching a node of degree k through following a link from a susceptible node, $p(k|k', s_{k'})$. Then, we multiply by the probability that this node is also susceptible given both the node and the neighbor's degrees, and the fact that the neighbor is also susceptible, $p(s_k|k, k', s_{k'})$. Since below and at the critical percolation threshold loops are irrelevant [5], one can ignore them for the calculation of the threshold. Therefore,

$$n_{l+1}(k) = p_b \sum_{k'} n_l(k')(k'-1)p(k|k', s_{k'})p(s_k|k, k', s_{k'}) \ . \tag{7}$$

By using Bayes' rule:

$$p(k|k', s_{k'}) = \frac{p(s_{k'}|k, k')p(k|k')}{p(s_{k'}|k')} \ . \tag{8}$$

Assuming that the network is uncorrelated (no degree-degree correlations), the probability $\phi(k)$ of reaching a node with degree k via a link is independent of k':

$$\phi(k) \equiv p(k|k') = kP(k)/\langle k\rangle \ . \tag{9}$$

(A study of cases where correlations exists can be found in [3,33,34]).

A random site (of degree k') is selected in each step with probability $1/N$. The probability of being redirected to a specific acquaintance is $1/k'$. Thus, the probability that the acquaintance is *not* selected in one particular attempt, is $1-1/(Nk')$, and in all Np vaccination attempts, it is

$$\nu_p(k') \equiv \left(1 - \frac{1}{Nk'}\right)^{Np} \approx e^{-p/k'} \ . \tag{10}$$

If the neighbor's degree is not known, the probability is $\nu_p \equiv \langle \nu_p(k)\rangle$, where the average (and all averages henceforth) is taken with respect to the probability distribution $\phi(k)$. In general, the probability that a node with degree k is susceptible is $p(s_k|k) = \langle e^{-p/k}\rangle^k$, if no other information exists on its neighbors. If the degree of one neighbor (which is the one through which the epidemic propagated) is known to be k': $p(s_k|k, k') = e^{-p/k'} \times \langle e^{-p/k}\rangle^{k-1}$. Since the fact that a neighbor with a known degree is immunized does not provide any further information about a node's probability of immunization, it follows that $p(s_k|k, k') = p(s_k|k, k', s_{k'})$. Using the above equations one obtains:

$$p(k'|k, s_k) = \frac{\phi(k')e^{-p/k'}}{\langle e^{-p/k}\rangle} \ . \tag{11}$$

Substituting these results in (7) yields:

$$n_{l+1}(k) = p_b \nu_p^{k-2} \phi(k) e^{-p/k} \sum_{k'} n_l(k')(k'-1)e^{-p/k'} \ . \tag{12}$$

Since the sum in (12) does not depend on k', it leads to the stable distribution of degree in a layer l: $n_l(k) = a_l \nu_p^{k-2} \phi(k) e^{-p/k}$, for some a_l. Substituting this into (12) yields:

$$n_{l+1}(k) = n_l(k) p_b \sum_{k'} \phi(k')(k'-1)\nu_p^{k'-2} e^{-2p/k'} \ . \tag{13}$$

Therefore, if the sum is larger than 1 the branching process will continue forever (the percolating phase), while if it is smaller than 1 immunization is sub-critical and the epidemic is arrested. Thus, we obtain a relation for p_c:

$$\sum_k \phi(k)(k-1)\nu_{p_c}^{k-2} e^{-2p_c/k} = p_b^{-1} \ , \tag{14}$$

where the case $p_b \to 1$ corresponds to full immunization, *i.e.* stopping the epidemic regardless of its infection rate.

The fraction of immunized nodes is easily obtained from the fraction of nodes which are not susceptible,

$$f_c = 1 - \sum_k P(k)p(s_k|k) = 1 - \sum_k P(k)\nu_{p_c}^k \ , \tag{15}$$

where $P(k)$ is the regular distribution, and p_c is found numerically using Eq. (14). The term $\nu_{p_c}^{k-2}$ in Eq. (14) induces an exponential cutoff on the degree distribution of susceptible nodes for $0 < \nu_{p_c} < 1$. Therefore, the sum in Eq. (14) becomes finite for some finite $\nu_{p_c} > 0$. Substituting this into Eq. (15) indicates that $f_c \neq 1$, and is finite even in the thermodynamic limit.

A related immunization strategy calls for the immunization of acquaintances referred to by at least n nodes. (Above, we specialized to $n = 1$.) The threshold is lower the larger n is, and may justify, under certain circumstances, this somewhat more involved protocol.

