Exact solution of site and bond percolation on small-world networks

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We study percolation on small-world networks, which has been proposed as a simple model of the propagation of disease. The occupation probabilities of sites and bonds correspond to the susceptibility of individuals to the disease, and the transmissibility of the disease respectively. We give an exact solution of the model for both site and bond percolation, including the position of the percolation transition at which epidemic behavior sets in, the values of the critical exponents governing this transition, the mean and variance of the distribution of cluster sizes (disease outbreaks) below the transition, and the size of the giant component (epidemic) above the transition.

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I. INTRODUCTION

In the late 1960s, Milgram performed a number of experiments that led him to conclude that, despite there being several billion human beings in the world, any two of them could be connected by only a short chain of intermediate acquaintances of typical length about six [1]. This result, known as the ''small-world effect,'' has been confirmed by subsequent studies and is now widely believed to be correct, although opinions differ about whether six is an accurate estimate of the typical chain length [2].

The small-world effect can be easily understood in terms of random graphs [3] for which typical vertex-vertex distances increase only as the logarithm of the total number of vertices. However, random graphs are a poor representation of the structure of real social networks, which show a ''clustering'' effect in which there is an increased probability of two people being acquainted if they have another acquaintance in common. This clustering is absent in random graphs. Recently, Watts and Strogatz [4] have proposed a new model of social networks that possesses both short vertex-vertex distances and a high degree of clustering. In this model, sites are arranged on a one-dimensional lattice of size L, and each site is connected to its nearest neighbors up to some fixed range k. Then additional links—''shortcuts''—are added between randomly selected pairs of sites with probability ϕ per link on the underlying lattice, giving an average of ϕkL shortcuts in total. The short-range connections produce the clustering effect while the long-range ones give average distances that increase logarithmically with system size, even for quite small values of ϕ.

This model, commonly referred to as the ''small-world model,'' has attracted a great deal of attention from the physics community. A number of authors have looked at the distribution of path lengths in the model, including scaling forms [5–7] and mean-field and exact results [8,9], while others have looked at a variety of dynamical systems on small-world networks [4,10,11]. A review of recent developments can be found in Ref. [12].

One of the most important consequences of the small-world effect is in the propagation of disease. Clearly a disease can spread much faster through a network in which the typical person-to-person distance is $O(\log L)$ than it can through one in which the distance is $O(L)$. Epidemiology recognizes two basic parameters governing the effects of a disease: the *susceptibility*—the probability that an individual exposed to a disease will contract it—and the *transmissibility*—the probability that infection results from a contact between an infected individual and a healthy but susceptible one. Newman and Watts [6] studied a model of disease in a small world which incorporates these variables. In this model a randomly chosen fraction p of the sites or bonds in the small-world model are ''occupied'' to represent the effects of finite susceptibility and transmissibility, and a disease outbreak that starts with a single individual can spread only within a connected cluster of occupied sites or bonds. Thus the problem of disease spreading maps onto a site or bond percolation problem. (The model is also similar to the so-called SIR models of the spread of infectious disease [13].) At some threshold value p_c of the percolation probability, the system undergoes a percolation transition which corresponds to the onset of epidemic behavior for the disease in question. Newman and Watts gave an approximate solution for the position of this transition.

In this paper, we give an exact solution for both site and bond percolation on small-world networks using a generating function method. Our method gives not only the exact position of the percolation threshold, but also the value of the critical exponents governing behavior close to the transition, the complete distribution of the sizes of disease outbreaks for any value of p, and closed-form expressions for the mean and variance of the distribution. A calculation of the value of p_c only, using a transfer-matrix method, has appeared previously in Ref. [14].

The basic idea behind our solution is to find the distribution of ''local clusters''—clusters of occupied sites or bonds on the underlying lattice—and then calculate how the shortcuts join these local clusters together to form larger ones. We focus on the quantity $P(n)$, which is the probability that a randomly chosen site belongs to a connected cluster of n sites. This is also the probability that a disease outbreak starting with a randomly chosen individual will affect n people. It is *not* the same as the distribution of cluster sizes for the percolation problem, since the probability of an outbreak

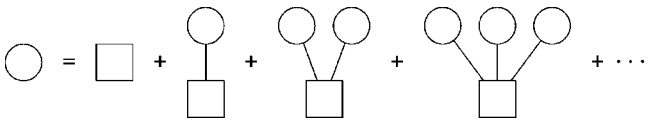

FIG. 1. Graphical representation of a cluster of connected sites. The entire cluster (circle) is equal to a single local cluster (square), with any number $m \geq 0$ of connected clusters attached to it by a single shortcut.

starting in a cluster of size n increases with cluster size in proportion to n, all other things being equal. The cluster size distribution is therefore proportional to $P(n)/n$. This distribution can be calculated easily from the results given in this paper, although we will not do so.

