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Y. Shang[a)]

AFFILIATIONS

Department of Computer and Information Sciences, Northumbria University, Newcastle upon Tyne NE1 8ST, United Kingdom

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[a)]**Author to whom correspondence should be addressed:** yilun.shang@northumbria.ac.uk

ABSTRACT

Modern view of network resilience and epidemic spreading has been shaped by percolation tools from statistical physics, where nodes and edges are removed or immunized randomly from a large-scale network. In this paper, we produce a theoretical framework for studying targeted immunization in networks, where only n nodes can be observed at a time with the most connected one among them being immunized and the immunity it has acquired may be lost subject to a decay probability ρ. We examine analytically the percolation properties as well as scaling laws, which uncover distinctive characters for Erdős–Rényi and power-law networks in the two dimensions of n and ρ. We study both the case of a fixed immunity loss rate as well as an asymptotic total loss scenario, paving the way to further understand temporary immunity in complex percolation processes with limited knowledge.

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Percolation theory has been extensively employed to study network resilience and spread of infectious diseases. It has successfully explained important behaviors such as Achilles heels of scale-free networks and targeted immunization. It is recently reported that partial observation ability in the targeted immunization strategy can effectively influence the network robustness. In this paper, we add a further dimension to the picture by incorporating temporary immunity, where a node that acquires immunity at one step may lose it later. By accommodating limitations in space (knowledge of node) and time (immunity of node), we investigate percolation properties and scaling lows analytically for networks with arbitrary degree distributions. Distinctive characteristics for targeted immunization in Erdős-Rényi networks and power-law networks have been revealed, extending, for example, the well-known Achilles heels phenomenon under the two dimensions of limitation. We solve for both cases of a fixed fading rate of immunity and an asymptotic total loss of immunity. Our results suggest that increasing level of knowledge in targeted immunization may not be as effective as one would expect in fighting some epidemics such as COVID-19.

I. INTRODUCTION

In recent years, our understanding of dynamical phenomena of large networks against adverse events such as malicious attacks and outbreaks of infectious diseases has expanded considerably. Network robustness and disease epidemics have been extensively probed by percolation theory from statistical physics,[1–4] where nodes and edges are randomly removed or immunized to reduce connectivity and thereby prevent damage from spreading through the network structure. Targeted immunization[5,6] is highly efficient in that a fraction of key nodes (mainly based on metrics such as degree and betweenness) is immunized to effectively fragment the network and inhibit an endemic stage of the population.

One common limitation of targeted immunization strategies is that they are dependent on the full network topology information, and the metrics involved are often badly skewed when some nodes and edges are not observed.[7–9] Moreover, assuming complete network data is uneconomical and even impractical for dealing with large-scale networks in many real-life situations. A recent work[10] has developed a percolation-based theoretical framework for targeted immunization under limited knowledge, where the degree

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information of a n number of nodes is obtained at each step and the node with the highest degree of them is immunized, i.e., removed. The process continues until $1-p$ fraction of nodes are removed from the network. This framework interpolates between a random immunization with $n=1$ and a traditional targeted immunization with $n=N$. Here, N tending to infinity is the network size, mimicking a large-scale network. Interestingly, it is revealed that with a relatively small value of n, the immunization strategy may approximate the targeted immunization with full network information. Similar partially observed networks have also been studied for problems such as targeted immunization,[11,12] attack robustness,[8,13] and influence maximization.[14] These works, however, are from the perspective of blinding a portion of nodes through random or some specific sampling schemes, which is related but not equivalent to the limited knowledge notion studied in Ref. 10. A mirror process with tunable limited knowledge is investigated very recently in Ref. 15.

Another issue regarding the immunization strategy is the loss of immunity. The immunity received for a node by vaccination or rehabilitation may not be permanent due to imperfect immunity or mutations of a virus, which lead to a different disease strain and mitigate the immunity. Examples include influenza, measles, rubella, mumps, and the recent COVID-19.[16] An analogous mechanism for fear propagation in an emergency over the crowd is also well-known in social psychology.[17] In the literature of epidemic dynamics, temporary immunity has a long history and has been intensively investigated in compartmental models such as susceptible–infected–recovered–susceptible (SIRS) and susceptible–exposed–infected–recovered–susceptible (SEIRS) models,[16,18,19] where a recovered individual may become susceptible again at a certain rate. A different active line of research focuses on network self-healing by allowing various strategic recoveries of damaged nodes or links, see, e.g., Refs. 20–22 However, the above works rely on complete network data or knowledge at a specific level to determine the accurate final outbreak size.

Here, we look into the interplay and influence of a partially observed network and temporary immunity on networks motivated by the above consideration. This paper presents a concise analytical percolation theory for targeted immunization, where the degrees of n nodes are obtained at each step and we immunize the one with the highest degree with a temporary immunity extending the framework of Ref. 10. Both fixed immunity fading rate ρ and asymptotic complete loss rule $\rho(t)$ are analyzed for networks with arbitrary degree distributions. We show that although a smaller level of knowledge n is needed for Erdős–Rényi networks to achieve a full targeted immunization effect than for power-law networks for a given ρ, the latter are more tolerant to the loss of immunity when ρ is small for a given n. This observation has practical implications as power-law like networks are prevalent. It is revealed that the effectiveness of targeted immunization is markedly affected by immunity loss for networks with different structure characteristics such as average degree and degree exponents. When dynamic decay of immunity is taken into consideration, we find that the level of knowledge largely has little effect on the percolation properties regardless of the degree distribution of the underlying interaction network.

