A Brief Introduction to Percolation Theory

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Abstract

Consider a cube of water-permeable material. What is the probability that that if water is poured on top of the cube it may drain all the way through the cube and out the opposite face? Initially developed by Paul Flory and Walter Stockmayer in 1944, percolation theory attempts to answer such questions by rephrasing them in terms of vertices (sites) and edges (bonds) of graphs and examining the connectedness of such graphs. The connectedness of these graphs—in the infinite case—is determined by a threshold probability, p_c, describing whether the water may pass through each site or bond. This essay will introduce the ideas of site and bond percolation as well as the notion of clusters and critical (threshold) probabilities. We will also analyse the one dimensional case to garner a basic understanding before exploring higher dimensional cases. After discussing the concepts of percolation theory, we will move on and look at the many applications of the theory discussed in the earlier parts of the essay.

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1 Introduction

Let us consider the example from the abstract of water filtering through a porus medium, but this time in two dimensions. How do we model this? One might imagine that the medium consists of many particles arranged (for simplicity) in an $n \times n$ square lattice and linked to each of their nearest neighbours. Clearly, this is the lattice on $\mathbb{Z}^2$. To set up the problem, each of the particles will be expressed as a vertex in a graph and each of the links will be an edge connecting two directly adjacent vertices. In the context of percolation theory, a vertex is called a site and an edge is called a bond; these sites and bonds form a graph which we refer to as a network.

So what does percolation actually mean? In order to talk about percolation, we need to think about what makes a percolation problem. If we think about the context we're in, some of the sites in the network will alow the water through and some of them won't. The sites that allow water to pass through are labeled **open** and the sites that don't allow water to pass through are labeled **closed**. This gives us **site** percolation. If we were to consider open and closed bonds instead of sites, then we would have **bond** percolation. We also let these sites or bonds be open with probability p and closed with probability $1 - p$. I will refer to this p as the **percolation probability**.

Now that we've defined site and bond percolation, what's the problem that we're trying to solve? In the case of water being poured on a porus medium, we would like to know whether there is a route that the water could take from the top of the medium to the bottom. This is called an **open path**. We shall model this using site percolation (in fact, all examples in this essay will be using site percolation unless explicitly stated otherwise).

Definition 1.1. Let $N = (V, E)$ be a network, we say that a path in N is **open** if every site in the path is open. [1]

Definition 1.2. Let $N = (V, E)$ be a network and let $A, B \in V$. The sites A, B are **openly connected** if there exists an open path connecting A and B. Throughout this essay, I will interchange the term **openly connected** with **connected**.

Definition 1.3. Let $N = (V, E)$ be a network and let $A, B \in V$. The sites A, B are **openly disconnected** if there does not exist an open path connecting A and B. Throughout this essay, I will intercahnge the term **openly disconnected** with **disconnected**.

Returning to the example, the probability that an open path from the top of the network to the bottom exists depends on both our choices of both p and n. As a result of our context, our value for n should be large—this

[1] This definition is trivially different for bond percolation.

is the case with most percolation models—but we shall use small n for the sake of example and simplicity. Let us now fix n and see what happens as we vary p. Obviously we have two trivial cases, $p = 0$ and $p = 1$, where the network is completely disconnected and completely connected respectively. What about when $p \in (0, 1)$? Let's inspect three different values of p on our network: $p = 0.25$, $p = 0.5$ and $p = 0.75$ as shown in figures 1a, 1b and 1c on page 5.

As one might expect, as p increases, so does the average size of a "cluster" of open sites. Also observe that the overall connectedness of the network increases as p increases. I.e. the probability of having an open path from the top of the netwoork to the bottom increases with p. This is the result that we expect given the context — as we reduce the number of things blocking the way for the water, it's easier for it to pass through the block of our chosen medium. Now let us consider two sites A and B in our example network. As we increase p, it's obvious that the probability of these two sites A and B being connected will increse. The question is: What's the relationship between p and the probability of A and B being connected? It turns out that there's a probability, p^*, such that for some ε close to zero

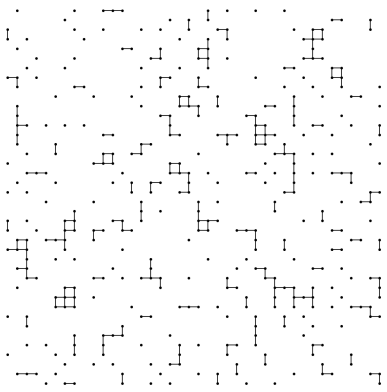
- for $p < p^* - \varepsilon$, the network is probability of A and B being connected is close to 0.
- for $p > p^* + \varepsilon$, the network is probability of A and B being connected is close to 1.

