CHAPTER 17

EPIDEMICS ON NETWORKS

An introduction to the theory of the epidemic processes by which diseases spread over networks of contact between humans, animals, plants, and even computers

ONE OF the reasons for the large investment the scientific community has made in the study of social networks is their connection with the spread of disease. Diseases spread over networks of contacts between individuals: airborne diseases like influenza or tuberculosis are communicated when two people breathe the air in the same room; contagious diseases and parasites can be communicated when people touch; HIV and other sexually transmitted diseases are communicated when people have sex. The patterns of such contacts can be represented as networks and a good deal of effort has been devoted to empirical studies of these networks' structure. We have already discussed some network aspects of epidemiology in the previous chapter when we considered site percolation as a model for the effects of vaccination. In this chapter we look in more detail at the connections between network structure and disease dynamics and at mathematical theories that allow us to understand and predict the outcomes of epidemics.

On a related topic, recent years have seen the emergence of a new type of infection, the computer virus, a self-reproducing computer program that spreads from computer to computer in a manner similar to the spread of pathogenic infections between humans or animals. Many of the ideas described in this chapter can be applied not only to human diseases but also to computer viruses.

17.1 MODELS OF THE SPREAD OF DISEASE

The biology of what happens when an individual (a "host" in the epidemiology jargon) catches an infection is complicated. The pathogen responsible

for the infection typically multiplies in the body while the immune system attempts to beat it back, often causing symptoms in the process. One or the other usually wins in the end, though sometimes neither, with the final result being the individual's recovery, their death, or a chronic disease state of permanent infection. In theory if we want to understand fully how diseases spread through populations we need to take all of this biology into account, but in practice that's usually a dauntingly large job and it is rarely, if ever, attempted. Luckily there are more tractable approaches based on simplified models of disease spread that give a good guide to disease behavior in many cases and it is on these that we focus in this chapter.

17.2 THE SI MODEL

In the typical mathematical representation of an epidemic the within-host dynamics of the disease is reduced to changes between a few basic disease states. In the simplest version there are just two states, *susceptible* and *infected*. An individual in the susceptible state is someone who does not have the disease yet but could catch it if they come into contact with someone who does. An individual in the infected state is someone who has the disease and can, potentially, pass it on if they come into contact with a susceptible individual.[1] Although this two-state classification sweeps a lot of biological details under the rug, it captures some of the gross features of disease dynamics and is a useful simplification in the case where, as here, we are focused more on what's happening at the level of networks and populations than on what's happening within the bodies of the individual population members.

Mathematical modeling of epidemics predates the study of networks by many years, stretching back at least as far as the pioneering work of Anderson McKendrick, a doctor and amateur mathematician who made foundational contributions to the field early in the twentieth century. The theories that he and others developed form the core of traditional mathematical epidemiology, which is an extensive and heavily researched field. Classic introductions to the

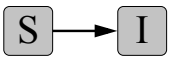
[1] If you look at the epidemiology literature you will sometimes see the infected state referred to as "infective." There's no difference between the two terms; they are synonymous. You may also see the word "infectious" used, but this may mean something slightly different. As discussed later in the chapter, more sophisticated models of disease distinguish between a state in which an individual has a disease but it has not yet developed to the point where the individual can pass it on, and a state where they can pass it on. This latter stage is sometimes called the "infectious" stage, a name chosen to emphasize that the disease can be communicated. (The former state is usually called the "exposed" state.) In the present simple two-state model, however, there is no difference between infected and infectious; all individuals who are one are also the other.

subject include the highly theoretical 1975 book by Bailey [25] and the more recent and practically oriented book by Anderson and May [17]. The review article by Hethcote is also a good resource [156].

The traditional approach avoids discussing contact networks at all by making use of a *fully mixed* or *mass-action approximation*, in which it is assumed that every individual has an equal chance, per unit time, of coming into contact with every other—people mingle and meet completely at random in this approach. This is, of course, not a realistic representation of the way the world is. In the real world, people have contact with only a small fraction of the population of the world, and that fraction is not chosen at random, which is precisely why networks play an important role in the spread of disease. Nonetheless, a familiarity with the traditional approaches will be useful to us in our study of network epidemiology, so we will spend a little time looking at its basic principles.

Consider a disease spreading through a population of individuals. Let $S(t)$ be the number of individuals who are susceptible at time t and let $X(t)$ be the number who are infected.[2] Technically, since the disease-spreading process is a random one, these numbers are not uniquely determined—if the disease were to spread through the same population more than once, even under very similar conditions, the numbers would probably be different each time. To get around this problem let us define S and X more carefully to be the average or expected numbers of susceptible and infected individuals, i.e., the numbers we would get if we ran the process many times under identical conditions and then averaged the results.[3]

The number of infected individuals goes up when susceptible individuals contract the disease from infected ones. Suppose that people meet and make contacts sufficient to result in the spread of disease entirely at random with a per-individual rate β, meaning that each individual has, on average, β contacts with randomly chosen others per unit time.



The allowed transitions between states can be represented by flow charts like this simple one for the SI model.

The disease is transmitted only when an infected person has contact with a susceptible one. If the total population consists of n people, then the average probability of a person you meet at random being susceptible is S/n, and hence an infected person has contact with an average of $\beta S/n$ susceptible people per unit time. Since there are on average X infected individuals in total that means the overall average rate of new infections will be $\beta SX/n$ and we can write a

[2] It might be more logical to use $I(t)$ for the number infected, and many authors do so, but we use X instead to avoid later confusion with the index i used to label vertices.