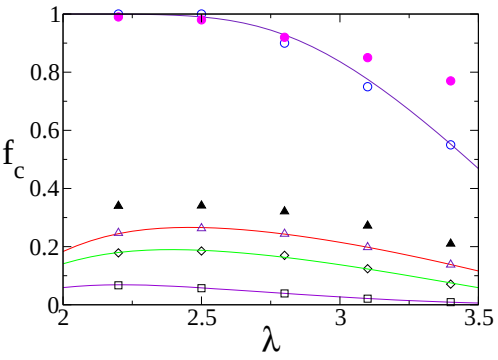

Fig. 1. Critical immunization threshold, f_c, as a function of γ in scale-free networks (with $m = 1$), for the random immunization ($\circ$), acquaintance immunization ($\triangle$), double acquaintance immunization ($\diamond$), and targeted immunization ($\square$) strategies. Curves represent analytical results, while data points represent simulation data, for a population $N = 10^6$ [Due to the population's final size $f_c < 1$ for random immunization even when $\gamma < 3$]. Full symbols are for random and acquaintance immunization of assortatively mixed networks (where links between sites of degree k_1 and $k_2 (> k_1)$ are rejected with probability $0.7\left(1 - \frac{k_1}{k_2}\right)$).

To analyze the threshold for the double acquaintance ($n = 2$) case, we should replace the probabilities for susceptibility with the appropriate probabilities considering the fact that a node is immunized only if 2 of its contacts point at it. Since the process is a Poisson process (in the limit of large N), the probabilities are:

$$p(s_k|k,k') = e^{-p/k'}\langle e^{-p/k}\rangle^{k-2} \times \left[\langle\frac{pe^{-p/k}}{k}\rangle(k-1) + \langle e^{-p/k}\rangle\left(1+\frac{p}{k'}\right)\right] , \quad (16)$$

and

$$p(s_k|k) = \langle e^{-p/k}\rangle^{k-1}\left[\langle\frac{pe^{-p/k}}{k}\rangle k + \langle e^{-p/k}\rangle\right] . \quad (17)$$

We will use the notation $\nu_p \equiv \langle e^{-p/k}\rangle$ and $\mu_p \equiv p\langle e^{-p/k}/k\rangle$. Using Bayes' rule as before and substituting into Eq. (7), one obtains

$$n_{l+1}(k) = p_b \sum_{k'} n_l(k')\phi(k)e^{-p/k}e^{-p/k'}{\nu_p}^{k-3} \times \frac{(k'-1)\left[(k'-1)\mu_p+\left(1+\frac{p}{k}\right)\nu_p\right]\left[(k-1)\mu_p+\left(1+\frac{p}{k'}\right)\nu_p\right]}{\nu_p+k'\mu_p} . \quad (18)$$

It can now be seen that the kernel of Eq. (18) is separable into three functions

$$n_l(k) = \phi(k){\nu_p}^{k-3}e^{-p/k}\left(a_l + b_l k + \frac{c_l}{k}\right) . \quad (19)$$

Substituting this back into Eq. (18) leads to the matrix notation

$$\begin{pmatrix} a_{l+1} \\ b_{l+1} \\ c_{l+1} \end{pmatrix} = p_b \sum_{k'} \frac{\phi(k')(k'-1)e^{-2p/k'}{\nu_p}^{k'-3}}{\nu_p + k'\mu_p}\mathbf{M}\begin{pmatrix} a_l \\ b_l \\ c_l \end{pmatrix} , \quad (20)$$

where $\mathbf{M}$ is the matrix:

$$\mathbf{M} = \begin{pmatrix} A_p(k') & k'A_p(k') & \frac{A_p(k')}{k'} \\ \mu_p B_p(k') & k'\mu_p B_p(k') & \frac{\mu_p B_p(k')}{k'} \\ p\nu_p C_p(k') & k'p\nu_p C_p(k') & \frac{p\nu_p C_p(k')}{k'} \end{pmatrix} , \quad (21)$$

and we have used the definitions $B_p(k') \equiv \nu_p + k'\mu_p - \mu_p$, $C_p(k') \equiv \nu_p - \mu_p + \frac{\nu_p p}{k'}$ and $A_p(k') \equiv C_p(k')B_p(k') + p\mu_p\nu_p$.

