Infection dynamics on scale-free networks

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We discuss properties of infection processes on scale-free networks, relating them to the node-connectivity distribution that characterizes the network. Considering the epidemiologically important case of a disease that confers permanent immunity upon recovery, we derive analytic expressions for the final size of an epidemic in an infinite closed population and for the dependence of infection probability on an individual's degree of connectivity within the population. As in an earlier study [R. Pastor-Satorras and A. Vesipignani, Phys. Rev. Lett. **86**, 3200 (2001); Phys. Rev. E. **63**, 006117 (2001)] for an infection that did not confer immunity upon recovery, the epidemic process—in contrast with many traditional epidemiological models—does not exhibit threshold behavior, and we demonstrate that this is a consequence of the extreme heterogeneity in the connectivity distribution of a scale-free network. Finally, we discuss effects that arise from finite population sizes, showing that networks of finite size do exhibit threshold effects: infections cannot spread for arbitrarily low transmission probabilities.

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A wide class of infection processes can be modeled using a network-based approach, in which individuals are modeled as nodes and possible contacts between individuals by edges between the nodes [1,2]. An immediate question that then arises is how the properties of the disease and network topology combine to determine the dynamics of the infection. Recent work [1], inspired by the study of large real-world social and communication networks [3–6], has examined the spread of computer viruses [7,8] on the scale-free networks [9,10] that provide a good description of the connectivity structure seen in the Internet and the World Wide Web [4,5]. Using a susceptible-infected-susceptible (SIS) model (in which infected nodes recover to a susceptible state), it was shown that epidemic processes on scale-free networks exhibit several unexpected behaviors. In particular, they do not exhibit the threshold phenomenon typically seen in epidemiology: computer viruses can spread and persist even when the probability of transmission is vanishingly small [1].

Mathematical epidemiologists have long appreciated the important role played by heterogeneity in population structure in determining properties of disease invasion, spread and persistence, and consequently have developed many techniques to facilitate the study of disease spread in heterogeneous populations and derived many general results [11,12]. Here, we employ these techniques and results to study the infection dynamics of epidemics on scale-free networks, whose node-connectivity distribution (i.e., the distribution of probabilities that nodes have exactly k neighbors) follows a power law of the form $P(k)\sim k^{-\nu}$, where $2<\nu\leqslant 3$, and for which the least-connected nodes have connectivity m. We first focus on the $\nu=3$ case, which corresponds to the scale-free network generated by the simplest algorithm of Barabási and Albert [9], and then generalize to $2<\nu\leqslant 3$ [10].

We consider a description of the infection process that, compared with the SIS model, is more appropriate both for many epidemiological situations and also, we argue, for computer viruses. We assume that nodes do not recover to a susceptible state, but rather are permanently immune to further infection (either by the development of an appropriate immune response, or by the installation of antiviral software). The resulting model is known as a susceptible-infected-recovered (SIR) model. The long-term maintenance of the infection is now impossible in a closed population due to the depletion of susceptible nodes as the epidemic spreads through the population. In general, if there were a sufficiently rapid input of new susceptibles (either by births, or with the addition of new, unprotected computers to the network), an endemic level of infection could be reached, but in this study we restrict our attention to the case of a closed population.

We assume that the probability of a susceptible node acquiring infection from a given neighboring infected node in a short time interval dt is βdt, and the the rate at which infected nodes recover is γ, i.e., the average duration of infection is $D=1/\gamma$. Furthermore, we assume that the mixing pattern of the population is random, by which we mean that the probability that a given neighbor of a node of type i is of type j depends only on the node-connectivity distribution of the population, which means that the probability is given by $jP(j)/\Sigma kP(k)$ (see, for instance, Refs. [11,12]).