[3] For convenience we will usually drop the explicit t-dependence of $S(t)$ and $X(t)$ and, as here, just write S and X.

differential equation for the rate of change of X thus:

$$\frac{\mathrm{d}X}{\mathrm{d}t} = \beta\frac{SX}{n}. \tag{17.1}$$

At the same time the number of susceptible individuals goes down at the same rate:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\beta\frac{SX}{n}. \tag{17.2}$$

This simple mathematical model for the spread of a disease is called the *fully mixed susceptible–infected model*, or *SI model* for short.

It is often convenient to define variables representing the fractions of susceptible and infected individuals thus:

$$s = \frac{S}{n}, \qquad x = \frac{X}{n}, \tag{17.3}$$

in terms of which Eqs. (17.1) and (17.2) can be written

$$\frac{\mathrm{d}s}{\mathrm{d}t} = -\beta sx, \tag{17.4a}$$

$$\frac{\mathrm{d}x}{\mathrm{d}t} = \beta sx. \tag{17.4b}$$

In fact, we don't really need both of these equations, since it is also true that $S + X = n$ or equivalently $s + x = 1$ because every individual must be either susceptible or infected. With this condition it is easy to show that Eqs. (17.1) and (17.2) are really the same equation. Alternatively, we can eliminate s from the equations altogether by writing $s = 1 - x$, which gives

$$\frac{\mathrm{d}x}{\mathrm{d}t} = \beta(1-x)x. \tag{17.5}$$

This equation, which occurs in many places in biology, physics, and elsewhere, is called the *logistic growth equation*. It can be solved using standard methods to give

$$x(t) = \frac{x_0 \mathrm{e}^{\beta t}}{1 - x_0 + x_0 \mathrm{e}^{\beta t}} \tag{17.6}$$

where x_0 is the value of x at $t = 0$. Generically this produces an S-shaped "logistic growth curve" for the fraction of infected individuals, as shown in Fig. 17.1. The curve increases exponentially for short time, corresponding to the initial phase of the disease in which most of the population is susceptible, and then saturates as the number of susceptibles dwindles and the disease has a harder and harder time finding new victims.[4]

[4]There aren't many diseases that really saturate their population like this. Most real diseases

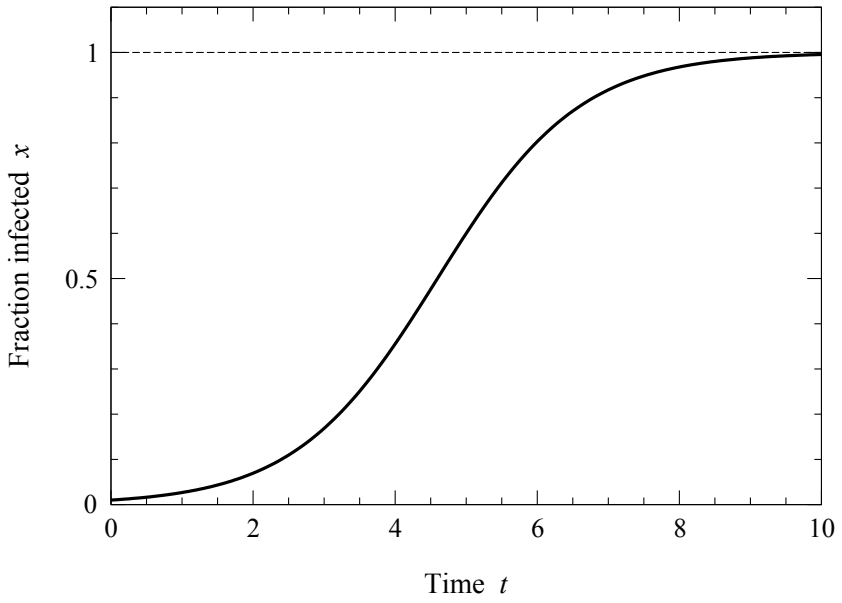


Figure 17.1: The classic logistic growth curve of the SI epidemic model. A small initial number of infected individuals in an SI model (1% in this example) will at first grow exponentially as they infect others, but growth eventually saturates as the supply of susceptible individuals is exhausted, and the curve levels off at $x = 1$.

17.3 THE SIR MODEL

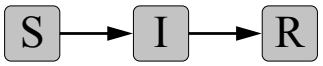
The SI model is the simplest possible model of infection. There are many ways in which it can be extended to make it more realistic or more appropriate as a model of specific diseases. One common extension deals with recovery from disease.

In the SI model individuals, once infected, are infected (and infectious) forever. For many real diseases, however, people recover from infection after a certain time because their immune system fights off the agent causing the disease. Furthermore, people often retain their immunity to the disease after such a recovery so that they cannot catch it again. To represent this behavior in our

that don't kill their victims are eventually defeated by the immune system. In addition, for many diseases some fraction of the population has a natural immunity that prevents them from being infected (meaning that when exposed to the pathogen their immune system sees it off so quickly that they never become infectious). And some diseases spread so slowly that a large fraction of the population never catches them because they die of other causes first. None of these phenomena is represented in this model.

model we need a new third disease state, usually denoted R for *recovered*. The corresponding three-state model is called the *susceptible–infected–recovered* or *SIR model*.