The existence of such a p^* indicates that there must be some threshold where the network transitions from being mostly disconnected to mostly connected. To understand the structure of the network as a whole and not just two points, we have to consider clusters.

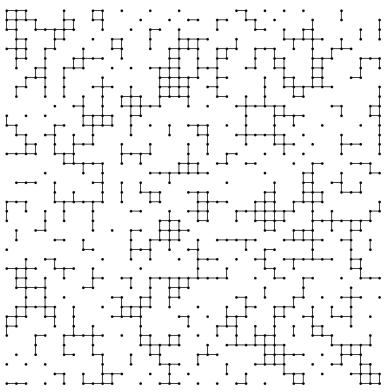
Definition 1.4. Let $N = (V, E)$ be a network and let $C \subseteq V$. We say that C is a cluster if $\forall u, v \in C$ then u and v are openly connected.

Definition 1.5. Let $N = (V, E)$ be a network and let $C \subseteq V$. We say that C is of size s if C has s vertices. We denote this by $|C| = s$. We say that a cluster C is infinite $\iff |C| = \infty$.

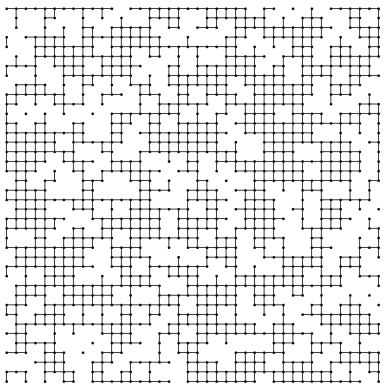
In our example, because we're working in a finite case, there's no single value where this change in structure is obvious — it happens over a range of values. In the infinite case however, the change in structure happens instantaneously. I.e. we have the same as the above but without the epsilons. This change in structure is formally defined as the point at which the network is *guaranteed* to have a cluster of infinite size. The probability p_c that gives rise to this change in structure is referred to as the **critical probability** and is dependent on the structure of the network.



(a) $p = 0.25$



(b) $p = 0.5$



(c) $p = 0.75$

Figure 1: Examples of bond percolation for $p \in (0, 1)$ on a 40×40 network where if a site is open in the model it appears present in the diagram and bonds are only present if they connect two open sites.

1.1 Other network configurations

We have already seen the lattice on $\mathbb{Z}^2$ as an example of one network configuration, but there are many more. To remain within the scope of this essay, we shall only breifly mention some two and three dimensional examples and print their site and bond critical probabilities and a diagram. Before talking about different network configurations, it's important to introduce the following two qualities that we use to describe them.

- A network is considered **regular** if every site in that network has the same number of bonds attached to it.
- The **coordination number** of a regular network is the number of bonds attached at every site. This quantity is denoted using the letter Z. I.e. the lattice on $\mathbb{Z}^2$ has a coordination number of $Z = 4$.

Two dimensional network configurations

Clearly, one two dimensional network configuration is the lattice on $\mathbb{Z}^2$. In context this is referred to as the square lattice. Other regular two dimensional network configurations include, but are not limited to, the Bethe Lattice (Figure 4a), Honeycomb Lattice (Figure 4b), Kagome Lattice (Figure 4c) and the Triangular Lattice (Figure 4d). As one might imagine, each of these configurations has a different (but not necessarily distinct) critical probability. Below is a table showing the critical probabilities for each of the aforementioned network configurations. It should be noted that probabilities marked with a * (star) are exact results.