II. ANALYTICAL FRAMEWORK OF NETWORK IMMUNIZATION

Consider a configuration model[1] of network $G(V,E)$ with degree distribution $P(k)=P(k;0)$ at time step $t=0$, which is the probability of a node having k neighbors initially. Here, by convention in graph theory, V and E are the sets of nodes and edges, respectively. Denote by $N=|V|$ the number of nodes in G. For $k\geq 0$, the initial cumulative distribution function is defined as $F(k)=F(k;0)=\sum_{s=0}^{k}P(s)$, which indicates the probability of a node having degree no more than k.

Following the limited knowledge setting,[10] we assume n $(1\leq n\leq N)$ random nodes are observed at each time step t and the one with the highest degree is immunized (i.e., removed) from the network. Moreover, given a node is immunized, it has a probability ρ to revert to existence and hence may be observed again later. The probability ρ is potentially a time-dependent function to reflect the decay of immunity induced by ambient factors,[16] which we will discuss in Sec. III. For the time being, we assume that ρ is a constant. The above process continues until a fraction of $1-p$ nodes are immunized (regardless of whether it will be recovered) from $G(V,E)$. Obviously, the case $\rho=0$ suggests perfect immunity,[10,15] and the other extremal case of $\rho=1$ is trivial as no nodes will be removed and the network remains intact.

To calculate the relative fraction P_∞ of the giant component and the percolation threshold p_c, we first assume that if an immunized node remains in the immunized, i.e., deleted, state (which happens with probability $1-\rho$), we only remove the node but keep the remaining half-edges of its neighbors; cf. Fig. 1. Denote by $P(k;t)$ the degree distribution of a random node in the remaining network at time t. The corresponding cumulative distribution function is given by $F(k;t)=\sum_{s=0}^{k}P(s;t)$, which indicates the probability that a remaining node at time t has degree less than or equal to k.

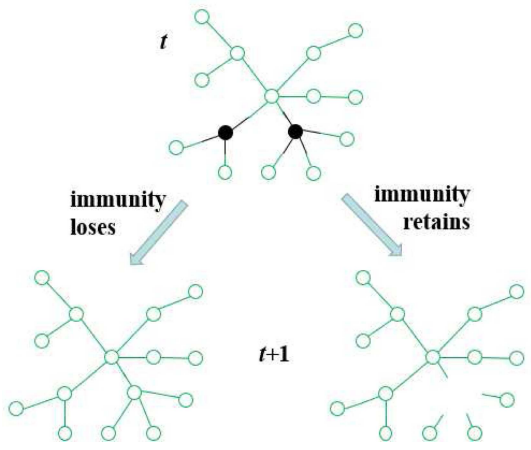

FIG. 1. At step t, $n=2$ nodes (solid) are observed. At the next step, the node with degree four is immunized, i.e., deleted (not affecting half-edges connected to its four neighbors) if immunity retains; otherwise, the immunized node will be recovered.

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In the light of the order statistics (cf. Theorem 8.1 in Ref. 23), the degree distribution $P_i(k;t)$ of the observed highest-degree node at time t is equal to

$$F(k;t)^n - F(k-1;t)^n = \Delta(F(k;t)^n) \tag{1}$$

for $k \geq 0$, where Δ is the difference operator with respect to degree k. By definition, $P(k;t) = \Delta F(k;t)$. Moreover, for any t, we set $F(-1;t) = 0$ since $P_i(0;t) = F(0;t)^n$. Recall that given a node is immunized (i.e., removed), it will be recovered with probability ρ. Let $N(k;t)$ represent the number of nodes having degree k in the remaining network at t. It follows that

$$N(k;t+1) = N(k;t) - P_i(k;t)\cdot(1-\rho) \tag{2}$$

since we only remove nodes but not edges.

In the continuous limit, by using (1) and (2), we observe that

$$\begin{aligned}\frac{\partial N(k;t)}{\partial t} &= -(1-\rho)\Delta(F(k;t)^n)\\ &= (N - t(1-\rho))\frac{\partial P(k;t)}{\partial t} - (1-\rho)P(k;t)\end{aligned} \tag{3}$$

since $N(k;t) = (N - t(1-\rho))P(k;t)$. Therefore,

$$\begin{aligned}\Delta\Bigg(&-(1-\rho)F(k;t) + (N - t(1-\rho))\frac{\partial F(k;t)}{\partial t}\\ &+ (1-\rho)F(k;t)^n\Bigg) = 0.\end{aligned} \tag{4}$$

Noting $F(-1;t) = 0$ for $t \geq 0$, we derive from (4) that for $k \geq 0$,

$$\begin{cases}(N - t(1-\rho))\dfrac{\partial F(k;t)}{\partial t}\\ \quad = (1-\rho)F(k;t) - (1-\rho)F(k;t)^n,\ t > 0,\\ F(k;0) = F(k).\end{cases} \tag{5}$$

When $n > 1$, by directly integrating (5), we arrive at

$$F(k;t) = \left(1 + (F(k)^{1-n} - 1)\cdot e^{(n-1)\ln\left(\frac{N-t(1-\rho)}{N}\right)}\right)^{-\frac{1}{n-1}}. \tag{6}$$

Since $(1-p)N = t$, the expression (6) can be recast as

$$F_p(k) = \left(1 + (F(k)^{1-n} - 1)(p + \rho(1-p))^{n-1}\right)^{-\frac{1}{n-1}}, \tag{7}$$

where $F_p(k)$ is the cumulative distribution of the degree of a random remaining node after a $1-p$ fraction of nodes are immunized (regardless of whether immunity loses). In the case of $n = 1$, taking the limit $n \to 1^+$ in (7) yields $F_p(k) = F(k)$ as one would expect.

