Epidemics, disorder, and percolation

L. M. Sander, * C. P. Warren

Michigan Center for Theoretical Physics, University of Michigan, Ann Arbor, Michigan - 48109, U.S.A.

I. M. Sokolov

Institut für Physik, Humboldt-Universität zu Berlin, Germany Invalidenstr. 110, 10115 Berlin

Abstract

Spatial models for spread of an epidemic may be mapped onto bond percolation. We point out that with disorder in the strength of contacts between individuals patchiness in the spread of the epidemic is very likely, and the criterion for epidemic outbreak depends strongly on the disorder because the critical region of the corresponding percolation model is broadened. In some networks the percolation threshold is zero if another kind of disorder is present, namely divergent fluctuations in the number of contacts. We give an example, a network with a well defined geography, where this is not necessarily so, and discuss whether real infection networks are likely to have this property.

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1 Introduction

Models for the spread of epidemics are an interesting application of non-equilibrium statistical physics. In this paper we discuss two aspects of this sort of study in cases when disorder is present. First we generalize the well-known [1] application of percolation theory to epidemics for which variation in the strength of coupling. Second, we consider the contact networks which carry epidemics. If there is large variation in the number of contacts between

* Corresponding author
members of the population, then the network structure can lead to a percolation threshold of zero, for example if the distribution of contacts is a suitable power law. We show an example of a network which seems to have \( p_c \neq 0 \) for the same power-law distribution, and which might have some relevance to real infections.

2 SIR epidemics in an inhomogeneous population

The SIR (Susceptible, Infected, Recovered) model is a classic way to think about the spread of epidemic infections [3]. It is based on the idea that in many epidemics an individual catches a disease, infects others at rate \( x \) for a certain period, \( \tau \), and then recovers or dies, and is thus removed from the infection network. A mean-field version of this model is given by the SIR equations[3] for a perfectly mixed population:

\[
dS/dt = -xSI/N, \quad dI/dt = xSI/N - I/\tau
\] (1)

Here \( N \) is the size of the population, and we normalize \( x \) in this way so that we can think about the infection rate per contact. Clearly, we have an outbreak if \( R_o = xS(0)\tau/N > 1 \).

We can think of Eq. (1) as describing dynamics on a fully conjugated graph – every node sees every other equally. In this paper we will consider a model on a lattice, where each individual only sees its neighbors. Each site of the lattice is initially occupied by a susceptible (S) individual. One lattice site is initially infected, \( I \). Any S can be infected by \( I \)'s which are nearest neighbors. The probability of infection per unit time along the \( i^{th} \) bond, \( x_i \), is chosen from a distribution \( f(x) \). For a case near the threshold for the epidemic (see below) the pattern of infection is shown in Figure 1. The similarity of this pattern to a percolation cluster is very clear.

The existence of a large epidemic depends on the parameters just as in the mean-field case. For fixed \( x_i \), if the recovery time, \( \tau \), is too small the epidemic will die out, and if \( \tau \) is large enough it will persist. A critical value, \( \tau_c \), marks the threshold. However, even for large \( \tau \) there is a finite probability that the epidemic dies. We define a spanning probability, \( M(\tau) \), the probability that an infection started from a single site in the middle of a large lattice reaches the edge. \( M \) grows rapidly for \( \tau > \tau_c \). In Figure 2 we show \( M \) for a 200 x 200 lattice as a function of \( \tau \) for two different choices of \( f(x) \), one rectangular, and one strongly asymmetric.

This model is not obviously related to percolation theory. For a very narrow distribution, \( f(x) = \delta(x - x_o) \) Grassberger [1] showed that we do have perco-
Fig. 1. Pattern of recovered after the epidemic has died out on a 256x256 lattice. Recovered sites are gray, and uninfected black.