Configuration	Z	p_c for bond percolation	p_c for site percolation
Bethe ($Z = 3$)	3	0.5	1/3
Honeycomb	3	$1 - 2\sin(\pi/18)$*	0.6962
$\mathbb{Z}^2$ (Square)	4	1/2*	0.5927
Kagome	4	0.522	0.652
Triangular	6	$2\sin(\pi/18)$*	1/2*

Figure 2: Critical probabilities for various configurations of two dimensional networks[Sahimi, 1994, p. 11]

Three dimensional network configurations

It shouldn't be hard to guess that the lattice on $\mathbb{Z}^3$ is a potential configuration for three dimensional networks. We call this configuration the Simple Cubic Lattice (Figure 5b). Similarly to the two dimensional case, there many other regular three dimensional network configurations. These include, but

Configuration	Z	p_c for site percolation	p_c for bond percolation
Diamond	4	0.3886	0.4299
Simple Cubic	6	0.2488	0.3116
BCC	8	0.1795	0.2464
FCC	12	0.198	0.119

Figure 3: Critical probabilities for various configurations of three dimensional networks[Sahimi, 1994, p. 11]

again are not limited to, the Diamond Lattice (Figure 5a), the Body Centered Cubic (BCC) Lattice (Figure 5c) and the Face Centered Cubic (FCC) Lattice (Figure 5d). Notice how none of these results are precise.

2 Analysis of different network configurations

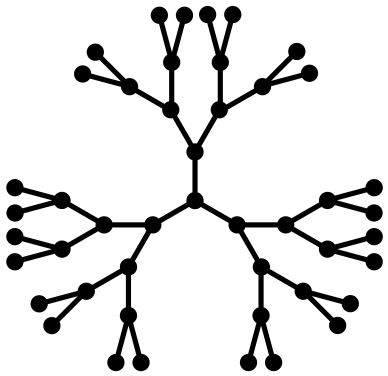
In this section, we will look at a couple of techniques used to analyse different network configurations to determine their critical probabilities.

2.1 The one dimensional case

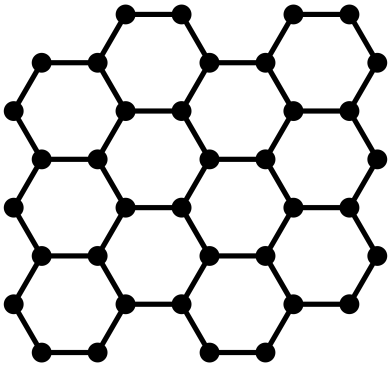
To develop an understanding of how to analyse these networks, we shall consider the one dimensional case where our network is the lattice on $\mathbb{Z}$, or a "chain". If we recall the definition of the critical probability, the value of p_c that we're looking for is intuitively 1 because that's the only way that everything is connected. This is true for both site and bond percolation. In order to more rigorously understand this result, we shall introduce some more machinery. This machinery isn't necessary for the "proof", but it helps with understanding and will allow us to analyse more interesting cases later on. The following line of argument is inspired by Dietrich Stauffer's *Introduction to Percolation Theory* [Dietrich Stauffer, 1991].

If we let each of the sites in our chain be open with probability p, then the probability that any s arbitrary sites are all open is p^s. Now if we wanted to create a cluster in our chain of size s, we would have to have one closed site followed by s open sites followed by a final closed site to cap it off. This means that the probability of any given site being the left end of a cluster of size s is $(1-p)p^s(1-p) = p^s(1-p)^2$.

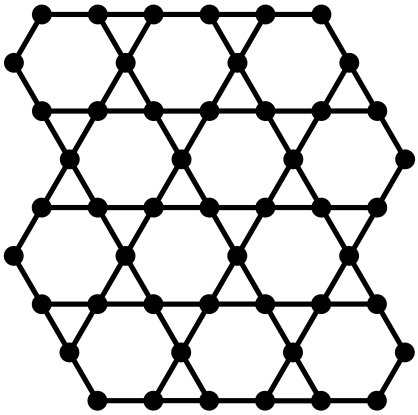
So now how many clusters of size s are there in our chain? If we suppose that the length of the chain is $L >> s$, then we have $Lp^s(1-p)^2$ clusters of size s in our chain of length L (ignoring small errors produced at the right end of the chain). It's practical for us to talk about the number of clusters at any given site in the chain which is $\frac{Lp^s(1-p)^2}{L} = p^s(1-p)^2$ which we denote by n_s and refer to as the **normalised cluster number**. Now it's clear that