The probability of a randomly chosen node in the remaining network having degree k, when a fraction of $1-p$ nodes are immunized (regardless of whether immunity loses), is

$$P_p(k) = \Delta F_p(k) = F_p(k) - F_p(k-1). \tag{8}$$

Let u be the probability that a random edge does not lead to the giant component. We have

$$1 - u = \sum_{k=0}^{\infty}\frac{kP(k)}{\langle k\rangle}P(O|k)(1-u^{k-1}), \tag{9}$$

where $P(O|k)$ is the probability that a node is in the remaining network given it has degree k. Since edges are retained during the process, $P(k)P(O|k) = (1-(1-\rho)(1-p))P_p(k) = (p+\rho(1-p))P_p(k)$. By (9), we obtain

$$1 - u = \frac{p + \rho(1-p)}{\langle k\rangle}\sum_{k=0}^{\infty}kP_p(k)(1-u^{k-1}). \tag{10}$$

The fraction of a giant component can be derived as follows:

$$\begin{aligned}P_\infty &= \sum_{k=0}^{\infty}P(k)P(O|k)(1-u^k)\\ &= (p+\rho(1-p))\sum_{k=0}^{\infty}P_p(k)(1-u^k),\end{aligned} \tag{11}$$

where u is determined by solving (10). Clearly, if we remove all superfluous half-edges, P_∞ will not be affected. Moreover, we note that the critical value p_c occurs when (10) starts to have solution $u < 1$. Equating the derivatives of both sides of (10) at $u = 1$ gives the critical occupation probability p_c as follows:

$$\langle k\rangle = (p_c + \rho(1-p_c))\sum_{k=2}^{\infty}k(k-1)P_{p_c}(k). \tag{12}$$

When $\rho = 1$, the network G remains at its initial status. For any n, we have $F_p(k) = F(k)$ and $P_p(k) = P(k)$ by (7) and (8). It then follows from (10) and (11) that the relative size of the giant component

$$P_\infty = \sum_{k=0}^{\infty}P(k)(1-u^k), \tag{13}$$

where u satisfies

$$u = \sum_{k=1}^{\infty}\frac{kP(k)}{\langle k\rangle}u^{k-1}. \tag{14}$$

This recovers the classical results for a configuration model [Eqs. (34) and (35) in Ref. 24] presented in the form of generating functions. Moreover, setting $\rho = 1$ in (12), we obtain the equality

$$\langle k\rangle = \sum_{k=2}^{\infty}k(k-1)P(k), \tag{15}$$

which agrees with Eq. (32) in Ref. 24 pinpointing the phase transition at which a giant component first emerges.

Erdős–Rényi (ER) random networks feature a degree distribution $P(k) = e^{-\langle k\rangle}\langle k\rangle^k/k!$ with a single parameter $\langle k\rangle$ being its average degree. The analytical and numerical results for P_∞ are shown in Fig. 2(a) with respect to different knowledge level n and immunity decay probability ρ. As one can see from the figure, the increase of n makes the network more vulnerable as high degree nodes are more likely to be removed, while the increase of ρ makes it more robust (and more harmful from the perspective of virus immunization). The influence of ρ for different n can be better appreciated in Fig. 2(b). For a given ρ, p_c saturates at a plateau around $n = 10$, meaning that a higher level of knowledge is essentially superfluous in terms of the percolation threshold p_c. Interestingly,

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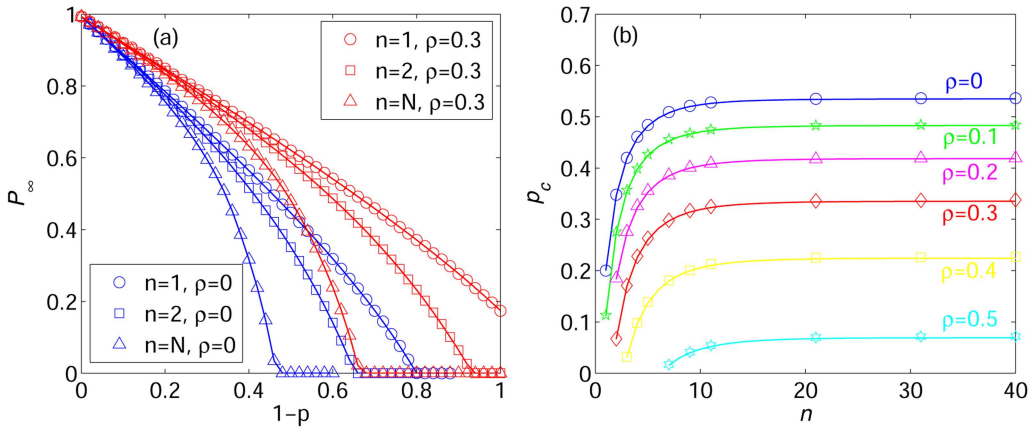