The outline of this paper is as follows. In Sec. II we derive a complete solution for site percolation on the small-world network. In Sec. III we do the same for bond percolation for $k=1$ and $k=2$, indicating how solutions for higher k can be obtained. In Sec. IV we discuss simultaneous site and bond percolation. In Sec. V we give our conclusions.

II. SITE PERCOLATION

We start by examining the site percolation problem, which is the simpler case. We consider first the situation below the percolation threshold. Since $P(n)$ is difficult to evaluate directly, we turn to a generating function method for its calculation. We define

$$H(z)=\sum_{n=0}^{\infty} P(n)z^n. \tag{1}$$

For $p<p_c$, as we show below, the distribution of clusters falls off exponentially with cluster size. This implies that the probability of two shortcuts connecting the same pair of clusters varies as L^{-1} and so can be neglected in the limit of large L. In this limit, therefore, any connected cluster of sites consists of a local cluster with $m \geq 0$ shortcuts leading from it to m other clusters. Thus $H(z)$ satisfies the Dyson-equation-like iterative condition illustrated graphically in Fig. 1: every cluster of connected sites consists of a single local cluster joined by shortcuts to some number m of other clusters. Thus we can write $H(z)$ self-consistently as

$$H(z)=\sum_{n=0}^{\infty} P_0(n)z^n \sum_{m=0}^{\infty} P(m|n)[H(z)]^m. \tag{2}$$

In this equation $P_0(n)$ is the probability of a randomly chosen site belonging to a local cluster of size n, which is

$$P_0(n)=\begin{cases} 1-p & \text{for } n=0 \\ npq^{n-1}(1-q)^2 & \text{for } n \geq 1, \end{cases} \tag{3}$$

with $q=1-(1-p)^k$. $P(m|n)$ is the probability of there being exactly m shortcuts emerging from a local cluster of size n. Since there are $2\phi kL$ ends of shortcuts in the network, $P(m|n)$ is given by the binomial

$$P(m|n)=\binom{2\phi kL}{m}\left[\frac{n}{L}\right]^m\left[1-\frac{n}{L}\right]^{2\phi kL-m}. \tag{4}$$

Using this expression Eq. (2) becomes

$$H(z)=\sum_{n=0}^{\infty} P_0(n)z^n\left[1+(H(z)-1)\frac{n}{L}\right]^{2\phi kL}$$

$$=\sum_{n=0}^{\infty} P_0(n)[ze^{2k\phi[H(z)-1]}]^n, \tag{5}$$

for L large. The remaining sum over n can now be performed conveniently by defining

$$H_0(z)=\sum_{n=0}^{\infty} P_0(n)z^n=1-p+pz\frac{(1-q)^2}{(1-qz)^2}, \tag{6}$$

where we have made use of Eq. (3). $H_0(z)$ is the generating function for the local clusters. Now we notice that $H(z)$ in Eq. (5) is of the same form as $H_0(z)$, but with $z \to ze^{2k\phi[H(z)-1]}$. Thus

$$H(z)=H_0(ze^{2k\phi[H(z)-1]}). \tag{7}$$

A similar equation has been derived for SIR models by Ball *et al.* [15].

$H(z)$ can be calculated directly by iteration of this equation starting with $H(z)=1$ to give the complete distribution of sizes of epidemics in the model. It takes n steps of the iteration to calculate $P(n)$ exactly. The first few steps give

$$P(0)=1-p, \tag{8}$$

$$P(1)=p(1-q)^2e^{-2k\phi p}, \tag{9}$$

$$P(2)=p(1-q)^2[2q+2k\phi p(1-q)^2]e^{-4k\phi p}. \tag{10}$$

It is straightforward to verify that these are correct. We could also iterate Eq. (7) numerically and then estimate $P(n)$ us-