With some other diseases people do not recover, but instead die after some interval. Although this is the complete opposite of recovery in human terms, it is essentially the same thing in epidemiological terms: it makes little difference to the disease whether a person is immune or dead—either way they are effectively removed from the pool of potential hosts for the disease.[5] Both recovery and death can be represented by the R state in our model. Diseases with mixed outcomes where people sometimes recover and sometimes die can also be modeled in this way—from a mathematical point of view we don't care whether the individuals in the R state are recovered or dead. For this reason some people say that the R stands for *removed* rather than recovered, so as to encompass both possibilities, and they refer to the corresponding model as the *susceptible–infected–removed* model.



The flow chart for the SIR model.

The dynamics of the fully mixed SIR model has two stages. In the first stage, susceptible individuals become infected when they have contact with infected individuals. Contacts between individuals are assumed to happen at an average rate β per person as before. In the second stage, infected individuals recover (or die) at some constant average rate γ.

Given the value of γ we can calculate the length of time τ that an infected individual is likely to remain infected before they recover. The probability of recovering in any time interval $\delta\tau$ is $\gamma\,\delta\tau$ and the probability of not doing so is $1-\gamma\,\delta\tau$. Thus the probability that the individual is still infected after a total time τ is given by

$$\lim_{\delta\tau\to 0}(1-\gamma\,\delta\tau)^{\tau/\delta\tau} = \mathrm{e}^{-\gamma\tau}, \tag{17.7}$$

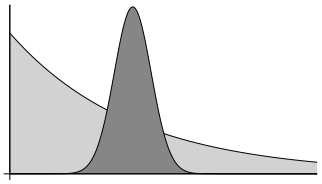
and the probability $p(\tau)\,\mathrm{d}\tau$ that the individual remains infected this long and then recovers in the interval between τ and $\tau+\mathrm{d}\tau$ is this quantity times $\gamma\,\mathrm{d}\tau$:

$$p(\tau)\,\mathrm{d}\tau = \gamma\mathrm{e}^{-\gamma\tau}\,\mathrm{d}\tau, \tag{17.8}$$

[5]This is only approximately true. If people really do have a certain average number of contacts per unit time and assuming those contacts are with living people, then the presence of living but recovered people in the population reduces the number of contacts between infected and susceptible individuals. If, on the other hand, people die rather than recover from the disease then only susceptible and infected individuals are alive and the number of contacts between them will be correspondingly greater. In effect, a person whose acquaintance dies from the disease will (on average) gain one new acquaintance from among the living to replace them, and that new acquaintance might be infected, or might become infected, thereby increasing the chance of transmission of the disease. This effect can easily be incorporated into the model, but we don't do so here.

which is a standard exponential distribution. Thus an infected person is most likely to recover just after becoming infected, but might in theory remain in the infected state for quite a long time—many times the mean infectious time (which is just $1/\gamma$).

Neither of these behaviors is very realistic for most real diseases. With real diseases, most victims remain infected for about the same length of time, such as a week, say, or a month. Few stay in the infected state for much longer or shorter than the average (see figure). Nonetheless, we will for the moment stick with this model because it makes the mathematics simple. This is one thing that will improve when we come to look at network models of epidemics.



The distribution of times for which an individual remains infected is typically narrowly peaked around some average value for real diseases, quite unlike the exponential distribution assumed by the SIR model.

In terms of the fractions s, x, and r of individuals in the three states, the equations for the SIR model are

$$\frac{\mathrm{d}s}{\mathrm{d}t} = -\beta s x, \tag{17.9a}$$

$$\frac{\mathrm{d}x}{\mathrm{d}t} = \beta s x - \gamma x, \tag{17.9b}$$

$$\frac{\mathrm{d}r}{\mathrm{d}t} = \gamma x, \tag{17.9c}$$

and in addition the three variables necessarily satisfy

$$s + x + r = 1. \tag{17.10}$$

To solve these equations we eliminate x between Eqs. (17.9a) and (17.9c), giving

$$\frac{1}{s}\frac{\mathrm{d}s}{\mathrm{d}t} = -\frac{\beta}{\gamma}\frac{\mathrm{d}r}{\mathrm{d}t}, \tag{17.11}$$

and then integrate both sides with respect to t to get:

$$s = s_0 \mathrm{e}^{-\beta r/\gamma}, \tag{17.12}$$

where s_0 is the value of s at $t = 0$ and we have chosen the constant of integration so that there are no individuals in the recovered state at $t = 0$. (Other choices are possible but we'll use this one for now.)

Now we put $x = 1 - s - r$ in Eq. (17.9c) and use Eq. (17.12) to get

$$\frac{\mathrm{d}r}{\mathrm{d}t} = \gamma\bigl(1 - r - s_0 \mathrm{e}^{-\beta r/\gamma}\bigr). \tag{17.13}$$

If we can solve this equation for r then we can find s from Eq. (17.12) and x from Eq. (17.10).