Assuming an infinite population, we can formulate the network model in way directly equivalent to that used by epidemiologists to study the transmission dynamics of sexually transmitted diseases, such as gonorrhoea and HIV [11–13]. We denote the fraction of nodes that are both of type i (i.e., have i neighbors) and which are susceptible to infection by x_i, and the corresponding fraction of nodes that are both of type i and are infected by y_i. The time evolution of these quantities is given by

$$\dot{x}_i=-x_i\sum_j \beta_{ij}y_j \tag{1}$$

$$\dot{y}_i=x_i\sum_j \beta_{ij}y_j-\gamma y_i, \tag{2}$$

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for $i \geqslant m$ and where β_{ij} denotes the per capita infection rate at which sites of type i acquire infection from sites of type j. Given the previously discussed assumption of random mixing, $\beta_{ij} = ij\beta/\langle k \rangle$. Here $\langle k \rangle$ denotes the average connectivity of the nodes. (Throughout this paper, we denote the averaged value of a function $f(k)$ taken over the networks by $\langle f(k) \rangle$. For the analysis of the infinite system, we approximate the infinite sums involved in such averages by the corresponding integrals.) In the initial disease-free state, before the introduction of infection, all individuals are susceptible, so $y_j = 0$ for all j, and $x_j = P(j)$.

We define $\rho_0 = \beta D \langle k \rangle$. The quantity ρ_0 would be the average number of secondary infections caused by the introduction of a single infected individual into an entirely susceptible population, if the population were homogeneous (i.e., if every individual had exactly $\langle k \rangle$ neighbors). This average number of secondary infections is a central quantity in epidemiological theory, and is known as the basic reproductive number [12].

The standard epidemiological theory [11,12] shows that this model exhibits threshold behavior, with the introduction of infection leading to an epidemic outbreak if $R_0 > 1$, where, for the heterogeneous network defined above, the basic reproductive number R_0 equals [12,14]

$$R_0 = \rho_0 (1 + C_V^2). \tag{3}$$

Here C_V denotes the coefficient of variation of the connectivity distribution, i.e., $C_V^2 = \langle k^2 \rangle / \langle k \rangle^2 - 1$. For the scale-free distributions considered here, C_V is infinite because the variance of the connectivity distribution is infinite. In contrast with the case of a homogeneous network, which exhibits threshold behavior at $\rho_0 = 1$, R_0 for the scale-free network (at least for the infinite population case) is infinite for any nonzero transmission probability and so an outbreak can always occur [1].

Furthermore, the epidemic theory [11,12] shows [by direct integration of Eqs. (1) and (2)] that the fraction of nodes ever infected (the so-called final epidemic size) I equals

$$I = \langle 1 - e^{-k\alpha} \rangle, \tag{4}$$

where α is determined by the equation

$$\alpha = \rho_0 \frac{\langle k(1 - e^{-k\alpha}) \rangle}{\langle k \rangle^2}. \tag{5}$$

In order to calculate these averages, as discussed above, we approximate k by a continuous quantity, taking $P(k) = 2m^2/k^3$. Here m denotes the connectivity of the least connected nodes and the constant of proportionality is determined by requiring $\int_m^\infty P(k)dk = 1$. The average connectivity of the nodes is $\langle k \rangle = 2m$ and the second moment, $\langle k^2 \rangle$, is the limiting value of $2m^2 \ln(k_{\max}/m)$ as $k_{\max} \to \infty$, which is infinite.

Substituting this form of $P(k)$ into expresion (5) gives

$$\alpha = \rho_0 \frac{2m^2}{4m^2} \int_m^\infty \frac{dk}{k^2} (1 - e^{-k\alpha}), \tag{6}$$

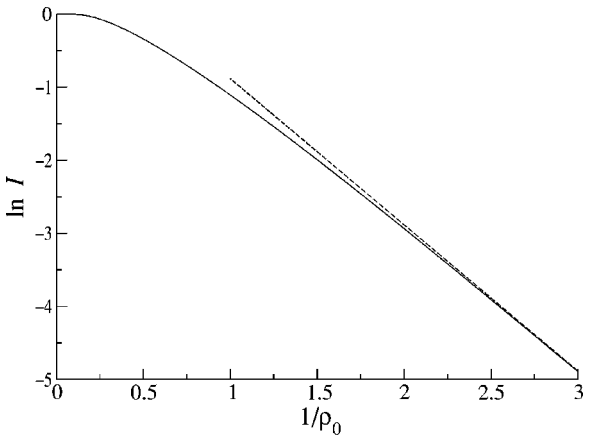

FIG. 1. Fraction ever infected in the SIR model on a scale-free network with $\nu = 3$ and $m = 4$. Solid curve gives the exact solution obtained from Eq. (9), and the broken curve the approximation (12), which is indeed a good approximation for $\rho_0 \ll 1$.