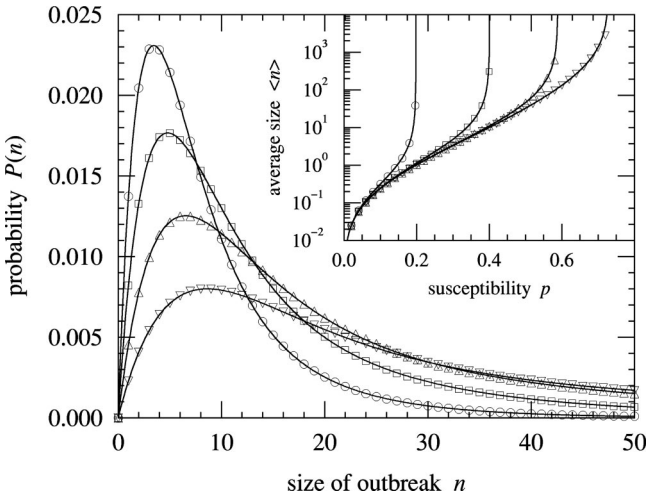

FIG. 2. The distribution of outbreak sizes in simulations of the site percolation model with $L=10^7$, $k=5$, $\phi=0.01$, and $p=0.25$, 0.30, 0.35, and $p=p_c=0.401\,01$ (circles, squares, and up- and down-pointed triangles, respectively). The solid lines are the same distributions calculated using Eqs. (7) and (11). Inset: The average size of disease outbreaks as a function of p for (left to right) $\phi=10^{-1},10^{-2},10^{-3},10^{-4}$. The points are numerical results for $L=10^7$, $k=5$ and the solid lines are the exact result, Eq. (12).

ing, for instance, forward differences at $z=0$. Unfortunately, like many calculations involving numerical derivatives, this method suffers from severe machine-precision problems which limit us to small values of n, on the order of $n \lesssim 20$. A much better technique is to evaluate $H(z)$ around a contour in the complex plane and calculate the derivatives using the Cauchy integral formula

$$P(n)=\frac{1}{n!}\frac{d^n H}{dz^n}\bigg|_{z=0}=\frac{1}{2\pi i}\oint \frac{H(z)}{z^{n+1}}dz. \tag{11}$$

A suitable choice of contour in the present case is the unit circle $|z|=1$. Using this method we have been able to calculate the first thousand derivatives of $H(z)$ without difficulty.

In Fig. 2 we show the distribution of outbreak sizes calculated from Eq. (11) for a variety of values of p below the percolation threshold. On the same plot we also show the distribution of outbreaks measured in computer simulations of the model on systems of $L=10^7$ sites. As the figure shows, the agreement between the two is excellent.

We can also calculate any moment of the distribution $P(n)$ in closed form using Eq. (7). For example, the mean outbreak size is given by the first derivative of H:

$$\langle n\rangle = H'(1)=\frac{H_0'(1)}{1-2k\phi H_0'(1)}=\frac{p(1+q)}{1-q-2k\phi p(1+q)}, \tag{12}$$

and the variance is given by

$$\langle n^2\rangle-\langle n\rangle^2=H''(1)+H'(1)-[H'(1)]^2=\frac{p[1+3q-3q^2-q^3-p(1-q)(1+q)^2+2k\phi p^2(1+q)^3]}{[1-q-2k\phi p(1+q)]^3}. \tag{13}$$

In the inset of Fig. 2 we show Eq. (12) for various values of ϕ along with numerical results from simulations of the model, and the two are again in good agreement.

The mean outbreak size diverges at the percolation threshold $p=p_c$. This threshold marks the onset of epidemic behavior in the model [6] and occurs at the zero of the denominator of Eq. (12):

$$H_0'(1)=\frac{1}{2k\phi}, \tag{14}$$

giving

$$\phi=\frac{1-q_c}{2kp_c(1+q_c)}=\frac{(1-p_c)^k}{2kp_c[2-(1-p_c)^k]}, \tag{15}$$

in agreement with Ref. [14]. The value of p_c calculated from this expression is shown in the left panel of Fig. 3 for three different values of k.

The denominator of Eq. (12) is analytic at $p=p_c$ and has a nonzero first derivative with respect to p, so that to leading order the divergence in $\langle n\rangle$ varies as $(p_c-p)^{-1}$ as we approach percolation. Defining a critical exponent σ in the conventional fashion $\langle n\rangle \sim (p_c-p)^{-1/\sigma}$, we then have

$$\sigma=1. \tag{16}$$

Near p_c we expect $P(n)$ to behave as

$$P(n)\sim n^{-\tau}e^{-n/n^*} \quad \text{as} \quad n\to\infty. \tag{17}$$

Both the typical outbreak size n^* and the exponent τ are governed by the singularity of $H(z)$ closest to the origin as follows [16]. Equation (17) implies that $H(z)$ can be written in the form