Fig. 2. $M(\tau)$ for a triangular lattice, $\triangle$, and a square lattice, $\square$. Solid lines: $f(x)$ from Eq. (3) $x_{max} = 0.003$. Dotted lines: Eq. (4) with $x_{min} = e^{-15}$.

lation as follows: the probability to infect a neighbor is $p(\tau) = 1 - (1 - x_o)^\tau$. Thus $p$ is the fraction of bonds completed in a given epidemic, and the infection process is mapped onto bond percolation with $p(\tau)$ playing the role of the percolation probability. For a square lattice in two dimensions, $p_c = 1/2$, so we expect that when $p$ is greater than $1/2$ we will have an epidemic. This gives a critical recovery time: $\tau_c = -\ln 2/\ln(1 - x_o)$. For any other network, $p_c = 1 - (1 - x_o)^{\tau_c}$ determines the threshold for epidemic spread. For arbitrary $f$’s, we can write:

$$p(\tau) = 1 - \int (1 - x)^\tau f(x)dx = p_c.$$  \hspace{1cm} (2)

Thus, for any $f$ we have mapped our infection problem to percolation. The solution of Eq. (2) gives $\tau_c$. The mapping of a dynamic problem onto static percolation is not trivial: different runs for infection spreading in a system with a given realization of $x_i$ give different, but not independent realizations of a percolation problem, i.e. of a set of a completed bonds. In previous work [5] we have verified Eq. (2) by doing a data collapse for various different choices of $f(x)$. After data collapse, the function $M$, expressed as a function of $p$ using Eq. (2) is identical to the mass of the infinite cluster in ordinary percolation theory. All of the results of this work may be applied easily for any network.

In order to understand the effect of the distribution of bond strengths we studied $f$’s which are either weakly or strongly asymmetric. An example of a symmetric $f$ is:

$$f_w(x) = 1/x_{max}, \quad 0 \leq x \leq x_{max}.$$  \hspace{1cm} (3)
In cases like this where \( f(x) \) is concentrated near its mean value, it is easy to show the spread of \( x_i \) does influence the infection propagation very much.

However, the dependence \( p(\tau) \), Eq.(2), is strongly nonlinear and is dominated by the behavior of \( f(x) \) in vicinity of \( x = 0 \): the spread of the infection is controlled by weakest bonds. Thus strongly asymmetric distributions concentrated near zero should be very different from the case discussed above. Consider, for example, power-laws \( f(x) = (\alpha - 1)x^{-\alpha} \) with \( 0 < \alpha < 1 \). In this case \( \int (1 - x)\tau f(x)dx = (\alpha - 1)B(\tau + 1, 1 - \alpha) \), where \( B(x, y) \) is the beta-function. Strongly asymmetric distributions correspond to values of \( \alpha \) in vicinity of 1. Using the fact that for such distributions \( \tau \gg 1 \), we can use the Stirling formula to get in leading order: 

\[
\tau_c \simeq \left[ \Gamma(2 - \alpha)(1 - p_c) \right]^{-1/(1-\alpha)}
\]

which diverges for \( \alpha \rightarrow 1 \).

An even more extreme case is that of functions \( f(x) \) with \( \alpha \geq 1 \). These are not acceptable probability densities since they are not normalizable on \([0,1]\). However, one can truncate the \( f \) in the vicinity of zero to allow normalization. For example:

\[
f_s(x) = C/x \quad x_{\text{min}} \leq x \leq 1; \quad C = 1/|\ln(x_{\text{min}})|.
\]

The two distributions considered in Figure 2 are given by Eq. (4) with \( x_{\text{min}} = e^{-15} \), an extremely asymmetric case, and Eq.(3), the symmetric case.

In Figure 2 we see that the effect of strong asymmetry is to spread out the critical region, i.e. the region where \( M \) is not close to either 1 or 0. This is the parameter region where the epidemic pattern is patchy, as in Figure 1. For a strongly asymmetric \( f \) the patchy pattern corresponds to a large range of the parameter \( \tau \). We recall from percolation theory that the regime where \( M \) is neither very small nor very close to 1 is where the fractal nature of the percolation cluster at \( p_c \) persists over large length scales. This could have implications for practical epidemiology: if the actual distribution of \( x_i \) is very asymmetric, we would be very likely to observe patchiness. For such distributions mean-field theory is very inaccurate \([5]\) and sampling of populations must be done with care because of the spatial correlations.