Since this is a branching process, it is controlled by the largest eigenvalue of the matrix $\mathbf{N}$,

$$\mathbf{N} = \sum_{k'} \frac{\phi(k')(k'-1)e^{-2p/k'}{\nu_p}^{k'-3}}{\nu_p + k'\mu_p}\mathbf{M} . \quad (22)$$

This eigenvalue can be calculated numerically using standard methods and the immunization threshold is obtained when $\lambda_1 \equiv \max_{\mathbf{v}} ||\mathbf{Mv}||/||\mathbf{v}||$, the largest eigenvalue of $\mathbf{N}$, satisfies $\lambda_1 = 1/p_b$. This can be solved numerically for a given degree distribution $P(k)$. The critical value p_c is then obtained and can be used to evaluate f_c, the fraction of immunized individuals,

$$f_c = 1 - \sum_k P(k){\nu_{p_c}}^{k-1}(\nu_{p_c} + pk\mu_{p_c}) . \quad (23)$$

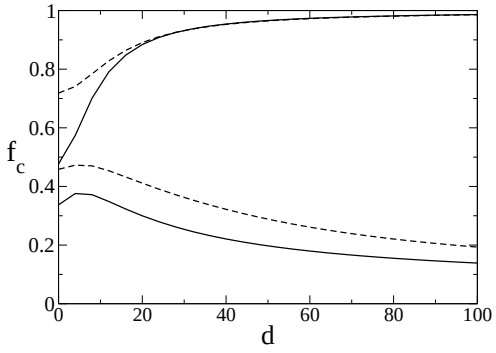

Fig. 2. Critical concentration, f_c, for the bimodal distribution (of two Gaussians) as a function of d, the distance between the modes. The first Gaussian is centered at $k = 3$ and the second one at $k = d+3$ with height 5% of the first. Both have variance 2 (solid lines) or 8 (dashed lines). Top 2 lines are for random immunization. The bottom 2 lines are for acquaintance immunization. All curves are analytically derived from Eqs. (15) and (14). Very similar results have been obtained for bimodal distributions of two Poissonians. Note that also for the case $d = 0$, i.e. a single Gaussian, the value of f_c reduces considerably due to the acquaintance immunization strategy. Thus the strategy gives improved performance even for relatively narrow distributions [36].

3.3.3 Discussion

The acquaintance immunization strategy is effective for any broad-scale distributed network. Here we give examples for scale-free and bimodal distributions, which are common in many natural networks. We also give an example of an assortatively mixed network (where high degree nodes tend to connect to other high degree nodes [35]). We also discuss the effectiveness of the strategy in conjunction with the SIR epidemiological model.

In Fig. 1, we show the immunization threshold f_c needed to stop an epidemic in networks with $2 < \gamma < 3.5$ (this covers all known cases). Plotted are curves for the (inefficient) random strategy, and the strategy advanced here, for the cases of $n = 1$ and 2. Note that while $f_c = 1$ for networks with $2 < \gamma < 3$ (e.g. the Internet) it decreases dramatically to values $f_c \approx 0.25$ with the suggested strategy. The figure also shows the strategy's effectiveness in case of assortatively mixed networks [35], *i.e.*, in cases where $p(k'|k)$ does depend on k, and high degree nodes tend to connect to other high degree nodes, which is the case for many real networks.

Fig. 2 gives similar results for a bimodal distribution (consisting of two Gaussians, where high degree nodes are rare compared to low degree ones). This distribution is also believed to exist for some social networks, in particular, for some networks of sexual contacts. The improvement gained by the use of the acquaintance immunization strategy is evident in Fig. 2.

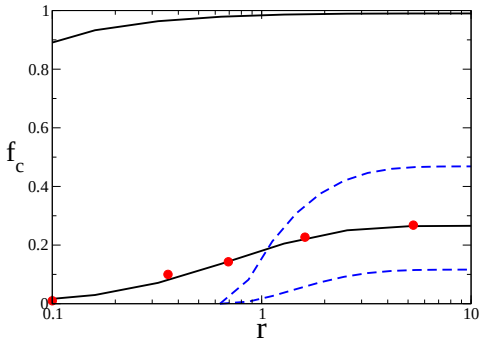

Fig. 3. Critical concentration, f_c, vs r, the infection rate, for the SIR model with $\tau = 1$. The solid lines are for random (top) and acquaintance immunization (bottom) for scale-free networks with $\gamma = 2.5$. The dashed lines are for $\gamma = 3.5$ (top – random, bottom – acquaintance immunization).The circles represent simulation results for acquaintance immunization for scale-free networks with $\gamma = 2.5$.