$$H(z)=\sum_{n=0}^{a} P(n)z^n + C\sum_{n=a}^{\infty} n^{-\tau}\exp\left(n\left[\log z-\frac{1}{n^*}\right]\right) + \epsilon(a), \tag{18}$$

where C is a constant and $\epsilon(a)\to 0$ as $a\to\infty$. The first term in this expression is a finite polynomial and therefore has no singularities on the finite plane; the singularity resides in the second term. However, no singularity can be produced as long as the exponent in this second term is negative, and hence the singularity appears when $1/n^*=\log z$. Thus we can calculate n^* from the position z^* of this singularity according to

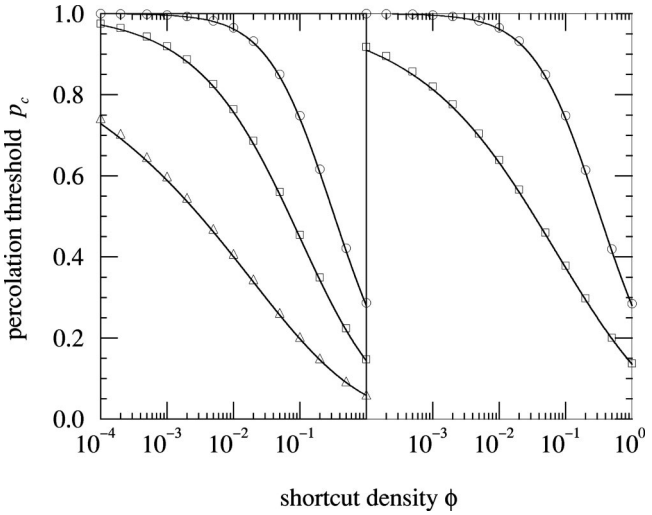

FIG. 3. Numerical results for the percolation threshold as a function of shortcut density ϕ for systems of size $L=10^6$ (points). Left panel: site percolation with $k=1$ (circles), 2 (squares), and 5 (triangles). Right panel: bond percolation with $k=1$ (circles) and 2 (squares). The solid lines are the analytic expressions for the same quantities, Eqs. (15), (34), and (35).

$$n^* = \frac{1}{\log z^*}. \tag{19}$$

Although we do not have a closed-form expression for $H(z)$, it is simple to derive one for its functional inverse $H^{-1}(w)$. Putting $H(z) \to w$ and $z \to H^{-1}(w)$ in Eq. (7) and rearranging we find

$$H^{-1}(w) = H_0^{-1}(w) e^{2k\phi(1-w)}. \tag{20}$$

The singularity in $H(z)$ corresponds to the point w^* at which the derivative of $H^{-1}(w)$ is zero, which gives $2k\phi z^* H_0'(z^*) = 1$, making $z^* = e^{1/n^*}$ a real root of the cubic equation

$$(1-qz)^3 - 2k\phi p z (1-q)^2 (1+qz) = 0. \tag{21}$$

To calculate the value of the exponent τ, we note that as we approach the percolation threshold from below n^* diverges by definition, and hence $z^* \to 1$. The singularity in $H(z)$ at this point is necessarily a finite singularity since $H(1) = \Sigma_n P(n) = 1$. Putting $p = p_c$ and $w = 1 - \epsilon$ in Eq. (20) gives $H^{-1}(1-\epsilon) = 1 + O(\epsilon^2)$, since terms of order ϵ cancel out at the transition point because of Eq. (14). This then implies that

$$H(z) \sim (1-z)^{-\alpha} \quad \text{as} \quad z \to 1, \tag{22}$$

with

$$\alpha = \tfrac{1}{2}. \tag{23}$$

The exponent α can also be calculated from Eq. (18) thus:

$$\begin{aligned} \alpha &= \lim_{z\to 1}\left[1 + (z-1)\frac{H''(z)}{H'(z)}\right] \\ &= \lim_{a\to\infty}\lim_{z\to 1}\left[\frac{1}{z} + \frac{z-1}{z}\frac{\sum_{n=a}^{\infty} n^{2-\tau} z^{n-1}}{\sum_{n=a}^{\infty} n^{1-\tau} z^{n-1}}\right] \\ &= \lim_{a\to\infty}\lim_{z\to 1}\left[\frac{1}{z} + \frac{1-z}{z\log z}\frac{\Gamma(3-\tau, -a\log z)}{\Gamma(2-\tau, -a\log z)}\right], \end{aligned} \tag{24}$$

where we replace the sums with integrals when a is large, and $\Gamma(\nu,\mu)$ is the incomplete Γ function. Taking the limits in the order specified and rearranging for τ, we then get

$$\tau = \alpha + 1 = \frac{3}{2} \tag{25}$$

for the outbreak size exponent. A power-law fit to the simulation data for $P(n)$ shown in Fig. 2 gives $\tau = 1.501 \pm 0.001$, in good agreement with this result.