FIG. 2. (a) The giant component fraction P_∞ for ER networks with $\langle k\rangle = 5$ and $N = 10^7$ as a function of occupation probability p. The points represent numerical simulations for $\rho = 0$ (blue) and $\rho = 0.3$ (red) with $n = 1$ (circles), $n = 2$ (squares), and $n = N$ (triangles). (b) The percolation threshold p_c as a function of n for $\rho = 0$ (blue circles), $\rho = 0.1$ (green pentagrams), $\rho = 0.2$ (magenta triangles), $\rho = 0.3$ (red diamonds), $\rho = 0.4$ (yellow squares), and $\rho = 0.5$ (cyan hexagrams). Solid curves are analytical calculations.

p_c approaches zero quickly for a relatively small ρ. For example, p_c is close to zero when $\rho = 0.5$ for n less than around 10. It seems that $\rho \approx 0.4$ achieves the highest gradient of p_c for the ER networks in consideration.

The influence of network density on robustness is shown in Fig. 3 for ER networks with different average degrees. At a given decay probability ρ, denser networks are more robust, i.e., have smaller p_c, as one would expect. When $\rho = 0.3$, for example, ER networks with an average degree greater than about 6 are extremely robust against the targeted immunization strategy with knowledge level $n = 2$.

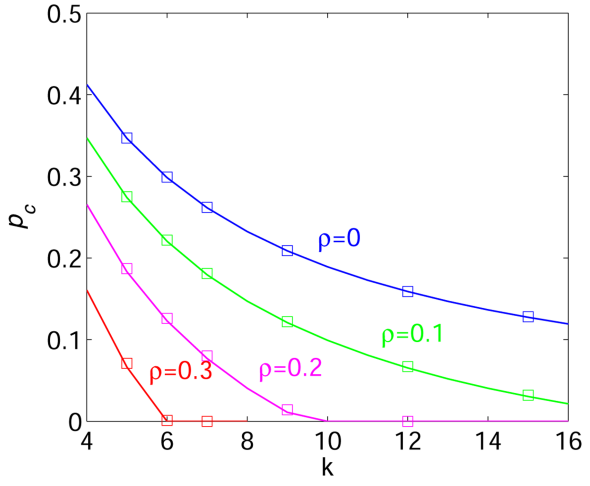

FIG. 3. The percolation threshold p_c for ER networks as a function of average degree $\langle k\rangle$ with $n = 2$. The points represent numerical simulations for $\rho = 0$ (blue), $\rho = 0.1$ (green), $\rho = 0.2$ (magenta), and $\rho = 0.3$ (red). Solid curves are analytical calculations.

In Fig. 4, we show the counterpart results for power-law networks with degree distribution $P(k) \propto k^{-\gamma}$, where the degree exponent $\gamma = 2.4$, minimum degree $k_{\min} = 2$, and cutoff $k_{\text{cut}} = 3000$. Comparing with ER networks in Fig. 2, we have the following observations. First, it generally requires a larger n (especially when ρ is large) to near the level of p_c for targeted immunization with complete knowledge (i.e., $n = N$). This confirms and extends the observation in Refs. 10 and 15. Second, for a given pair of ρ and n, the power-law networks generally have a much higher p_c than the ER networks, although both networks considered here have similar density (i.e., $\langle k\rangle \approx 5$). For instance, for $\rho = 0.5$ and $n = 20$, $p_c \approx 0.5$ in Fig. 4(b), while $p_c < 0.1$ in Fig. 2(b). The discrepancy is more prominent for larger n. This echoes the well-known robustness and fragility hallmark of scale-free networks,[25] but here, two additional dimensions have been refined in terms of targeted immunization. Note that even for the case of $\rho = 0$, such effect of n was not revealed previously in Ref. 10. Third, the variation of p_c with respect to ρ is very gentle for small ρ, and the top of the gradient of p_c is achieved at a higher value of $\rho \approx 0.7$ here compared to the situation in ER networks. This indicates that (at a given level n of knowledge) power-law networks are less sensitive to the loss of immunity during the initial stage compared to ER networks. A relevant insight from this 'higher-order" observation is that, in many real-world networks (which often feature a power-law like distribution), a low level of loss in immunity is likely to be tolerated.

In Fig. 5, we show the critical occupation probability p_c with respect to a range of degree exponents γ for power-law networks. As larger γ in our construction means sparser networks, p_c grows with the increment of γ (at any given ρ). This is similar with the observation in Fig. 3 for ER networks. From Figs. 3 and 5, we know that the influence of immunity loss on robustness (and hence the effectiveness of targeted immunization) is clearly noticeable for both homogeneous and heterogeneous networks across the spectrum of network densities and degree exponents.