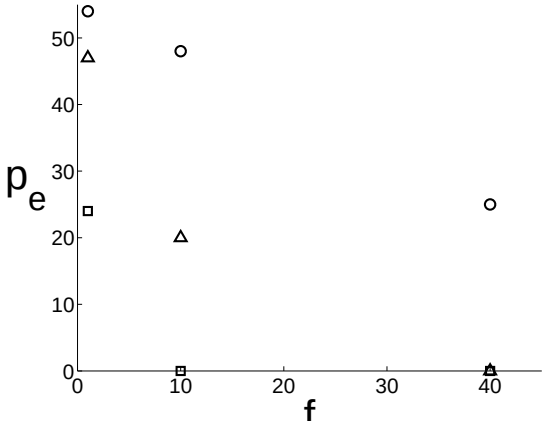

Fig. 4. Fraction of endemic outbreaks, p_e, as a function of the fraction of immunized individuals f, for random immunization (○), acquaintance immunization (△), and targeted immunization (□) strategies.

3.3.4 Practical issues

Various immunization strategies have been proposed, mainly for the case of an already spread disease, and are based on tracing the chain of infection towards the super-spreaders of the disease [13]. This approach is different from our proposed approach, since it is mainly aimed at stopping an epidemic after the outbreak began. It is also applicable for cases where no immunization exists and only treatment for already infected individuals is possible. Our approach, on the other hand, can be used even before the epidemic starts spreading, since it does not require any knowledge of the chain of infection.

In practice, any population immunization strategy must take into account issues of attempted manipulation. We would expect the suggested strategy to be less sensitive to manipulations than targeted immunization strategies. This is due to its dependence on acquaintance reports, rather than on *self*-estimates of number of contacts. Since a node's reported contacts pose a direct threat to the node (and relations), we anticipate that manipulations would be less frequent. Furthermore, we would suggest adding some randomness to the process: for example, reported acquaintances are not immunized, with some small probability (smaller than the random epidemic threshold), while randomly selected individuals are immunized directly, with some low probability. This will have a small impact on the efficiency, while enhancing privacy and rendering manipulations less practical.

3.4 Numerical results

Fig. 5 presents the dynamic of the spreading of the disease in the SIR model, as well as while implementing the three methods of immunization which were discussed above (random, acquaintance and targeted).

In the event of an epidemic outbreak the fraction of Infected individuals grows until reaching a certain maximum, after which it decays to zero, if given sufficient time. When there is no epidemic outbreak, however, the number on Infected (and therefore Removed) individuals is relatively so low, and the number of Susceptible individuals is relatively so high, that their plotting versus the time reveals no change after a very short time, and the respective graphs appear as horizontal lines. This may happen even with no immunization, for some network realizations or due to the low degree or cluster size of the first Infected individual. The fraction of such occurrences grows higher with immunization, and higher still as the immunized fraction of the population grows, in each of the three immunization methods we checked. In fact, a very similar endemic fraction can be detected in randomly immunizing 10% of the population and in acquaintance immunization of 1% of it.

Fig. 4 shows the fraction of endemic outbreaks, out of all the network realizations a randomly infecting an individual. As mentioned above, random immunization requires a high fraction of immunized nodes in order to be effective, while targeted immunization terminates the disease completely in a fraction immunization of 10%. This fraction is not enough for acquaintance immunization. Despite lowering considerably the endemic fraction, this strategy requires a higher immunization fraction in order to eradicate the disease.

In Fig. 5 it can easily be seen that for a low immunization fraction, the dynamics of random immunization are very similar to the regular SIR model. Targeted immunization is already highly effective - not only most of its realizations are devoid of endemic state, but when the epidemic does spread, a significantly smaller fraction of individuals is Infected. Acquaintance immunization shows a higher fraction of endemic states than the targeted, but its fraction of Infected individuals is still lower than that obtained with the random immunization strategy.

4 Conclusions

In conclusion, we have proposed a novel efficient strategy for immunization, requiring no knowledge of the nodes' degrees or any other global information. This strategy is efficient for networks of any broad-degree distribution and allows for a low threshold of immunization, even where random immunization requires the entire population to be immunized. We have presented analytical results for the critical immunization fraction in both a static model and the kinetic SIR model.

As a final remark, we note that our approach may be relevant to other networks, such as ecological networks of predator-prey [37,38], metabolic networks [39], networks of cellular proteins [40], and terrorist networks. For terrorist networks, our findings suggest that an efficient way to disintegrate the network, is to focus more on removing individuals whose name is obtained from another member of the network.