Turning now to the case of $p > p_c$, we can use the same techniques to study the sizes of epidemics in the model. Above p_c there is a giant component (epidemic) of connected vertices of size $O(L)$, along with a large number of smaller clusters whose distribution falls off exponentially with cluster size. To account for the giant component, we redefine $P(n)$ to be the probability that a randomly chosen site belongs to a cluster of size n which is not part of the giant component. This means that the probabilities $P(n)$ now sum not to 1 as they did for $p < p_c$, but to the probability that a random site is not a member of the giant component. Thus, if we define the generating function $H(z)$ according to Eq. (1) again, the volume of the giant component is $x = 1 - H(1)$.

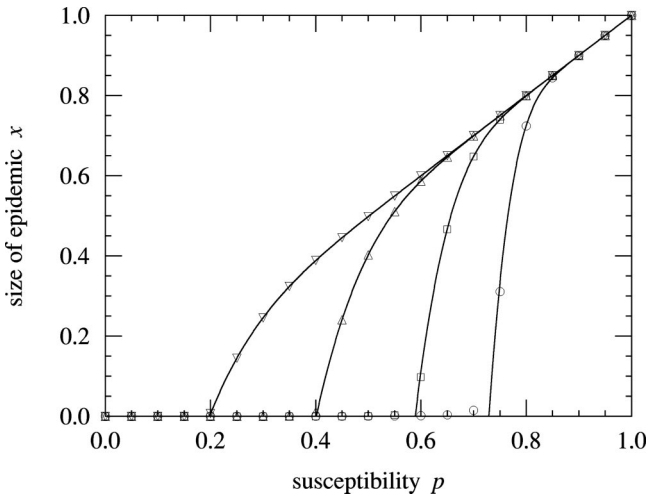

FIG. 4. The average size of an epidemic (giant component) as a function of the susceptibility p in the site percolation model for $k = 5$ and $\phi = 10^{-4}$ (circles), 10^{-3} (squares), 10^{-2} (upward-pointing triangles), and 10^{-1} (downward pointing triangles). The points are results from computer simulations on systems of $L = 10^6$ sites and the solid lines are the exact solution.

Figure 1 and the corresponding Eq. (7) apply to our new $H(z)$ just as before. Setting $z = 1$ gives

$$x = 1 - H_0(e^{-2k\phi x}). \tag{26}$$

This equation has two solutions, one with $x = 0$ for all p and one for which x is in general nonzero. Below the percolation transition the latter is unphysical with $x < 0$, and at the transition the two coincide at $x = 0$. A bifurcation takes place when the derivative of the right-hand side is 1, giving the same result for p_c as Eq. (15). In Fig. 4 we show the values of x as a function of p calculated from numerical solution of Eq. (26) along with simulation results for the size of the largest cluster in systems of $L = 10^6$ sites. Once again the two are in good agreement.

Although the nontrivial root of Eq. (26) appears not to have a closed-form solution, we can calculate it in the vicinity of p_c by linearizing about the bifurcation. If $p = p_c + \delta p$ then to leading order the nontrivial solution is

$$x = \frac{2}{1 + 2k\phi H_0''(1)} \left.\frac{\partial H_0'(1)}{\partial p}\right|_{p=p_c} \delta p. \tag{27}$$

Defining a critical exponent β above the percolation transition in the conventional fashion,

$$x \sim (p - p_c)^{\beta}, \tag{28}$$

we see that $\beta = 1$, since Eq. (27) is linear in δp.

The values $\sigma=1$, $\tau=\frac{3}{2}$, and $\beta=1$ of the critical exponents put the small-world percolation problem in the same universality class as percolation on a Bethe lattice [17], which seems reasonable since the effective dimension of the small-world model in the limit of large system size is infinite [6], just as it is for the Bethe lattice.