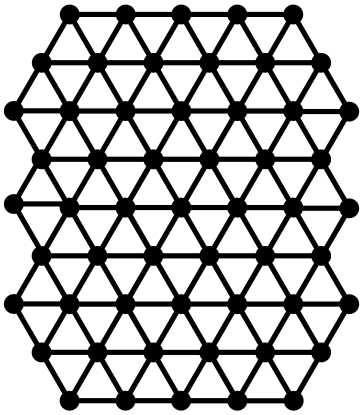
(a) Bethe Lattice



(b) Honeycomb Lattice

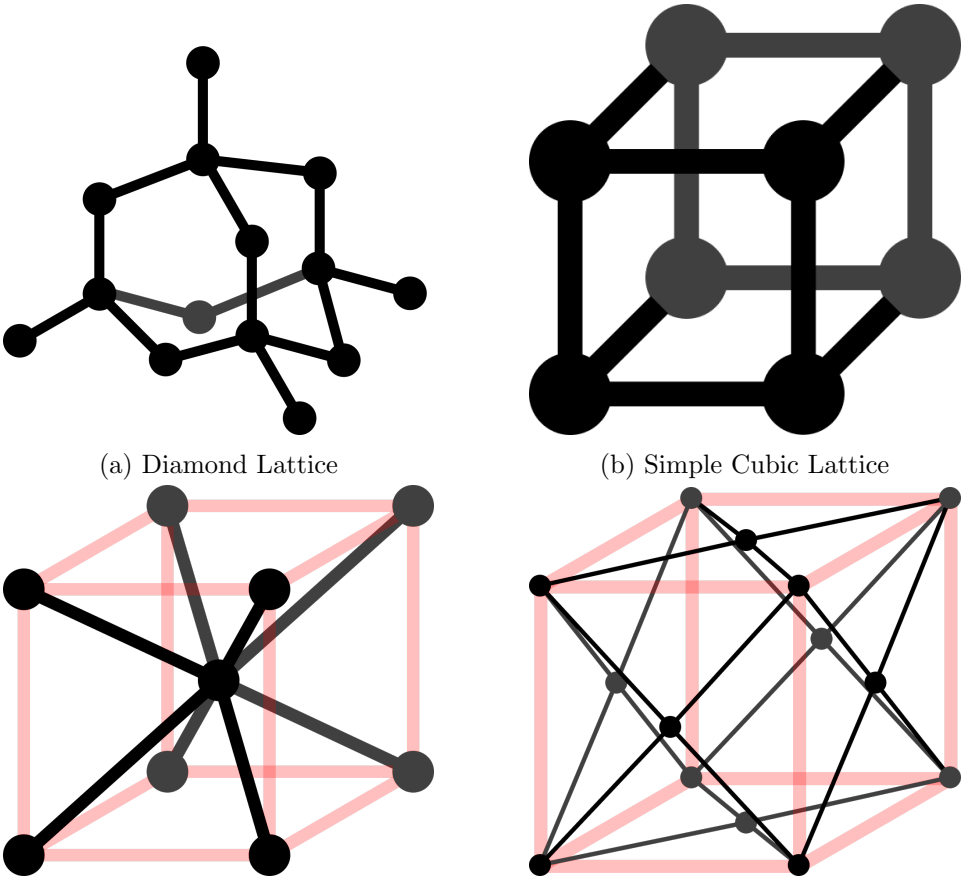


(c) Kagome Lattice



(d) Triangular Lattice

Figure 4: Two dimensional regular network configurations



(a) Diamond Lattice

(b) Simple Cubic Lattice

(c) Body Centered Cubic Lattice

(d) Face Centered Cubic Lattice

Figure 5: Three dimensional regular network configurations. Black and grey lines represent sites and bonds, red lines are present for perspective.

the probability of any site belonging to a cluster of size is is larger than our normalised cluster number by a factor of s yielding $n_s s$. Now we can consider the critical probability for this configuration. We want every site in the chain to be occupied so that there are no holes. If $p = 1$, this is clearly true and the whole chain is one single cluster. For $p < 1$ there will be some holes. For a chain of length L, on average we will have $L(1-p)$ empty sites. As we let $L \to \infty$ for a fixed $p < 1$ we get that $L(1-p) \to \infty$. Therefore there is at least one empty site in the chain so we don't have percolation. Thus $p_c = 1$.

