Firewalls, Disorder, and Percolation in Epidemics

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We consider a spatial model related to bond percolation for the spread of a disease that includes variation in the susceptibility to infection. We work on a lattice with random bond strengths and show that with strong disorder, i.e., a wide range of variation of susceptibility, patchiness in the spread of the epidemic is very likely, and the criterion for epidemic outbreak depends strongly on the disorder. These results are qualitatively different from those of standard models in epidemiology, but correspond to real effects.

The most commonly used models for the spread of an epidemic assume perfect mixing: i.e., all individuals are able to infect all others. In an inhomogeneous population the susceptibility to infection is replaced by its average. To account for spatial effects many workers use reaction-diffusion equations to describe traveling waves of infection. In both cases important phenomena are not reproduced by the models. For example, it is common experience that in plant diseases islands of susceptible individuals can be protected by a band of immune ones, a ‘firewall’. A map of the infected areas looks patchy. Even in human diseases such as AIDS these effects seem to be important. Such effects do not occur in perfect mixing or reaction-diffusion theories in which susceptibilities are replaced by averages. In this work we give a simple model for these phenomena using ideas taken from percolation theory. We show that for inhomogeneous populations, the variability of susceptibility to infection among individuals gives rise to qualitatively new effects.

Percolation theory is a natural way to think about populations which are not well mixed. Consider a spatially random distribution of susceptible individuals which occur with probability $p$. A (site) percolation model for an epidemic is a lattice algorithm where, with probability $p$, a site is occupied and susceptible sites infect their susceptible neighbors. For diseases where some agents travel long distances, ‘small-world’ theory extends the percolation model. In this approach a few sites are considered to be neighbors of distant sites.

Percolation accounts for the existence of firewalls and islands because near the percolation threshold only part of the lattice belongs to the spanning cluster. However, a close look makes such an application suspect. Islands and firewalls occur only in a rather narrow transition region of critical probability, $p_c$. In nature it would be unlikely that a fine-tuning of parameters to be near $p_c$ would occur. This kind of difficulty always plagues attempts to apply theories of critical phenomena in the natural world. Here, we show that introducing disorder in the susceptibility to infection solves this problem in an elegant way because it broadens the transition region for the outbreak, so that a generic epidemic could produce islands and firewalls.

There is a further unexpected result of this study. By definition, an epidemic starts when a sick individual infects more than one other before she recovers, i.e., $R_o \geq 1$, where $R_o$, the reproduction number, is the mean number of infections produced by a site. The usual expression for $R_o$ is $xS(0)\tau$ where $x$ is the probability per unit time to infect neighbors, $S(0)$ the number which can be infected, and $\tau$ the time to recovery. We will show that disorder can change this formula by a large amount.

Our model is of the SIR (Susceptible, Infected, Recovered) type. We use bond percolation: each site of a square lattice is occupied by an individual which can be infected by neighbors connected by bonds (four in our case). To account for variability the probability per unit time of infection along a given bond is chosen from a distribution $f(x)$. After exactly $\tau$ time steps any infected site recovers. We start with one $I$ in the middle of the lattice and the rest $S$. We say that we have an epidemic when the $I$ and $R$ sites span a large lattice, i.e., reach the edges. A snapshot is shown in Figure 1. 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 of $\tau$ plays the role of $p_c$. In Figure 2 we show the spanning probability, $M$, for a 256 x 256 lattice as a function of $\tau$ for two different choices of $f(x)$. $M$ is the probability to span starting from a single site. We should note that $M$ is the same as the mass of the infinite cluster in percolation theory i.e., the fraction of sites which belong to the spanning cluster. This is true because the probability to span starting at any given site is the probability that the site is a member of the infinite cluster.

We have studied two classes of $f$’s which we call weak and strong disorder. An example of weak disorder is:

$$f_w(x) = 1/x_{\text{max}}, \quad 0 \leq x \leq x_{\text{max}}.$$  \hspace{1cm} (1)

Other functions with a rather narrow range of $x$ give
similar effects, and the results do not differ very much from the case with no disorder [1].