We close our analysis of the site percolation problem by noting that Eq. (7) is similar in structure to the equation $H(z)=ze^{H(z)}$ for the generating function of the set of rooted, labeled trees. This leads us to conjecture that it may be possible to find a closed-form expression for the coefficients of the generating function $H(z)$ using the Lagrange inversion formula [18].

III. BOND PERCOLATION

Turning to bond percolation, we can apply the same formalism as above with only two modifications. First, the probability $P_0(n)$ that a site belongs to a local cluster of size n is different for bond percolation and consequently so is $H_0(z)$ [Eq. (6)]. For the case $k=1$

$$P_0(n)=np^{n-1}(1-p)^2, \tag{29}$$

where p is now the bond occupation probability. (Note that this is not the same as the probability of a shortcut existing between two nodes. Rather, it is the probability that a given bond—whether a local one or a shortcut—will transmit the disease in question.) This expression is the same as Eq. (3) for the site percolation case except that $P_0(0)$ is now zero and $P_0(n\geq 1)$ contains one less factor of p. $H_0(z)$ for $k=1$ is

$$H_0(z)=z\frac{(1-p)^2}{(1-pz)^2}. \tag{30}$$

For $k>1$, calculating $P_0(n)$ is considerably more complex, and in fact it is not clear whether a closed-form solution exists. However, it is possible to write down the form of $H_0(z)$ directly using the method given in Ref. [14]. For $k=2$, for instance,

$$H_0(z)=\frac{z(1-p)^4(1-2pz+p^3(1-z)z+p^2z^2)}{1-4pz+p^5(2-3z)z^2-p^6(1-z)z^2+p^4z^2(1+3z)+p^2z(4+3z)-p^3z(1+5z+z^2)}. \tag{31}$$

The second modification to the method is that in order to connect two local clusters a shortcut now must not only be present (which happens with probability ϕ) but must also be occupied (which happens with probability p). This means that every former occurrence of ϕ is replaced with ϕp. The rest of the analysis follows through as before and we find that below the percolation transition $H(z)$ satisfies the recurrence relation

$$H(z)=H_0(ze^{2k\phi p[H(z)-1]}), \tag{32}$$

with H_0 as above. Thus, for example, the mean outbreak size is now

$$\langle n\rangle=H'(1)=\frac{H_0'(1)}{1-2k\phi pH_0'(1)}, \tag{33}$$

and the percolation transition occurs at $2k\phi pH_0'(1)=1$, which gives

$$\phi=\frac{1-p_c}{2p_c(1+p_c)} \tag{34}$$

for $k=1$ and

$$\phi=\frac{(1-p_c)^3(1-p_c+p_c^2)}{4p_c(1+3p_c^2-3p_c^3-2p_c^4+5p_c^5-2p_c^6)} \tag{35}$$

for $k=2$. As in the site percolation case, the critical exponents are $\sigma=1$, $\tau=\frac{3}{2}$, and $\beta=1$. In the right panel of Fig. 3 we show curves of p_c as a function of ϕ for the bond percolation model for $k=1$ and $k=2$, along with numerical results for the same quantities. The agreement between the exact solution and the simulation results is good.

IV. SIMULTANEOUS SITE AND BOND PERCOLATION

We can also apply our method to the case of simultaneous site and bond percolation, by replacing $P_0(n)$ with the appropriate distribution of local cluster sizes and making the replacement $\phi\to\phi p_{\text{bond}}$ as above. The developments are simple for the case $k=1$ but the combinatorics become tedious for larger k and so we leave these calculations to the interested (and ambitious) reader.

V. CONCLUSION

To conclude, we have studied the site and bond percolation problems in the Watts-Strogatz small-world model as a simple model of the spread of disease. Using a generating function method we have calculated exactly the position of the percolation transition at which epidemics first appear, the values of the critical exponents describing this transition, and the sizes of disease outbreaks both above and below the transition. We have confirmed our results with extensive computer simulations of disease spread in small-world networks.

Finally, we would like to point out that the method described here can in principle be extended to small-world networks built on underlying lattices of higher dimensions [2,6]. Only the generating function for the local clusters $H_0(z)$ needs to be recalculated, although this is no trivial task; such

a calculation for a square lattice with $k=1$ would be equivalent to a solution of the normal site percolation problem on such a lattice, something which has not yet been achieved. Even without a knowledge of $H_0(z)$, however, it is possible to deduce some results. For example, we believe that the critical exponents will take the values $\sigma=1$, $\tau=\frac{3}{2}$, $\beta=1$, just as in the one-dimensional case, for the same reasons. It would be possible to test this conjecture numerically.

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