The solution is easy to write down in principle. It is given by

$$t = \frac{1}{\gamma}\int_0^r \frac{\mathrm{d}u}{1 - u - s_0 \mathrm{e}^{-\beta u/\gamma}}\,. \tag{17.14}$$

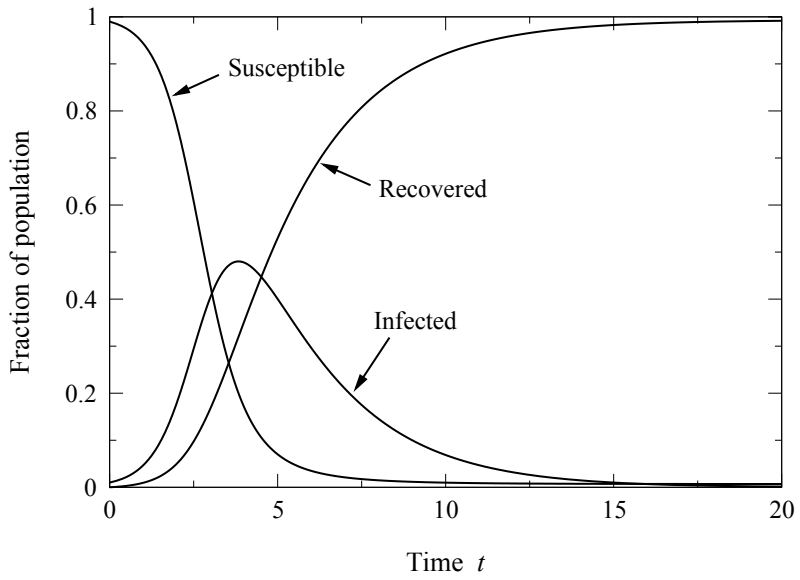


Figure 17.2: Time evolution of the SIR model. The three curves in this figure show the fractions of the population in the susceptible, infected, and recovered states as a function of time. The parameters are $\beta = 1$, $\gamma = 0.4$, $s_0 = 0.99$, $x_0 = 0.01$, and $r_0 = 0$.

Unfortunately, in practice we can't evaluate the integral in closed form. We can however evaluate it numerically. An example is shown in Fig. 17.2.

There are a number of notable things about this figure. The fraction of susceptibles in the population decreases monotonically as susceptibles are infected and the fraction of recovered individuals increases monotonically. The fraction infected, however, goes up at first as people get infected, then down again as they recover, and eventually goes to zero as $t \to \infty$.

Note however that the number of susceptibles does not go to zero; a close inspection shows that the curve for $s(t)$ ends a little above the axis. This is because when $x \to 0$ there are no infected individuals left to infect the remaining susceptibles. Any individuals who survive to late enough times without being infected will probably never get the disease at all. They are the lucky ones who made it through the outbreak and out the other side. Similarly the fraction of recovered individuals does not quite reach one as $t \to \infty$.

The asymptotic value of r has an important practical interpretation: it is the total number of individuals who ever catch the disease during the entire course of the epidemic—the total size of the outbreak. It can be calculated from Eq. (17.13) as the value at which $\mathrm{d}r/\mathrm{d}t = 0$, which gives $r = 1 - s_0 \mathrm{e}^{-\beta r/\gamma}$.

The initial conditions for the model can be chosen in a variety of ways, but the most common is to assume that the disease starts with either a single infected individual or a small number c of individuals and everyone else in the susceptible state. In other words, the initial values of the variables are $s_0 = 1 - c/n$, $x_0 = c/n$, and $r_0 = 0$. In the limit of large population size $n \to \infty$, we can then write $s_0 \simeq 1$, and our final value of r satisfies

$$r = 1 - e^{-\beta r/\gamma}. \tag{17.15}$$

Interestingly, this is the same as the equation we derived in Section 12.5 for the size S of the giant component of a Poisson random graph, Eq. (12.15), provided we equate β/γ with the mean degree of the random graph, and this correspondence allows us immediately to say several useful things. First, we know what the size of the epidemic must look like (in the limit of large n) as a function of the parameters β and γ: it will look like the plot of giant component size shown in the right-hand panel of Fig. 12.1 on page 406, with $c = \beta/\gamma$. Second, it tells us that the size of the epidemic goes continuously to zero as β/γ approaches one from above and for $\beta/\gamma \leq 1$, or equivalently $\beta \leq \gamma$, there is no epidemic at all. The simple explanation for this result is that if $\beta \leq \gamma$ then infected individuals recover faster than susceptible individuals become infected, so the disease cannot get a toehold in the population. The number of infected individuals, which starts small, goes down, not up, and the disease dies out instead of spreading.

The transition between the epidemic and non-epidemic regimes happens at the point $\beta = \gamma$ and is called the *epidemic transition*. Note that there was no epidemic transition in the simpler SI model: in that model the disease always spreads because individuals once infected never recover and hence the number of infected individuals cannot decrease. (One can think of the SI model as the special case of the SIR model in which $\gamma = 0$, so that β can never be less than γ.)

An important quantity in the study of epidemics is the *basic reproduction number*, denoted R_0, which is defined as follows. Consider the spread of a disease when it is just starting out, when there are only a few cases of the disease and the rest of the population is susceptible—what is called a *naive population* in the epidemiology jargon—and consider a susceptible who catches the disease in this early stage of the outbreak. The basic reproduction number is defined to be the average number of additional people that such a person passes the disease onto before they recover. For instance, if each person catching the disease passes it onto two others on average, then $R_0 = 2$. If half of them pass it on to just one person and the rest to none at all, then $R_0 = \frac{1}{2}$, and so forth.

If we had $R_0 = 2$ then each person catching the disease would pass it on to two others on average, each of them would pass it on to two more, and so

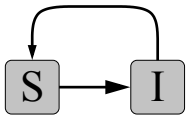
forth, so that the number of new cases of the disease would double at each round, thus growing exponentially. Conversely if $R_0 = \frac{1}{2}$ the disease would die out exponentially. The point $R_0 = 1$ separates the growing and shrinking behaviors and thus marks the *epidemic threshold* between regimes in which the disease either multiplies or dies out.