The above results allow us to get some more interesting information about the behaviour of our system. For example, we can deduce the following:

Corollary. *When considering the lattice on $\mathbb{Z}$ with percolation probability $p \in [0, 1)$, the following equality holds:*

$$\sum_s n_s s = p$$

This result comes from the fact that every open site must belong to a cluster of some size s. So summing $n_s s$ over all s must give us p. This equality may also be derived using the definition of n_s and the formula for a geometric series.

Proof.

$$\begin{aligned}
\sum_s n_s s &= \sum_s p^s (1-p)^2 s \\
&= (1-p)^2 \sum_s p \frac{d(p^s)}{dp} \\
&= (1-p)^2 p \frac{d(\sum_s p^s)}{dp} \\
&= (1-p)^2 p \frac{d(p/(1-p))}{dp} \\
&= p
\end{aligned}$$

□

It's worth noting that this equality doesn't hold for $p = 1$, because $n_s = 1^s(1-1)^2 = 0$ so $\sum_s n_s s = 0$.

2.2 Bethe Lattice

Another easy case to analyse is the Bethe lattice as showin in Figure 4a. The Bethe lattice is another configuration that's easy for us to analyse.

When looking at the infinite Bethe lattice, it's clear that we could choose any site to be the root of the lattice just by moving the all the other sites around to make the visual representation clearer. This self-similarity means that we only have to consider one branch of the Bethe lattice and if that percolates then so does the whole structure. So if we assume that some part of the lattice has already been percolated through and look at what's remaining we can see that we're left with a binary tree. In order for percolation to take place here, we need that at least one of these branches is present so we require that $p \geq 1/2$ which gives us our critical probability of $p_c = 1/2$. Applying this same process to the case where $Z = n \in \mathbb{N} \setminus \{0, 1\}$, it's clear that the bond percolation probability should be $p_c = \frac{1}{Z-1}$.

2.3 More advanced configurations

Unfortunately, it's outside the scope of this essay to consider more intricate configurations due to the extreme complexity that arises very quickly. Consider, for example, the lattice on $\mathbb{Z}^2$. It's troublesome to use the technique of considering clusters and the number of empty sites surrounding such clusters because of the many forms that any given cluster can take. If we have a cluster C of size $|C| = 1$ then there's only one possible form for that where it's one open site surrounded by four closed sites giving us a probability of the site existing of $p(1-p)^4$. Now consider a cluster C of size $|C| = 9$. This cluster can take many different shapes that have different "perimiters". It could be a 3×3 square of open sites surrounded by 12 closed ones giving us a probability of $p^9(1-p)^{12}$ of this cluster existing. It could also be a 9×1 line of open sites that has a total of 20 closed sites surrounding it resulting in a probability of $p^9(1-p)^{20}$ of this cluster existing. The fact that the probability of these clusters existing isn't uniquely defined by their size makes analysing these problems this way much more problematic. As such, this essay will not explore the methods for calculating the percolation probabilities for these more complicated configurations.

3 Applications

This section will consider some of the applications of percolation theory and this idea of critical thresholds — the idea that there exists a point in a system where the structure of said system changes massively.

3.1 Epidemiology

It wouldn't be an essay written in 2020-2021 if I didn't mention epidemiology in one way or another. This part of the essay will focus on and summarise a paper written in 2011 by Eben Kenah and Joel C. Miller at the University of Washington [Eben Kenah, 2011]. The aforementioned paper sets up a

Susceptible, Exposed, Infected, Removed (SEIR) model. Every member of the population is in one of these four states at any given time. If a member of the population is:

- susceptible then they're in a position where they could contract the disease
- exposed then they have been exposed to the disease and it is currently in a latent period during which the member is infected but not infectious
- infected then the member is now infectious with the disease
- removed then the member is no longer susceptible to the disease and nor are they infectious.

The E and I stages take some time to transition through and in the model are equipped with an ε_i and a r_i to describe the amount of time it takes to become infectous after being exposed and the amouont of time it takes to recover after becoming infected. Furthermore, we say that the *size* of an epidemic is the total number of people infected over all time periods and that the *attack rate* of an epidemic is the number of people infected in any given time frame.