and defining $\phi = m\alpha$ and $k = mx$, we get

$$\phi = \frac{\rho_0}{2} \int_1^\infty \frac{dx}{x^2} (1 - e^{-\phi x}) = \frac{\rho_0}{2} [1 - E_2(\phi)], \tag{7}$$

where $E_2(\phi)$ denotes the exponential integral of the second kind of ϕ. Use of a standard formula [15] [Eq. (5.1.14)] allows Eq. (7) to be rewritten in terms of exponential integrals of the first kind,

$$\frac{2}{\rho_0} = \left(\frac{1 - e^{-\phi}}{\phi} \right) + E_1(\phi). \tag{8}$$

This equation can be solved (at least numerically) to obtain $\phi(\rho_0)$ and hence $I(\rho_0)$ can be obtained by substituting into Eq. (4),

$$I = 2m^2 \int_m^\infty \frac{dk}{k^3} (1 - e^{-k\alpha}) = 1 - 2E_3(\phi). \tag{9}$$

When $\rho_0 \ll 1$, we can find an approximate analytic solution of Eq. (8) and hence the approximate final epidemic size. It is easy see that if $\rho_0 \ll 1$, then ϕ must be small, so we make use of the small value expansion of $E_1(\phi)$ [15] [Eq. (5.1.11)] to give

$$2/\rho_0 = -\ln \phi + (1 - \gamma) + \phi/2 + \ldots, \tag{10}$$

here γ is the Euler-Mascheroni constant, $0.577\ldots$. Hence

$$\phi = \kappa e^{-2/\rho_0} \{1 + O(e^{-2/\rho_0})\}, \tag{11}$$

where $\kappa = e^{1-\gamma} \approx e^{0.423\ldots} = 1.527\ldots$. Finally, use of a standard expression for $E_3(\phi)$ [15] [Eq. (5.1.12)] gives $I = 2\phi\{1 + O(\phi \ln \phi)\}$, or

$$I = 2\kappa e^{-2/\rho_0} \left\{ 1 + O\left(\frac{e^{-2/\rho_0}}{\rho_0} \right) \right\}. \tag{12}$$

Figure 1 illustrates final epidemic sizes calculated using both exact (9) and approximate (12) solutions.

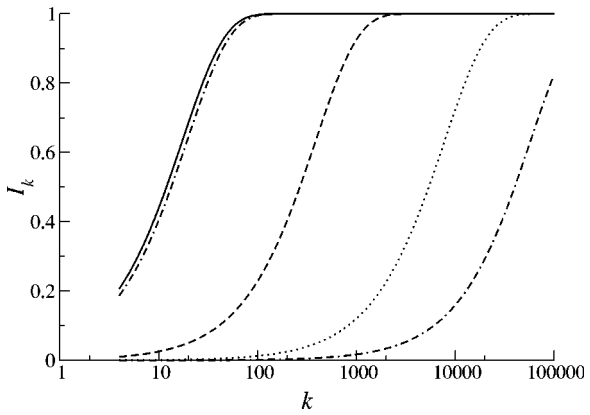

FIG. 2. Fraction of nodes of each connectivity type that are ever infected in the SIR epidemic of Fig. 1. The broken curves show results for four different values of ρ_0 (from left to right: 1.0, 0.4, 0.25, and 0.2), and the solid curve illustrates, for $\rho_0=1$, the approximation discussed in the text. (For the smaller values of ρ_0, the approximate curves are indistinguishable from the corresponding exact curves.)