We can calculate R_0 straightforwardly for our model. If an individual remains infectious for a time τ then the expected number of others they will have contact with during that time is $\beta\tau$. The definition of R_0 is specifically for a naive population, and in a naive population all of the people with whom one has contact will be susceptible, and hence $\beta\tau$ is also the total number of people our infected individual will infect. Then we average over the distribution of τ, Eq. (17.8), to get the average number R_0:

$$R_0 = \beta\gamma \int_0^\infty \tau e^{-\gamma\tau}\, d\tau = \frac{\beta}{\gamma}. \tag{17.16}$$

This gives us an alternative way of deriving the epidemic threshold in the SIR: the epidemic threshold falls at $R_0 = 1$, which corresponds in this model to the point $\beta = \gamma$, the same result as we found above by considering the long-time behavior.[6]

17.4 THE SIS MODEL



Flow chart for the SIS model.

A different extension of the SI model is one that allows for *reinfection*, i.e., for diseases that don't confer immunity on their victims after recovery, or confer only limited immunity, so that individuals can be infected more than once. The simplest such model is the *SIS model*, in which there are just two states, susceptible and infected, and infected individuals move back into the susceptible state upon recovery. The differential equations for this model are

$$\frac{ds}{dt} = \gamma x - \beta s x, \tag{17.17a}$$

$$\frac{dx}{dt} = \beta s x - \gamma x, \tag{17.17b}$$

with

$$s + x = 1. \tag{17.18}$$

[6]Note that when $\gamma = 0$, as in the SI model, Eq. (17.16) implies that $R_0 \to \infty$. This is because an infected individual remains infected indefinitely in the SI model and hence can infect an arbitrary number of others, so that R_0 is formally infinite. In any population of finite size, however, the empirical value of R_0 will be finite.

Putting $s = 1 - x$ in Eq. (17.17b) gives

$$\frac{\mathrm{d}x}{\mathrm{d}t} = (\beta - \gamma - \beta x)x, \tag{17.19}$$

which has the solution

$$x(t) = (1 - \gamma/\beta)\frac{Ce^{(\beta-\gamma)t}}{1 + Ce^{(\beta-\gamma)t}}, \tag{17.20}$$

where the integration constant C is fixed by the initial value of x to be

$$C = \frac{\beta x_0}{\beta - \gamma - \beta x_0}. \tag{17.21}$$

In the case of a large population and a small number of initial carriers of the disease we have $x_0 \to 0$ and $C = \beta x_0/(\beta - \gamma)$, which gives us the simpler solution

$$x(t) = x_0\frac{(\beta - \gamma)e^{(\beta-\gamma)t}}{\beta - \gamma + \beta x_0 e^{(\beta-\gamma)t}}. \tag{17.22}$$

If $\beta > \gamma$ this produces a logistic growth curve similar to that of the basic SI model—see Fig. 17.3—but differing in one important respect: we never have the whole population infected with the disease. In the limit of long time the system finds a stable state where the rates at which individuals are infected and recover from infection are exactly equal and a steady fraction of the population—but not all of them—is always infected with the disease. (Which particular individuals are infected changes over time, however, as some recover and others are infected.) The fraction of infected individuals can be found from Eq. (17.22), or more directly from Eq. (17.19) by setting $\mathrm{d}x/\mathrm{d}t = 0$ to give $x = (\beta - \gamma)/\beta$. In the epidemiology jargon the steady state is called an *endemic disease state*.

Note that the fraction infected in the endemic state goes to zero as β approaches γ, and if $\beta < \gamma$ then Eq. (17.22) predicts that the disease will die out exponentially. Thus, as in the SIR model, the point $\beta = \gamma$ marks an epidemic transition between a state in which the disease spreads and one in which it doesn't. As before, we can calculate a basic reproduction number R_0, which again takes the value $R_0 = \beta/\gamma$, giving us an alternative derivation of the position of the transition as the point at which $R_0 = 1$.

17.5 THE SIRS MODEL

We will look at one more epidemic model before we turn to the properties of these models on networks. This is the *SIRS model*, another model incorporating reinfection. In this model individuals recover from infection and gain

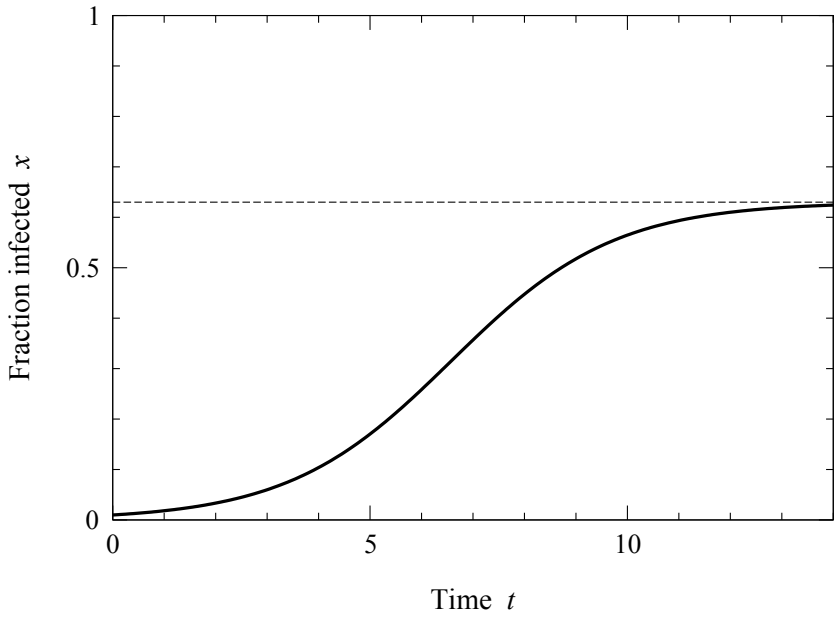
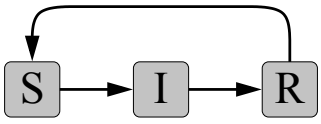