3 The network of contacts: power-law distributions

In the previous section we considered networks for which the number of contacts between individuals does not vary much, though we supposed that the strength of such contacts varied a good deal. However, there is data to suggest \([6]\) that some infection networks have a wide distribution of the number of contacts, so that the bond number, \( k \), is drawn from a distribution of the
form \( P(k) \propto 1/k^\alpha \) with \( 2 < \alpha < 3 \). In this case the mean value of the number of contacts, \( \langle k \rangle \), exists, but the fluctuation, \( \langle k^2 \rangle \), diverges.

We have seen that it is sufficient to consider percolation on such networks to understand SIR epidemics. This problem has been discussed a good deal [7,8,9]. There is general agreement that \( p_c \) is zero for many cases. The graphs considered by these authors are essentially random graphs with a given degree distribution. Thus the model has a kind of random mixing.

The random graphs dominate the the ensemble of all graphs with a given \( P(k) \). The study of this ensemble was introduced by Molloy and Reed [2] and a criterion for the formation of a giant component was derived. Callaway, et. al [8] and Cohen, et al. [7] express a related result for percolation: \( p_c(\langle k^2 \rangle / \langle k \rangle^2 - 2) = 1 \) Thus if \( \langle k^2 \rangle \) diverges we expect \( p_c = 0 \). We can see this by starting at a random node on the graph so that there are \( k_0 \) bonds of which \( pk_0 \) are active. If neighbors are chosen at random, the probability for the number of bonds on the next neighbor is \( p^2k_1P(k_1)/\langle k \rangle \). The factor of \( k_1 \) comes from the fact that highly bonded sites are more likely to have a bond with the first site. Now the number of infected increases by a factor \( \langle k_1 \rangle / \langle k_0 \rangle = p \langle k^2 \rangle / \langle k \rangle^2 \) at each such step. If \( \langle k^2 \rangle \) is finite, then this can be made less than unity for \( p \) small, and the infection will die out. If \( \langle k^2 \rangle \) is infinite, then \( p \) must be 0.

Now consider a model which has the same \( P(k) \) but has geography [10,11]. We assume that individuals infect their neighbors, but that the number of infections is drawn from \( P \). We construct this by taking each point on a \( d \)-dimensional lattice with probability \( p \) and connecting them all other points within a \( d \)-sphere of volume \( k \), centered on the point. In 2 dimensions we surround each point with a disk of radius of roughly \( R = [k/\pi]^{1/2} \). The probability distribution of \( R \) is given by \( P(R) = P(k)dk/dR \propto R^{-(dn-d+1)} \). Our model is a variant of an old two-dimensional circle model of continuum percolation [12] with variable radius of circles. See Figure 3. This is a small part of the Molloy-Reed ensemble, but we suggest that it may be relevant for real-world epidemics: sometimes infection is primarily local.

Note that a site can have the bonds it generates itself (‘proper’ bonds) and those from other disks. We show that the total number of bonds per node has the same power-law behavior as the proper bonds if \( \alpha > 2 \) by calculating the mean number of bonds connecting a node \( i \) to nodes \( j \) whose center is a distance \( r > R_i \) away. The probability to find \( R_j \) larger than \( r \) is \( \int_r^\infty P(r')dr' \). Thus, the mean number of nodes connected to \( i \) from outside is \( k_{out} \propto \int_{R_i}^\infty drr^{d-1} \int_r^\infty P(R_j)dR_j \). Thus \( k_{out} \propto R_i^{-d(\alpha-2)} \) which tends to zero for large \( R_i \) as long as \( \alpha > 2 \). This means that the probability distribution of the number of the ‘proper’ bonds and the actual number of the bonds of a node is the same for \( k \) large, and that bonds from outside are rare.
For \( d = 1 \) it is easy to see that the system never percolates, because for any \( p < 1 \) there will eventually be a gap of length \( \Delta \) in the chain of connected disks. Consider disks with radii between \( k \) and \( k + dk \); the concentration of their centers is \( pP(k)dk \). The probability that the gap is not covered by one of these disks is equal to the probability that none of their centers are found in the interval of length \( 2k + \Delta \) (in units of the lattice constant) centered at the gap, \( 1 - pP(k)(2k + \Delta)dk \approx \exp[-pP(k)(2k + \Delta)dk] \) The overall probability that no disk overlaps the gap is the product over \( k \):