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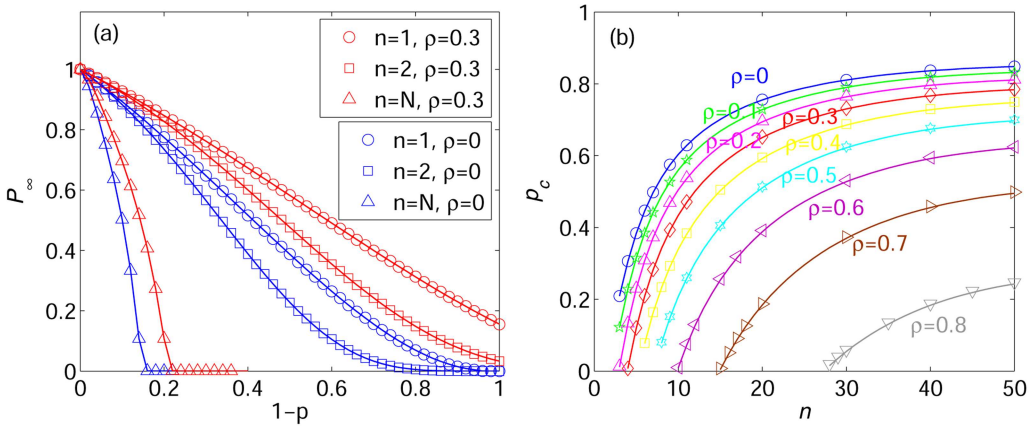

FIG. 4. (a) The giant component fraction P_∞ for power-law networks with degree exponent $\gamma = 2.4$, $k_{\min} = 2$, $k_{\text{cut}} = 3000$, and $N = 10^7$ as a function of occupation probability p. The points represent numerical simulations for $\rho = 0$ (blue) and $\rho = 0.3$ (red) with $n = 1$ (circles), $n = 2$ (squares), and $n = N$ (triangles). (b) The percolation threshold p_c as a function of n for $\rho = 0$ (blue circles), $\rho = 0.1$ (green pentagrams), $\rho = 0.2$ (magenta upward triangles), $\rho = 0.3$ (red diamonds), $\rho = 0.4$ (yellow squares), $\rho = 0.5$ (cyan hexagrams), $\rho = 0.6$ (purple left triangles), $\rho = 0.7$ (brown right triangles), and $\rho = 0.8$ (gray downward triangles). Solid curves are analytical calculations.

Next, we examine the scaling law of the percolation threshold p_c in the limit of $n \to \infty$. Let $p_c^* = p_c(n \to \infty)$. The critical occupation probability evolves as follows (see Appendix A):

$$p_c \sim p_c^* - \frac{a}{n} e^{-bn}. \tag{16}$$

Here, p_c^* is determined by the following:

$$(1-\rho)p_c^* = F(k_1) - \rho + \frac{1}{k_1 k_2}\left(\langle k\rangle - \sum_{k=2}^{k_1} k(k-1)P(k)\right), \tag{17}$$

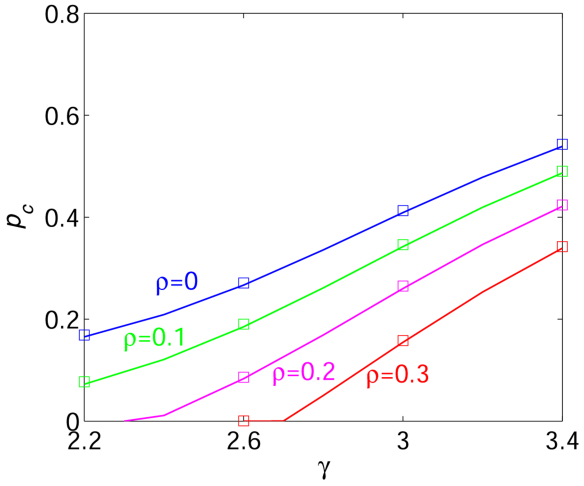

FIG. 5. The percolation threshold p_c for power-law networks as a function of degree exponent γ with $n = 3$. The points represent numerical simulations for $\rho = 0$ (blue), $\rho = 0.1$ (green), $\rho = 0.2$ (magenta), and $\rho = 0.3$ (red). Solid curves are analytical calculations.

where k_2 is the smallest degree satisfying $F(k) \geq p_c^* + \rho(1-p_c^*)$ and $k_1 = k_2 - 1$. The factor a in (16) is a constant given by $a = 2(1-\rho)^{-1}[p_c^* + \rho(1-p_c^*)]k_3/(k_1 k_2)$ and the decay rate $b = \min_k\{|\ln(p_c^* + \rho(1-p_c^*)) - \ln F(k)|\}$. By the definition of k_1 and k_2, the minimum rate b is attained at $k = k_1$ or $k = k_2$ since $F(k)$ is monotonic. The degree k_3 is the number k that attains the minimum rate b (see Appendix A).

Figures 6(a) and 6(b) present the analytical and simulation results for ER networks and power-law networks, respectively. Regardless of the specific percolation threshold p_c and immunity fading rate ρ, we find that the discrepancy follows a universal scaling law $p_c - p_c^* \sim n^{-1}$ for small values of n for both types of networks.

III. ANALYSIS OF AN ASYMPTOTIC COMPLETE LOSS OF IMMUNITY

In this section, we conduct an analysis for a type of loss of immunity characterized by a time-varying function $\rho(t)$ with $\lim_{t\to\infty}\rho(t) = 1$. In other words, immunity will be lost totally as time goes on. This is almost always the case in reality. For example, when fitting model to data, it is recently revealed that two coronaviruses have effective immunity duration around 60 weeks.[16] Specifically, we here consider the following immunity decay function:

$$\begin{cases} \rho(t) = 1 - t^{-\frac{1}{\alpha}}, & t \geq 1, \\ \rho(0) = 0, \end{cases} \tag{18}$$

where $\alpha > 1$ is a decay parameter. This function makes it amenable to analytical solutions.