Figure 17.3: Fraction of infected individuals in the SIS model. The fraction of infected individuals in the SIS model grows with time following a logistic curve, as in the SI model. Unlike the SI model, however, the fraction infected never reaches unity, tending instead to an intermediate value at which the rates of infection and recovery are balanced. (Compare this figure with Fig. 17.1 for the SI model.)



Flow chart for the SIRS model

immunity as in the SIR model, but that immunity is only temporary, and after a certain period of time individuals lose it and become susceptible again. We introduce a new parameter δ to represent the average rate at which individuals lose immunity. Then the equations for this model are

$$\frac{\mathrm{d}s}{\mathrm{d}t} = \delta r - \beta s x, \tag{17.23a}$$

$$\frac{\mathrm{d}x}{\mathrm{d}t} = \beta s x - \gamma x, \tag{17.23b}$$

$$\frac{\mathrm{d}r}{\mathrm{d}t} = \gamma x - \delta r, \tag{17.23c}$$

and

$$s + x + r = 1. \tag{17.24}$$

The SIRS model cannot be solved analytically, although it can be treated using linear stability analysis and other tricks from the non-linear dynamics toolbox. A more straightforward approach is numerical integration of the dif-

ferential equations, which reveals that the SIRS model has a rich palette of behaviors depending on the values of the three parameters, including behaviors where the disease persists in an endemic state, where it dies out, and where it oscillates between outbreaks and periods of remission. We will not delve into the behavior of the SIRS model further in this chapter; the interested reader can find more details in Ref. [156].

Many other epidemic models have also been proposed to model the spread of particular types of diseases. Extra states can be introduced such as an "exposed" state that represents people who have caught a disease but whose infection has not yet developed to the point where they can pass it on to others; or an initial immune state coming before the susceptible state, often used to represent the maternally derived immunity that newborn babies possess. There are also models that allow for new individuals to enter the population, by being born or immigrating, and models that distinguish between people who recover fully from disease and those who recover but remain carriers who can pass the disease to others. Those interested in pursuing the subject further are encouraged to take a look at the references given at the beginning of the chapter. For our purposes, however, the models we have seen so far will be enough. Let's look at how these models behave when we include network structure in our calculations.

17.6 EPIDEMIC MODELS ON NETWORKS

As discussed in Section 17.2, the standard approach to epidemic modeling described in the first part of this chapter assumes "full mixing" of the population, meaning that each individual can potentially have contact with any other, those contacts being realized, at a level sufficient to transmit the disease, with probability β per unit time.

In the real world, however, it is not a good assumption to say that any two people could potentially have contact with one another. The chance of a meeting between two people chosen at random from the population of the entire world is probably small enough to be negligible. Most people have a set of regular acquaintances, neighbors, coworkers, and so forth whom they meet with some regularity and most other members of the world population can safely be ignored. The set of a person's potential contacts can be represented as a network and the structure of that network can have a strong effect on the way a disease spreads through the population.

Network models of disease typically work in the same way as the fully

mixed models we have already seen but make use of this network of potential contacts instead of assuming that contact is possible with the entire population. Let us define the *transmission rate* or *infection rate* for our network disease process to be the probability per unit time that infection will be transmitted between two individuals, one susceptible and one infected, who are connected by an edge in the appropriate network. Alternatively it is the rate at which contact sufficient to spread the disease occurs between any two individuals connected by an edge. The transmission rate is commonly denoted β by analogy with the quantity appearing in the fully mixed models, and we will adopt that notation here, although you should note that the two parameters are not exactly equivalent since β in the fully mixed case is the rate of contacts between an infected individual and all others in the population, whereas in the network case it is the rate of contacts with just one other.

The transmission rate is a property of the disease. Some diseases are transmitted more easily than others and so have higher transmission rates. But transmission rate is also a property of the social and behavioral parameters of the population. In some countries, for example, it is common etiquette for people with minor respiratory infections such as colds to wear surgical face masks to prevent the spread of disease. Such conventions are absent in other countries, and the difference in conventions could produce a difference in transmission rate.

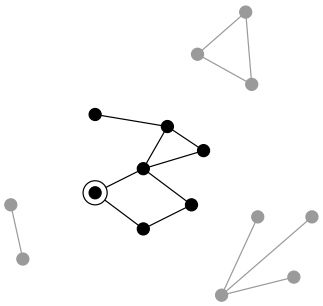
17.7 LATE-TIME PROPERTIES OF EPIDEMICS ON NETWORKS

Given a value for the transmission rate one can define models for the spread of disease over a network. Each of the models introduced in the first part of the chapter can be generalized to the network case. Consider the SI model, for instance. In the network version of this model we have n individuals represented by the vertices of our network, with most of them in the susceptible state at time $t = 0$ and just a small fraction x_0, or maybe even just a single vertex, in the infected state. With probability β per unit time, infected nodes spread the disease to their susceptible neighbors and over time the disease spreads across the network.