The model in this paper is called an Epidemic Percolation Network (EPN) and it relies on representing each individual i in the population as a vertex in a network. Then for every other j in the population the edge between i and j can be one of the four following things:

1. no edge between i and j,
2. a directed edge from i to j,
3. a directed edge from j to i,
4. an undirected edge between i and j.

Point 2 means that i will infect j if i is ever infected, point 3 means that j will infect i if j is ever infected, point 4 means that if i is infected then it will infect j or vice versa and point 1 means that if i is infected it will *not* infect j or vice versa. It's worth noting that these edges are assigned randomly and as a result we have a random network.

We define the *in-component* of i to be the set of nodes from which i can be reached by choosing the correct path. We also define the *out-component* of i to be the set of nodes that can be reached from i by following a series of edges. In both of these definitions, the undirected edges may be traversed in either direction. The idea for these definitions is that if any node in the

in-component of i is infected then i will eventually be infected and if i is infected then every node in the out-component of i will be infected eventually. As a result of the randomness of the network, any given individual i does not have a set in- or out-components. We consider the *epidemic threshold* of this SEIR model to correspond to the emergence of large components in the EPN. We define a *strongly connected component* (SCC) to be a group of nodes wherein each node can be reached by any other node. As a result of all these nodes being connected, each node in a SCC has the same in- and out-component as every other node in the SCC. As before, the transition in structure occurs when we go from having a mostly disconnected structure to a mostly connected structure. As such, an EPN below the epidemic threshold gives us many small SCCs, but an EPN above the epidemic threshold gives us one *giant strongly connected component* (GSCC) and many small SCCs. We lable the in-component of the GSCC as GIN and the out-component of the GSCC as GOUT. Armed with this extra terminology, we can now see that if all initial infections occur outside the GIN then we have a minor epidemic because the out-components of all initial infections are small. However, if the initial infection is in the GIN, then the infection necessarily spreads to the GSCC and also to the GOUT so we have a major epidemic.

This model turns out to be useful for understanding how we should vaccinate the population. This paper tested two pre-vaccination strategies on this network based EPN (i.e. vaccinating before the start of an outbreak). Strategy 1: Ranking all nodes in the network by decreasing degree. Strategy 2: Generate a single instance of the EPN and rank all of the nodes in the GSCC by the number of edges connecting them to other nodes in the GSCC. All other nodes were ranked randomly underneath the nodes in the GSCC. Then in both strategies, the paper chooses some vaccination fraction, ν, and vaccinates the first $n\nu$ nodes in the list where n is the number of nodes in the population. These strategies were tested on two types of network: Erdős-Rényi network with mean degree 5 and a scale-free network with $\alpha = 2$.

An Erdős-Rényi network of mean degree 5 just means that if you sum the degrees of all the nodes and divide that by the number of nodes you should get something close to 5. A scale-free network with $\alpha = 2$ means that the fraction of nodes in the network of degree k is given by $P(k) \sim k^{-\alpha}$.

The paper also adds a methodd for representing the variation in susceptibility and infectiousness between any two given nodes which is represented by the following quantity:

$$p_{ij} = 1 - e^{-100 \times \text{inf}_i \times \text{sus}_j}$$

Where $0 \leq \text{inf}_i \leq 1$ is the infectiousness of i and $0 \leq \text{sus}_j \leq 1$ is the susceptibility of j. The paper then allowed inf_i and sus_j to have different beta distributions. The two cases that were studied for a beta(2, 2) distribution

and a beta(0.25, 0.25) distribution. The beta(2, 2) distribution just has one peak at 0.5 and as a result there isn't much variation in susceptibility and infectiousness. The beta(0.25, 0.25) distribution, however, has peaks at 0 and 1 resulting in nodes either having very high or very low infectiousness or susceptibility.

As is clear from Figure 6, strategy 2 of targeting the GSCC is much better at reducing the probability of an epidemic and reducing the attack size of an epidemic when there's significant variation in the susceptibility and infectiousness of members of the population. In general, this paper's analysis of vaccination is that vaccinating the highly infectious (i.e. those likely to be in the GIN) will reduce the probability of an epidemic, and vaccinating the highly susceptible (i.e. those likely to be in the GOUT) will reduce its attack rate. Vaccinating those likely to be in the GSCC will do both. The major limitation with this model is that it's time-homogeneous meaning that this model cannot account for seasonality, change in behaviour of the population, or the effects of mitigating strategies that are implemented once a certain level of infection is reached. Say, for example, that vaccination starts after the epidemic has already started, then that epidemic may not necessarily be modelled accurately by an EPN.