With a time-dependent $\rho(t)$, we can obtain the following initial value problem for $F(k;t)$ for any $k \geq 0$ generalizing the system (5):

$$\begin{cases} (N - t(1-\rho(t)))\frac{\partial F(k;t)}{\partial t} = (1-\rho(t))F(k;t) \\ \quad -t\rho'(t)F(k;t) - (1-\rho(t))F(k;t)^n, \ t > 0, \\ F(k;0) = F(k). \end{cases} \tag{19}$$

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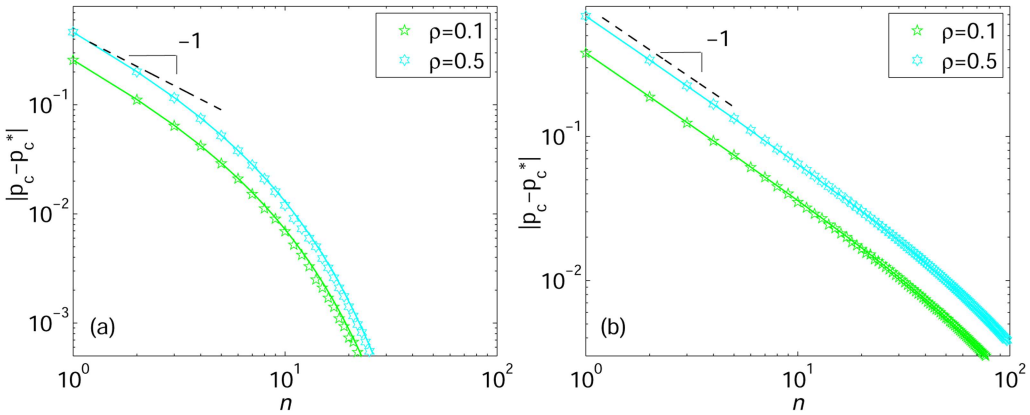

FIG. 6. The scaling law of $|p_c - p_c^*|$ for (a) ER networks with $\langle k\rangle = 5$ and (b) power-law networks with degree exponent $\gamma = 2.4$, $k_{\min} = 2$, $k_{\text{cut}} = 3000$, and $N = 10^7$ with respect to n. The points represent numerical simulations for $\rho = 0.1$ (green pentagrams) and $\rho = 0.5$ (cyan hexagrams). Solid curves are analytical calculations.

In view of (18), by setting $t = 1$ and $\rho = 0$ in (6), we have

$$F(k;1) = \left(1 + (F(k)^{1-n} - 1)\cdot e^{(n-1)\ln\left(\frac{N-1}{N}\right)}\right)^{-\frac{1}{n-1}}. \tag{20}$$

We now assume $n = o(N)$, which is relevant to most realistic large-scale networks. It is direct to check that $\left(\frac{N-1}{N}\right)^{n-1} \to 1$ as N tends to infinity. Hence, $F(k;1) = F(k)$ by (20). Invoking (18), we obtain from (19) that

$$\begin{cases} \dfrac{dF}{(1-\frac{1}{\alpha})F - F^n} = \dfrac{t^{-\frac{1}{\alpha}}dt}{N - t^{1-\frac{1}{\alpha}}}, \ t > 1, \\ F(k;1) = F(k). \end{cases} \tag{21}$$

The solution can be expressed as (see Appendix B)

$$F(k;t) = \left(\frac{\alpha}{\alpha-1}\right)^{-\frac{1}{n-1}} \cdot \left[1 + \left(\frac{\alpha-1}{\alpha}F(k)^{1-n} - 1\right) \cdot e^{(n-1)\ln\left(\frac{N-t^{\frac{\alpha-1}{\alpha}}}{N-1}\right)}\right]^{-\frac{1}{n-1}}. \tag{22}$$

Since $(1-p)N = t$ and $n = o(N)$, we obtain

$$(n-1)\ln\left(\frac{N - t^{\frac{\alpha-1}{\alpha}}}{N-1}\right) \sim -\frac{n(1-p)^{\frac{\alpha-1}{\alpha}}}{N^{\frac{1}{\alpha}}} \to 0, \tag{23}$$

as $N \to \infty$. It follows from (22) and (23) that $F(k;t) \sim F(k)$ for $t \geq 1$. In view of the initial condition at $t = 0$ in (19), we have $F(k;t) \sim F(k)$ for $t \geq 0$ or equivalently, $F_p(k) \sim F(k)$ for all $p \in [0,1]$. This means that when immunity is lost gradually following the function of (18), any knowledge of $n = o(N)$ nodes literally makes no difference for targeted immunization. This result is independent of the degree distribution of the underlying network. It is in sharp contrast with the observation in Sec. II for a fixed immunity fading rate ρ (especially for a small ρ, e.g., the case of $\rho = 0$ studied in Ref. 10). The important caveat here is that increasing the knowledge level in targeted immunization may not be as effective as one might be expecting when dealing with diseases such as COVID-19.