It is difficult to solve a model such as this for a general network, and in many cases the best we can do is to simulate it on a computer. There is, however, one respect in which the model is straightforward, and that is its late-time properties. It is clear that as $t \to \infty$ in this model every individual who *can* be infected by the disease is infected: since infected individuals remain infectious forever, their susceptible neighbors will always, in the end, also become infected, no matter how small the transmission rate, so long as it is not zero. The

only condition for being infected therefore is that a vertex must be connected to at least one infected individual by at least one path through the network, so that the disease can reach them.

Thus in the limit of long times the disease will spread from every initial carrier to infect all reachable vertices, meaning all vertices in the component to which the carrier belongs. In the simplest case, where the disease starts out with a single infected carrier, just one component will be infected.



An outbreak starting with a single infected individual (circled) will eventually affect all those in the same component of the network, but leave other components untouched.

As we have seen, however, most networks have a one large component that contains a significant fraction of all vertices in the network, plus, typically, a selection of smaller components. If we have this kind of structure then an interesting behavior emerges. If we start with a single infected individual, and if that individual turns out to belong to the large component, then the disease will infect the large component and we will have a large outbreak. If the individual belongs to one of the small components, however, the disease will only infect the few members of that small component and then die out. If the initial carrier of the disease is chosen uniformly at random from the network, the probability that it will fall in the large component and we will have a large outbreak is simply equal to S, the fraction of the network occupied by the large component, and the size of the outbreak as a fraction of the network will also be S. Conversely, with probability $1 - S$ the initial carrier will fall in one of the small components and the outbreak will be small. In the latter case the size of the outbreak will be given by the size of the appropriate small component. If we can calculate the distribution of sizes of the small components, either analytically or numerically, for the network of interest, then we also know the distribution of possible sizes of these small outbreaks, although unless we know exactly which component the disease will start in we cannot predict its size exactly.

This constitutes a new type of behavior not seen in fully mixed models. In fully mixed models the possible behaviors are also either a run-away epidemic that affects a large fraction of the population, or an outbreak that affects only a few then dies out. But the choice between these outcomes was uniquely determined by the choice of model and the model parameters. For a given model and parameter values the disease always either did one thing or the other. In our network model, however, the behavior depends on the network structure and on the position in the network of the first infected individual. Thus there is a new stochastic element in the process: with identical model parameters and an identical network the disease sometimes takes off and sometimes dies out.

17.8 LATE-TIME PROPERTIES OF THE SIR MODEL

The situation becomes more interesting still when we look at the SIR model. In the SIR model individuals remain infectious for only a finite amount of time and then they recover, so it is in general no longer true (as in the SI model) that the susceptible neighbor of an infected individual will always get infected in the end. If they are lucky, such neighbors may never catch the disease. The probability of this happening can be calculated in a manner similar to the calculation of Eq. (17.7), and is equal to $e^{-\beta\tau}$, where β is again the transmission rate and τ is the amount of time for which the infected individual remains infected. Thus the probability that the disease *is* transmitted is

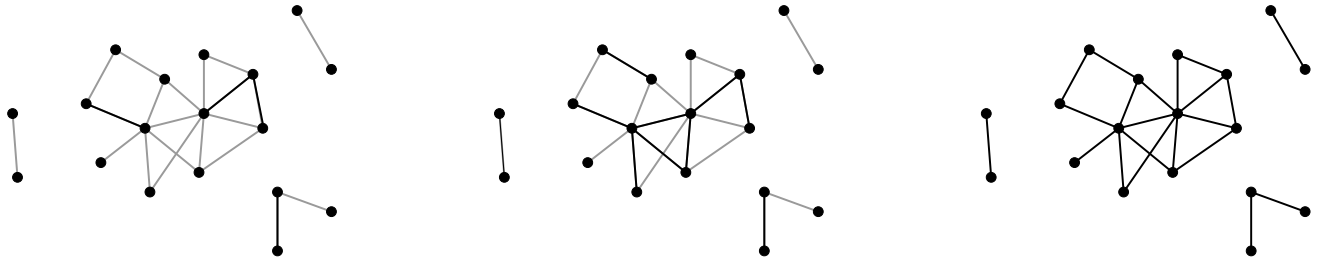
$$\phi = 1 - e^{-\beta\tau}. \tag{17.25}$$

For simplicity, let us suppose that every infected individual remains infectious for the same length of time. This differs from the fully mixed version of the model, where τ was distributed according to an exponential distribution (see Eq. (17.8)), but in many cases is actually more realistic. As mentioned in Section 17.3, observed values of τ for many diseases are narrowly concentrated about a mean value, and their distribution is far from being exponential.

With this assumption, the probability of transmission ϕ is a constant across the whole network. Every susceptible individual has equal probability ϕ of catching the disease from their infected neighbor. (Of course, if they have more than one infected neighbor the total probability is higher.)

Now here is a nice trick, developed originally by Mollison [223] and Grassberger [144]. Let us take our network and "color in" or "occupy" each edge with probability ϕ, or not with probability $1 - \phi$. This is just the ordinary bond percolation process introduced in Section 16.1, where a fraction ϕ of edges are occupied uniformly at random. The occupied edges represent those along which disease will be transmitted if it reaches either of the vertices at the ends of the edge. That is, the occupied edges represent contacts sufficient to spread the disease, but not necessarily actual disease transmission: if the disease doesn't reach either end of an occupied edge then disease will not be transmitted along that edge, so edge occupation only represents the potential for transmission if the disease reaches an edge.