Once ϕ is known, expressions for the fraction of nodes of connectivity k, which are ever infected during the course of the epidemic can be obtained. In terms of the scaled variable $i=k/m$, the fraction of nodes of type i that are ever infected is given by the standard (exact) result [11,12] that $I_i=1-e^{-i\phi}$. For $\rho_0 \ll 1$, this can be approximated as $I_i \approx 1-\exp(-i/i_c)$, where $i_c=e^{2/\rho_0}/\kappa$. We notice that, in general, few individuals are infected in the low-connectivity classes (although there are, of course, lots of these) but essentially all individuals are infected in high-connectivity classes (Fig. 2).

The above results can be generalized to a more general class of scale-free networks for which the exponent ν in the power law lies between 2 and 3 [10]. For this general case, we have that $P(k)=(\nu-1)m^{\nu-1}/k^{\nu}$, $\langle k\rangle=(\nu-1)/(\nu-2)$, and $\langle k^2\rangle$ is the limiting value of $(\nu-1)m^2\{(k_{\max}/m)^2-1\}/(3-\nu)$ as $k_{\max}\to\infty$, which is again infinite.

As before, substituting $P(k)$ into Eq. (5) gives an implicit equation that determines α. In terms of the scaled variable $\phi=m\alpha$, we then have

$$\phi=\rho_0\frac{(\nu-2)^2}{(\nu-1)}\phi^{\nu-2}\int_{\phi}^{\infty}\frac{ds}{s^{\nu-1}}(1-e^{-s}). \tag{13}$$

Once α (or ϕ) has been found, an expression for I follows upon substitution into Eq. (4)

$$I=(\nu-1)\int_1^{\infty}\frac{dx}{x^{\nu}}(1-e^{-x\phi}). \tag{14}$$

As before, if $\rho_0 \ll 1$ then ϕ must be small, and performing an integration by parts, one can show that the integral in expression (13) is given by

$$\int_{\phi}^{\infty}\frac{ds}{s^{\nu-1}}(1-e^{-s})=\frac{\Gamma(3-\nu)}{\nu-2}\{1+O(\phi^{3-\nu})\}, \tag{15}$$

and hence that ϕ is given by

$$\phi=\left[\left(\frac{\nu-2}{\nu-1}\right)\Gamma(3-\nu)\rho_0\right]^{1/(3-\nu)}\{1+O(\rho_0)\}. \tag{16}$$

Performing a similar manipulation on the integral in expression (14) and substituting the now known value of ϕ gives

$$I=\left(\frac{\nu-1}{\nu-2}\right)\left[\left(\frac{\nu-2}{\nu-1}\right)\Gamma(3-\nu)\rho_0\right]^{1/(3-\nu)} \times\{1+O(\rho_0,\rho_0^{(\nu-2)/(3-\nu)})\}. \tag{17}$$

Clearly, one could obtain results for the fraction of nodes of type i ever infected, as in the $\nu=3$ special case discussed above. But the interesting point here is that the essential singularity, $I\sim e^{-2/\rho_0}$, of the $\nu=3$ case is now replaced by milder power laws ($I\sim\rho_0^{1/(3-\nu)}$). As an example, for $\nu=2.5$, $\phi\approx(\pi^{1/2}\rho_0/3)^2\{1+O(\rho_0)\}$ and $I\approx(\pi/3)\rho_0^2\{1+O(\rho_0)\}$.

We finish this study with a brief discussion of finite-size effects. The deterministic model, as described by Eqs. (1) and (2), consists of an infinite set of equations. This set may be truncated (e.g., for simulation purposes) at connectivity $k=k_{\max}$. We take $P(k)=C/k^{\nu}$, but note that C is dependent on $k_{\max}$.