IV. CONCLUDING REMARKS

In conclusion, we have developed a mathematical framework of targeted immunization for studying network immunization with limited knowledge and temporary immunity. The limited knowledge of the host, where only n nodes are observed at a time, is complicated with the fading immunity of each immunized node. Percolation properties have been solved exactly for random networks with arbitrary degree distributions. Under a fixed decay probability of immunity ρ, we reveal the distinctive characteristics for targeted immunization in ER networks and power-law networks under two dimensions of n and ρ. While, given ρ, a much smaller level of knowledge n is needed for ER networks to achieve a full targeted immunization effect than for power-law networks, the latter turn out to be more tolerant to the loss of immunity when ρ is small for a given n. In the case of an asymptotic complete loss of immunity, we find that the level of knowledge is largely unaffected, which is irrespective of the degree distribution of the underlying interaction network. Our results suggest pertinent insights into network robustness and virus immunization in realistic complex processes, where limited knowledge and decay of immunization are well evidenced.

An important dimension in dealing with disease control and prevention in public health and epidemiology is the time aspect. Percolation theory, however, predominantly focuses on final outbreak size. An interesting direction would be to consider the counterpart of our framework in disease transmission models with immunization strategy. A standard SIRS model, for example, follows the dynamics,

$$\begin{cases} \frac{dS(t)}{dt} = -\beta S(t)I(t) + \lambda R(t), \\ \frac{dI(t)}{dt} = \beta S(t)I(t) - \kappa I(t), \\ \frac{dR(t)}{dt} = \kappa I(t) - \lambda R(t), \end{cases} \tag{24}$$

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where S, I, R represent the fraction of susceptible, infected, and recovered members, respectively, and β, κ, λ characterize the infection rate, the recovery rate, and the immunity loss rate, respectively, in a mass action process. The time evolution of such a system is well known. A non-vanishing infectedness, for example, indicates that the immunization strategy is not effective and the system enters into an endemic state.

It is natural to view λ on par with our parameter ρ, while β and κ can be closely related to the contact network topology and the immunization strategy. The parameters β and κ should be designed as functions of certain pertinent variables. Some variations of the design of parameters can be found in, e.g., , Refs. 26 and 27. The way to correlate epidemic models with the present theory framework and to explore the time evolution aspect would be interesting future work.

APPENDIX A: ASYMPTOTIC BEHAVIOR OF p_c AS $n \to \infty$

To investigate the convergence of p_c with respect to n from (12), we will first study $F_p(k)$ using (7). Write $F_p(k) = F_p^*(k) + \delta_p(k)$, where $F_p^*(k)$ is the constant leading term and $\delta_p(k)$ is vanishing as $n \to \infty$.

For small k satisfying $F(k) < p + \rho(1-p)$, we derive

$$\begin{aligned} F_p(k) &\sim \left(1 + \left(\frac{p+\rho(1-p)}{F(k)}\right)^n - (p+\rho(1-p))^n\right)^{-\frac{1}{n}} \\ &\sim \frac{F(k)}{p+\rho(1-p)}\left(1 + \left(\frac{F(k)}{p+\rho(1-p)}\right)^n\right)^{-\frac{1}{n}} \\ &\sim \frac{F(k)}{p+\rho(1-p)} \exp\left\{-\frac{1}{n}\left(\frac{F(k)}{p+\rho(1-p)}\right)^n\right\} \\ &\sim \frac{F(k)}{p+\rho(1-p)} - \frac{1}{n}\exp\left\{-n\ln\left(\frac{p+\rho(1-p)}{F(k)}\right)\right\}. \end{aligned} \tag{A1}$$

For $F(k) \to p + \rho(1-p)$ satisfying $[(p+\rho(1-p))/F(k)]^n \to 1$, set $\xi = 1 - (p+\rho(1-p))/F(k)$. Given $[(p+\rho(1-p))/F(k)]^n = (1-\xi)^n \sim 1 - n\xi$, we have

$$\begin{aligned} F_p(k) &\sim \left(1 + \left(\frac{p+\rho(1-p)}{F(k)}\right)^n - (p+\rho(1-p))^n\right)^{-\frac{1}{n}} \\ &\sim \left(2 - n\left(1 - \frac{p+\rho(1-p)}{F(k)}\right) - (p+\rho(1-p))^n\right)^{-\frac{1}{n}} \\ &= \exp\left\{-\frac{1}{n}\ln 2 - \frac{1}{n}\ln\left(1 - \frac{n}{2}\left(1 - \frac{p+\rho(1-p)}{F(k)}\right)\right)\right. \\ &\quad \left. -\frac{1}{2}(p+\rho(1-p))^n\right\} \\ &\sim 1 - \frac{\ln 2}{n} + \frac{1}{2}\left(1 - \frac{p+\rho(1-p)}{F(k)}\right). \end{aligned} \tag{A2}$$

For large k satisfying $p + \rho(1-p) < F(k) < 1$,

$$\begin{aligned} F_p(k) &\sim \left(1 + \left(\frac{p+\rho(1-p)}{F(k)}\right)^n - (p+\rho(1-p))^n\right)^{-\frac{1}{n}} \\ &\sim \exp\left\{-\frac{(p+\rho(1-p))^n}{nF(k)^n} + \frac{(p+\rho(1-p))^n}{n}\right\} \\ &\sim 1 - \frac{(p+\rho(1-p))^n}{nF(k)^n} + \frac{(p+\rho(1-p))^n}{n} \\ &\sim 1 - \frac{1}{n}e^{-n[\ln F(k) - \ln(p+\rho(1-p))]}. \end{aligned} \tag{A3}$$

When $F(k) = 1$, we clearly have $F_p(k) = 1$.