Strong disorder refers to broad distribution such as:

$$f_s(x) = C/x \quad x_{min} \leq x \leq 1; \quad C = 1/|\ln(x_{min})| \quad (2)$$

Any strongly skewed distribution should give similar results to those quoted here. Distribution functions of this type were studied [6] in random resistor networks, and are known, in that context, to give different behavior from the ordered case.

The black portion of Figure 1 corresponds to sites that are protected by the weak bonds at the edge of the infected cluster, the firewall. Effects of this type occur only in the transition region of Figure 2. The transition region is very broad for strong disorder. Firewalls and large islands of uninfected sites arise purely from the statistics of the problem, as in any percolation problem.

Ordinary bond percolation has some bonds present and some absent, which appears to be quite different from our approach. If $x$ takes on a single value, the relationship was pointed out by Grassberger [6]: the probability to infect a neighbor before recovery is $p = 1 - (1 - x)^\tau$. Thus $p$ is the fraction of bonds completed in a given epidemic.

If we have disorder we write:

$$p = 1 - \int (1 - x)^\tau f(x)dx. \quad (3)$$

For a square lattice $p_c = 1/2$, so we expect that when $p$ in Equation 3 is greater than 1/2 we will have an epidemic. We verify this in the inset to Figure 2 where plot $M$ against $p$ for various choices of $f(x)$ and compare them to ordinary bond percolation on the same lattice.

We can discuss the threshold for outbreak of an epidemic by using the usual SIR equations [1] for the perfect mixing case:

$$dS/dt = -xSI, \quad dI/dt = xSI - I/\tau \quad (4)$$

Here there is a rate of recovery $1/\tau$ rather than a fixed recovery time. However this is qualitatively the same. Clearly, we have an outbreak if $R_o = xS(0)\tau > 1$.

If we convert Equation 4 into a spatial model by adding diffusion terms then the criterion for outbreak is of the same form [1], where $S(0)$ now refers to the initial number of susceptibles which can be infected by a single infected site, a number of order unity. For the Grassberger model (or weak disorder) we have a similar looking result: we put $p = 1 - (1 - x)^\tau$ = $p_c$ = 1/2. For small $x$, $R_o$ = $x\tau$ = $O(1)$.

Strong disorder is quite different. We can estimate the integral in Eq. 3 using the $f_s(x)$ of Eq. 2 for $x_{min} << 1$. We get

$$\int (1 - x)^\tau f_s(x)dx \approx [-\Gamma - \ln(x_{min})]/[\ln(x_{min})] \quad (5)$$

where $\Gamma$ is Euler’s constant. Using this estimate we find $\tau \approx 0.56/\sqrt{x_{min}}$. To estimate $R_o$ we replace $x$ by its average, $\bar{x}$. The naive estimate for the reproduction number at the threshold for outbreak gives:

$$R_o = \tau_c \bar{x} \propto 1/[(\sqrt{x_{min}} \ln(x_{min})]. \quad (6)$$

This estimate for $R_o$ can be arbitrarily large. The effective $R_o$ for a disordered lattice is very much smaller than $\tau_c \bar{x}$.

This statement may seem reminiscent of the well-known fact [2] that for diseases such as AIDS the naive formula for $R_o$ does not work if there are very active agents, because active agents have an outsize effect in spreading the disease. However, the results are, in fact, in opposite directions. Here, the spatial correlations between strong bonds make them less effective than they might seem to be. A clump of very infective bonds does not do as much damage as we might suspect since the infected sites are sharing the same victims. In the transition region there are always bottlenecks to the spread of the infections, and thus random spatial correlations are always important.

We can understand the broadening of the transition region due to disorder in the same way. Using the expressions in Eq. 3 and Eq. 4 we find $\delta \tau/\tau_c = |\ln(x_{min})|\delta p$. We can define the transition region, $\delta p$, as the range over which $M$ increases from, say, 0.1 to 0.9; it is of order 0.05. We see that $\delta \tau/\tau_c$ is large as $x_{min} \rightarrow 0$.