The variance of the node-connectivity distribution is still large, but not infinite; hence R_0 [Eq. (3)] is no longer infinite. The finite model does exhibit threshold effects, albeit for much lower transmission probabilities than for the corresponding homogeneous situations. Using the continuous approximation discussed above, it is easy to see that, for $\nu=3$, $R_0\approx\frac{1}{2}\rho_0\ln(k_{\max}/m)$. Whilst it is straightforward to interpret finite-size effects in terms of $k_{\max}$, it is a little more tricky to interpret $k_{\max}$ in terms of the number of nodes N in the finite network. A rough argument, using the continuous approximation for $P(k)$ shows that $k_{\max}/m\approx N^{1/3}$ and that $R_0\approx\frac{1}{6}\rho_0\ln N$. As an example, this means that for a network of size $N=10^5$, with $m=4$, $k_{\max}\approx 190$; for comparison, such networks generated using the Barabási and Albert algorithm [9] have, on average, $k_{\max}\approx 700$.

Figure 3 illustrates this finite-size effect: disease invasion does indeed require higher transmission probabilities when $k_{\max}$ is smaller, as the vertical asymptotes of I for the finite models clearly demonstrate the existence of threshold behavior. This result contradicts the claim [1] that threshold behavior is absent in networks of finite size. We remark that, for all but the smallest-sized networks, the curves in Fig. 3 closely follow the analytic solution of the infinite model when viewed over a restricted range of values of $1/\rho_0$, and that the figures used to support the claim [1] employ somewhat restricted ranges of transmission probabilities, with relatively large transmission probabilities used for the smaller networks.

A second finite size effect arises from demographic stochasticity—the random effects that arise from the population consisting of a discrete set of individuals. For instance, in stochastic simulations of an epidemic on a given network, there will always be some chance that an initial infective

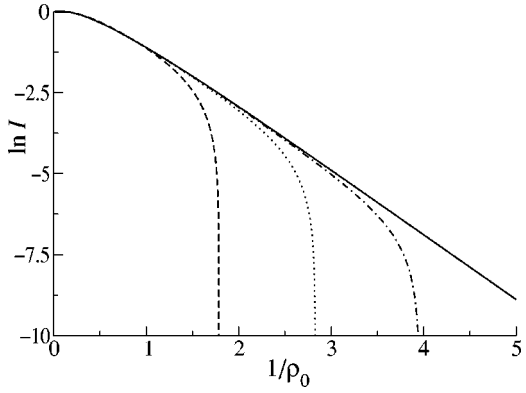

FIG. 3. Fraction ever infected in the SIR model, as in Fig. 1, with the heavy solid curve denoting the analytic solution for the infinite model, and broken curves denoting quantities calculated for the finite model with $k_{max}=m$ (i.e., a homogeneous network), 100, 1000, and 10 000 (from left to right). Notice the threshold phenomenon seen as the epidemic-size curves in finite models approach vertical asymptotes.

individual will recover before transmission of infection occurs, particularly if that initial infective has a low number of neighbors [14]. Further discussion of finite-size effects will appear elsewhere [16].

Our analysis assumes random mixing and ignores local network structure: both assumptions have received considerable attention in the epidemiological literature [17–19]. One alternative to random mixing is assortative mixing, in which individuals preferentially interact with similarly connected individuals. This increases the initial rate of increase of the epidemic, but reduces the final epidemic size [17,18]. Local structure slows disease spread, as the shortest path joining two individuals often involves many intermediates and also as such networks exhibit clique behavior, with pairs of connected individuals sharing many common neighbors [19]. Long-range transmission events, however, even if relatively infrequent, can substantially enhance disease spread [3].

An important finding that emerges from our analysis is the crucial role played by the most highly connected nodes in spreading infection and, in the SIS model, in maintaining infection. This has clear implications for control strategies for diseases that spread over heterogeneous networks. Clearly, control programs should be targetted towards the most highly connected nodes, and such programs will be much more effective than those that target nodes at random. We remark that this result is clearly analogous to recent observations made regarding the attack tolerance of scale-free networks [20]. Furthermore, this result is well known to epidemiologists [11–13]; its use in public health policy (for instance, programs to prevent the spread of sexually transmitted diseases often target high-risk groups, such as prostitutes) testifies that this is no mere academic curiosity.

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