Consequently, the leading term of $F_p(k)$ can be given by

$$F_p^*(k) \sim \begin{cases} \dfrac{F(k)}{p+\rho(1-p)}, & F(k) < p+\rho(1-p), \\ 1, & F(k) > p+\rho(1-p), \end{cases} \tag{A4}$$

and the vanishing error term is

$$\delta_p(k) = \begin{cases} -\frac{1}{n}e^{-b(k)n}, & F(k) < 1, \\ 0, & F(k) = 1, \end{cases} \tag{A5}$$

where the decay rate is $b(k) = |\ln(p+\rho(1-p)) - \ln F(k)|$.

With the decomposition of the distribution $F_p(k)$ as above, we then investigate the asymptotic behavior of the critical occupation probability p_c. Define $k_1 = \max_{F(k)<p+\rho(1-p)} k$, $k_2 = \min_{F(k)\geq p+\rho(1-p)} k$, and $k_3 = \min_{F(k)=1} k$. Employing (12), (A4), and (A5), we obtain

$$\begin{aligned} &\frac{\langle k\rangle}{p_c + \rho(1-p_c)} \\ &= \sum_{k=2}^{\infty} k(k-1)\Delta F_{p_c}(k) \\ &= \sum_{k=2}^{\infty} k(k-1)\Delta F_{p_c}^*(k) + \sum_{k=2}^{k_3} k(k-1)\Delta\delta_{p_c}(k) \\ &\sim \sum_{k=2}^{k_1} k(k-1)\frac{P(k)}{p_c+\rho(1-p_c)} + k_1k_2\left(1 - \frac{F(k_1)}{p_c+\rho(1-p_c)}\right) \\ &\quad - 2\sum_{k=1}^{k_3} k\delta_{p_c}(k). \end{aligned} \tag{A6}$$

Let $p_c^* = \lim_{n\to\infty} p_c$ and express p_c as $p_c = p_c^* + \delta_c$, where constant p_c^* is the leading term and the next term δ_c tends to zero as $n \to \infty$. We solve p_c^* via the following equation:

$$\begin{aligned} \frac{\langle k\rangle}{p_c^* + \rho(1-p_c^*)} &= k_1k_2\left(1 - \frac{F(k_1)}{p_c^*+\rho(1-p_c^*)}\right) \\ &\quad + \sum_{k=2}^{k_1} k(k-1)\frac{P(k)}{p_c^*+\rho(1-p_c^*)}, \end{aligned} \tag{A7}$$

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where k_1 and k_2 are determined by p_c^* herein, and they are independent of n. Employing the Taylor series at p_c^*, (A6) and (A7), we obtain

$$\delta_c \sim \hat{a} \sum_{k=1}^{k_3} k\delta_{p_c^*}(k), \tag{A8}$$

where $\hat{a} = 2(1-\rho)^{-1}(p_c^* + \rho(1-p_c^*))/(k_1 k_2)$. Thanks to (A5) and the monotonicity of $F(k)$, it is clear that the dominant decay rate becomes $b = \min_k b(k) = \min_k\{|\ln(p_c^* + \rho(1-p_c^*)) - \ln F(k)|\}$, where the optimum is achieved at k_1 or k_2. Pulling these observations together, we have

$$p_c \sim p_c^* - \frac{\hat{a}k_3}{n} e^{-bn} \tag{A9}$$

as $n \to \infty$, where k_3 is the degree k that achieves the minimum rate b.

APPENDIX B: SOLVING THE SYSTEM (21)

It is relatively straightforward to solve this system by integration. Replace t with s on the right-hand side of the equation and integrate it over the interval $[1, t]$ with respect to s. We have

$$\int_1^t \frac{s^{-\frac{1}{\alpha}} ds}{N - s^{1-\frac{1}{\alpha}}} = c_1 - \frac{\alpha}{\alpha - 1} \ln\left(N - t^{1-\frac{1}{\alpha}}\right) \tag{B1}$$

for some constant c_1. Integrate the left-hand side of the equation similarly gives

$$\begin{aligned} &\int_1^t \frac{dF}{(1-\frac{1}{\alpha})F - F^n} \\ &= \frac{\alpha}{\alpha-1}\int_1^t \frac{1}{F} dF + \frac{\alpha}{\alpha-1}\int_1^t \frac{F^{n-2}}{1 - \frac{1}{\alpha} - F^{n-1}} dF \\ &= c_2 + \frac{\alpha}{\alpha-1} \cdot \frac{1}{n-1} \ln\left(\frac{F^n}{\left(1-\frac{1}{\alpha}\right)F - F^n}\right) \end{aligned} \tag{B2}$$

for some constant c_2. Equating (B1) with (B2), we derive

$$F(k;t) = \left[\frac{\alpha}{\alpha-1}\left(1 + c_3 e^{(n-1)\ln\left(N - t^{\frac{\alpha-1}{\alpha}}\right)}\right)\right]^{-\frac{1}{n-1}} \tag{B3}$$

for some constant c_3. Setting $t = 1$ in (B3) and use the initial condition, we arrive at

$$c_3 = \left(\frac{\alpha-1}{\alpha} F(k)^{1-n} - 1\right) \cdot e^{-(n-1)\ln(N-1)}, \tag{B4}$$

which readily yields the solution.

DATA AVAILABILITY

The data that support the findings of this study are available within the article.

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