In epidemics of human diseases some (usually a small number) of individuals travel long distances. We use a small-world lattice to account for these contacts by adding a small fraction, $\phi$, of ‘long’ bonds connecting randomly chosen sites [6]. The result is shown in Figure 3. Even a very small $\phi$ breaks up the large uninfected regions. The percolation threshold is lowered, and below the old $p_c$ there are many small regions of epidemic connected by long bonds. However, the effects of disorder remain. Long distance contacts are not the same as perfect mixing; firewalls still occur over a substantial range of parameters.

To understand these results we extend the work of Newman and Watts [10] to our case. The infected islands of Figure 3 can be considered to be nodes of a random graph whose edges are the long bonds. The graph percolates if there are two islands per long bond [11]; this occurs for some $p < p_c$.

We need to count the nodes of the random graph. To do this we use the average island size defined as

$$\bar{\pi} = \sum s^2 n_s / \sum s n_s$$

where $n_s$ is the number of clusters of size $s$. This is the correct average because it counts the probability that a given site (the end of a long bond) belongs to a cluster [6]. Scaling theory gives

$$\bar{\pi} = K[1-p_c]^{-\gamma}$$

where $K$ is a constant, and $\gamma$ is a critical exponent equal to 43/18 = 2.39 for our case. The number of islands is
\[ N_i = N/\Pi, \text{ where } N \text{ is the total number of sites.} \]
The number of long bonds that connect two sites that are parts of islands is \( B = N\phi[1-(1-p)^4]^{2}. \) Setting \( 2N_i = B \) gives a criterion for percolation on a small world lattice with one adjustable parameter, \( K \):

\[ \phi = K(p_c - p)\gamma(1 - (1 - p)^4)^{-2} \tag{7} \]

In Figure 4 we show the depression of the percolation threshold as a function of \( \phi \). We exhibit data as a function of \( p \) rather than \( \tau \). They are related by Eq. (3). The threshold is obtained by requiring that an epidemic infects 20\% of the lattice sites. (In this case, with long bonds, the notion of spanning loses its meaning). We also show a fit to the formula in Eq. (7) with \( \gamma \) and \( K \) as adjustable parameters. The best fit gives \( \gamma = 2.40 \), remarkably close to the theoretical value quoted above. However, as we see in Figure 3 there are still islands surrounded by a firewall, and all of the above considerations carry over in the strongly disordered case. In particular, the size of the transition region in terms of \( \tau \) is expanded \( 12 \).

Our model for percolation with strong disorder illustrates two effects: there is a mechanism which produces patches of uninfected but susceptible individuals without fine tuning of parameters. And for this case \( R_c \) depends on the disorder. Whether strong disorder is a valid description of nature needs to be determined by consideration of real epidemics. It is known, for example, that variability in susceptibility increases when epidemics recur. Our model may apply best to that case.

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FIG. 2. The probability, $M$, that an epidemic started from a point spans the lattice as a function of infection time $\tau$ for two different bond distributions. Strong disorder broadens the transition region in $\tau$. The transition region (e.g., the range $0.1 < M < 0.9$) is about 12 times larger for strong disorder than for weak. For $f_w$, we use $x_{max} = 0.003$, $f_s$ uses $x_{min} = e^{-15}$. Averages are over 2000 simulations on a 200x200 lattice. [Inset] Data collapse of $M$ as a function of $p$, compared with bond percolation. The bond distributions used are: (o) bond percolation, (+) $f_s$ with $x_{min} = e^{-15}$, (x) $f_s$ with $x_{min} = e^{-7}$, (*) $f_w$ with $x_{max} = .001$, and (A) $f_w$ with $x_{max} = .01$.

FIG. 3. Snapshot of the final cluster of recovered on a 256x256 small-world lattice with periodic boundary conditions and $\phi = 0.01$.

FIG. 4. Dependence of the percolation threshold on $\phi$ (o) compared with the scaling theory prediction on a 200x200 lattice.