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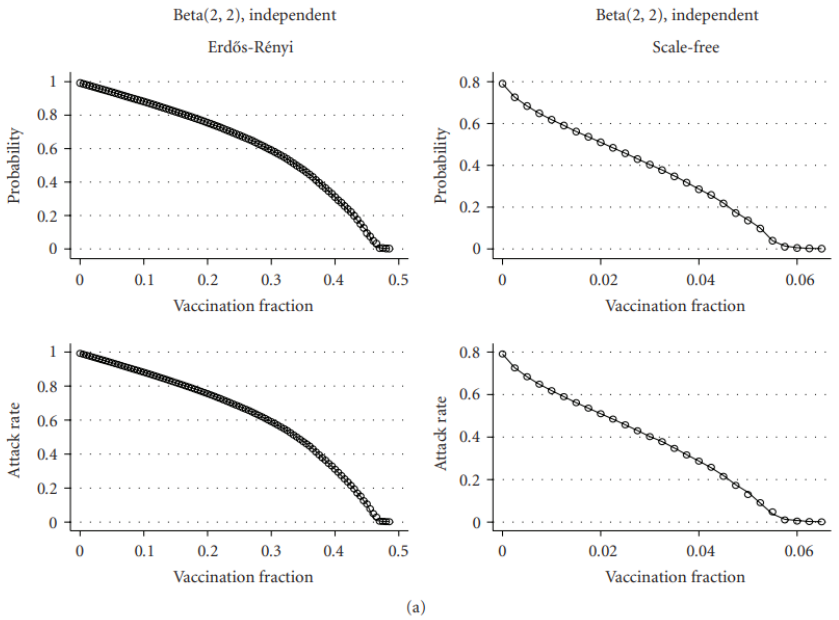

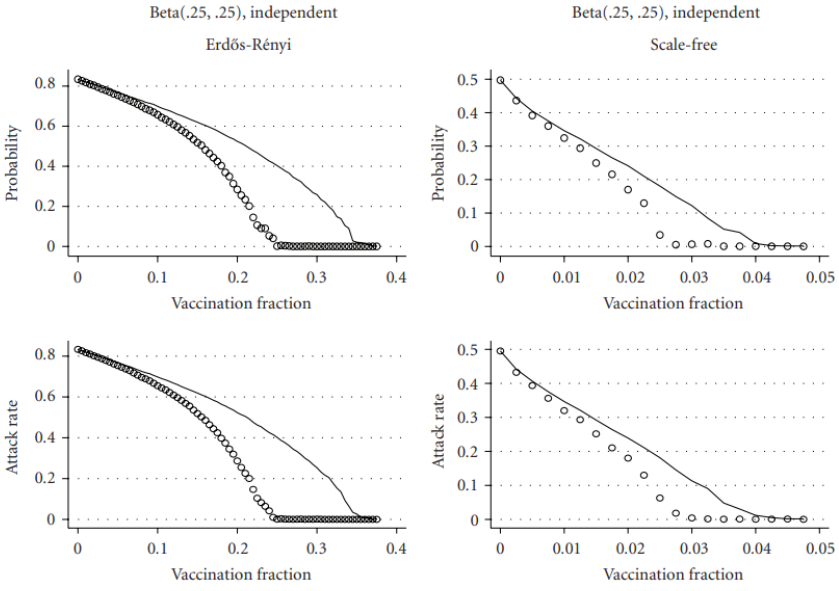

Figure 6: A comparison of strategy 1 (lines) versus strategy 2 (circles) in reducing the attack rate and probability of an epidemic on Erdős-Rényi networks and scale-free networks. When the infectiousness and susceptibility are beta(2, 2) distributed, both strategies produce nearly identical results. When infectiousness and susceptibility are beta(0.25, 0.25) distributed, targeting the GSCC is clearly more effective in reducing both the attack rate and the probability of an epidemic in both network configurations.[Eben Kenah, 2011]