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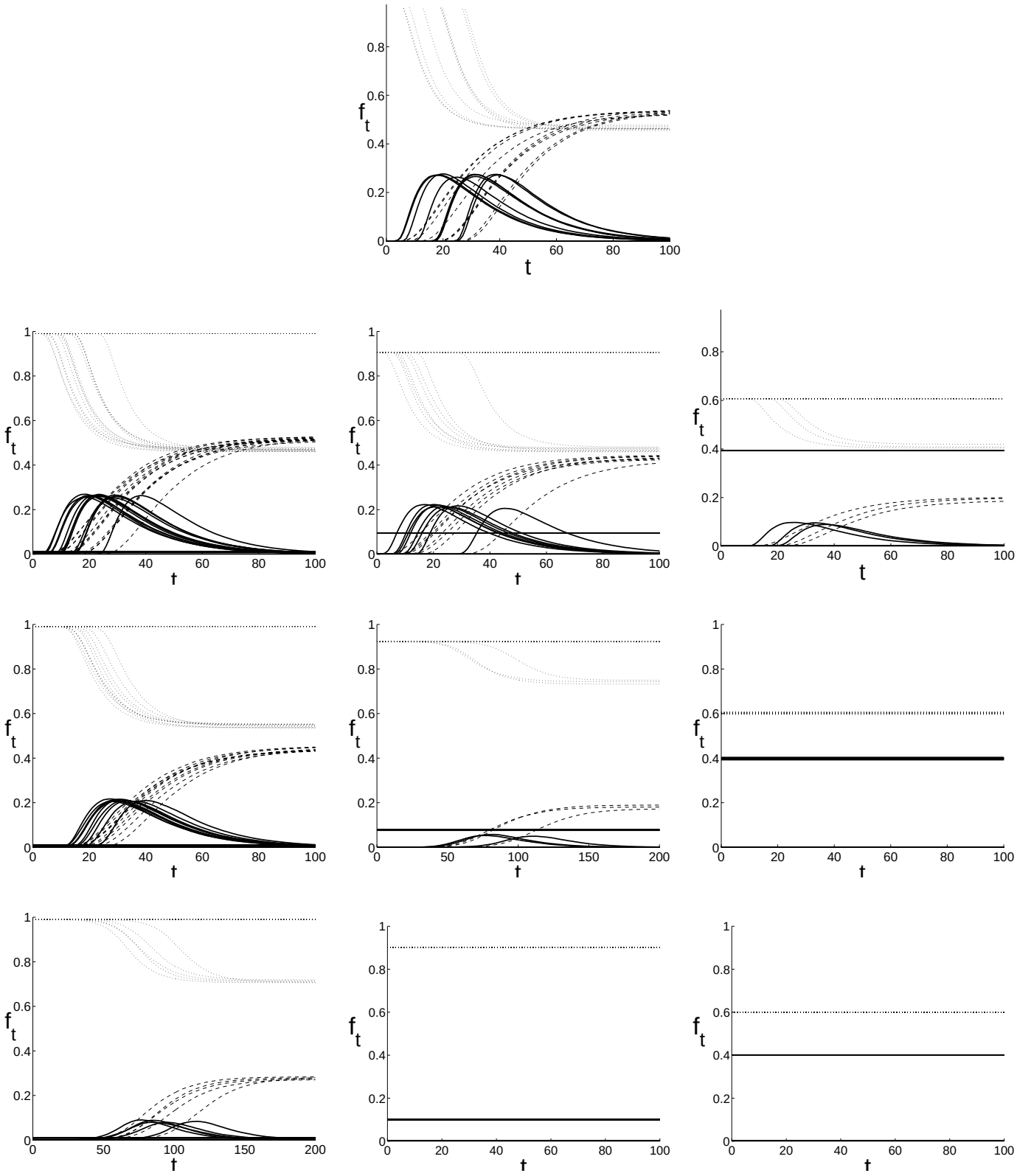

Fig. 5. Dynamics of SIR model on a scale free network: fraction of the population occupying each state, as a function of time. Fraction of Susceptible (dotted light grey line), Infected (solid black line) and Removed (dashed darker grey) individuals. Regular SIR model (top), Random immunization (second row), acquaintance immunization (third row) and targeted immunization (bottom), where p is 1% (left), 10% (middle column) and 40% (right). Immunized fraction is denoted by a black straight line. In all cases, the size of the network is 10^5, with $\lambda = 2.5$ and $m = 1$. The parameters of the SIR model are $r = 0.1$ and $\tau = 10$. Each graph contains 20 different realizations of the scale-free network.

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