\[
\exp\left(-\int_{0}^{\infty} pP(k)(2k + \Delta)dk\right) = \exp(-2p\langle k \rangle - p\Delta),
\]

which is finite as long as \( \langle k \rangle \) exists.

In 2 dimensions we did simulations to find \( p_c \). Disks were randomly added to an \( L \times L \) lattice at different sites until a cluster spanned the lattice. Figure 4 shows the average percolation threshold \( p_c(L) \) as a function of \( 1/L \). It appears from the plot that there is a finite percolation threshold for not just \( \alpha > 3 \) but \( 2 < \alpha < 3 \) as well, the region of interest for real world epidemics. For \( \alpha < 2 \), the results are consistent with \( p_c = 0 \) and seem to scale according as \( L^{2(\alpha-2)} \) for sufficiently large \( L \). We have shown [10] that this scaling is related to the presence of giant disks that span the whole sample. These occur with finite probability for \( \alpha < 2 \).

We can try to understand the results in Figure 4 by calculating \( \langle k_1 \rangle \) as above. Most of the bonds emanating from the center of disk point within it. These have degrees drawn from the distribution \( P(k_1) \), not \( k_1P(k_1) \). Thus, for the typical behavior we \( \langle k_1 \rangle = \langle k \rangle \) for \( \alpha > 2 \). However, there are rare events which correspond to the overlap of large disks from outside with the center of the starting disk. These give a large contribution. In fact, in 2
Fig. 4. $p_c$ on an $L \times L$ lattice as a function of $1/L$. From top to bottom, the data are for $\alpha = 2.5, 2.3, 2.05$ (dotted lines), 2, (solid line), and 1.95, 1.9, and 1.7 (dashed lines). The solid straight lines have slope $2(\alpha - 2)$.

dimensions for a disk of size $R_0$ we add up the contributions of outside disks of radius $R_1$:

$$< k_1 > \propto \int_{R_0}^{\infty} r dr \int_r^{\infty} R_1^2 P(R_1) dR_1 = \int_{R_0}^{\infty} dr r^{-(2\alpha - 5)}$$  \hspace{1cm} (6)

This diverges for $\alpha < 3$. Note that this is an overestimate since have not attempted to exclude already infected sites.

We have a confusing situation: the typical behavior is that the cluster of disks is finite in extent, but the average mass added diverges. There is a rigorous theorem in the study of continuum percolation which treats exactly this situation [12]. It states that if $< k^{2-1/d} >$ is finite, but $< k^2 >$ diverges (in 2 dimensions $2.5 < \alpha < 3$), then there is a $p_c$ such that for $p < p_c$ with probability unity all clusters are finite, but the expected number of points of the largest cluster is infinite. (The probability to span a large lattice is not addressed by this theorem.) We interpret this odd situation, at least for $\alpha > 2.5$ as a form of intermittancy. Typical epidemics are finite for small $p$, but huge, rare events occur. It is interesting to ask the physical question of what we would expect to observe in this case. We claim (perhaps too hopefully) that the situation is best represented by the computer simulations. Figure 4 shows that at $\alpha = 2.5$ there is quite solid evidence for a non-zero $p_c$, and this seems to extend down to $\alpha = 2$. (Note that the theorem does not exclude this possibility).
In summary we have a counterexample to the prevailing notion that $p_c$ must be zero if $< k^2 >$ is infinite, certainly in one dimension. In 2 dimensions the situation is complex, and will take more work to fully sort out.

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