With this in mind consider now the spread of a disease that starts at a randomly chosen vertex. We can immediately see that the set of vertices to which the disease will ultimately spread is precisely the set connected to the initial vertex by any path of occupied edges—the disease simply passes from one vertex to another by traversing occupied edges until all reachable vertices have been infected. The end result is that the disease infects all members of the bond



(a) $\phi = 0.2$ (b) $\phi = 0.5$ (c) $\phi = 1$

Figure 17.4: Bond percolation. In bond percolation, a fraction ϕ of the edges in a network are filled in or "occupied" at random to create connected clusters of vertices. (a) For small occupation probability ϕ the clusters are small. (b) Above the percolation threshold a large cluster forms, though there are usually still some small clusters as well. (c) When $\phi = 1$ all edges are occupied but the large cluster may still not fill the whole network: at $\phi = 1$ the largest cluster corresponds to the largest component of the network, which is often just a subset of the whole network.

percolation cluster to which the initial carrier belongs.

It is important to appreciate that, as with our treatment of the network SI model in the previous section, this process does not give us any information about the temporal evolution of the disease outbreak. Individual infection events are stochastic and a calculation of the curve of infections as a function of time requires a more complicated analysis that takes their randomness into account. However, if we want to know only about long-time behavior, about the overall total number of individuals infected by the disease, then all we need do is count the vertices in the appropriate percolation cluster.

Bond percolation is in many ways similar to the site percolation processes we studied in Chapter 16. Consider Fig. 17.4. For low edge occupation probability ϕ there are just a few occupied bonds which group into small disconnected clusters. But as ϕ increases there comes a point, the percolation transition, where the disconnected clusters grow large enough to join together and form a giant cluster, although usually there exist other small clusters as well that are not joined to the giant cluster. As ϕ increases still further, the giant cluster grows, reaching its maximum size when $\phi = 1$. Notice, however, that this maximum size is not generally equal to the size of the whole network. Even when every edge in the network is occupied, the size of the largest cluster is still limited to the size of the largest component on the network, which is usually smaller than the whole network.

Translating these ideas into the language of epidemiology, we see that for

small values of ϕ the cluster to which the initial carrier of a disease belongs must be small, since all clusters are small. Thus in this regime we will have only a small disease outbreak and most members of the population will be uninfected. Once we reach the percolation transition, however, and a giant cluster forms, then a large outbreak of the disease—an epidemic—becomes possible, although not guaranteed. If the giant cluster of the percolation process occupies a fraction S of the entire network, then our randomly chosen initial vertex will fall within it with probability S, and if it does then the disease will spread to infect the whole giant cluster, creating an epidemic reaching a fraction of the population also equal to S. With probability $1 - S$, on the other hand, the initial vertex will fall in one of the small clusters and we will have only a small outbreak of the disease. As ϕ increases, S also increases and hence both the probability and the size of an epidemic increase with ϕ.

Thus the percolation transition for bond percolation on our network corresponds precisely to the epidemic threshold for a disease on the same network, where the edge occupation probability ϕ is given in terms of the transmission rate β and recovery time τ for the disease by Eq. (17.25), and the sizes of outbreaks are given by the sizes of the bond percolation clusters. This mapping between percolation and epidemics is a powerful one that allows us to make a whole range of calculations of the effects of network structure on the spread of disease.

It is important to note that even when ϕ is above the epidemic threshold we are not guaranteed that there will be an epidemic. This is similar to the situation we saw in the simpler SI model, but different from the situation in the fully mixed SIR model of Section 17.3, where an epidemic always takes place if we are above the epidemic threshold. In many ways the behavior of our network model is more realistic than that of the fully mixed model. For many diseases it is true that outbreaks do not always result in epidemics. Sometimes a disease dies out because, just by chance, its earliest victims happen not to pass the disease on to others. Our theory tells us that the probability of this happening is $1 - S$, where S is the size of the giant cluster, which is also the size of the epidemic if it does happen. The value of $1 - S$ is usually small when we are well above the epidemic threshold, but can be quite large if we are only a little above threshold, meaning that the probability of the disease dying out can be quite large in this regime.

It is also important to bear in mind that percolation is a stochastic process. We occupy edges at random on our network to represent the random nature of the contacts that result in transmission of the disease. Two outbreaks happening under the same conditions on the same networks would not necessarily travel along the same edges and the shapes of the percolation clusters would

not necessarily be the same. Thus a vertex that happens to belong to the giant cluster on one occasion might not belong to it on another and our theory cannot make exact predictions about disease outcomes. The best we can do is calculate probabilities or average behaviors. We could for instance calculate the expected number of people who would be affected by an outbreak, but we cannot predict the exact number for any given outbreak.

17.8.1 SIR MODEL AND THE CONFIGURATION MODEL

In Section 16.2.1 we showed that it is possible to calculate exactly the average behavior of a site percolation process on configuration model networks. With only slight modification the same approach can also be used for bond percolation and hence we can make predictions about the size distribution of epidemics and the position of the epidemic threshold in such networks.

Consider an SIR epidemic process of the kind discussed in the previous section, taking place on a configuration model network with degree distribution p_k. Let u be the average probability that a vertex is not connected to the giant cluster via a specific one of its edges. There are two ways this can happen: either the edge in question can be unoccupied (with probability $1-\phi$), or it is occupied (probability ϕ) but the vertex at the other end of the edge is itself not a member of the giant cluster. The latter happens only if that vertex is not connected to the giant cluster via any of its other edges, which happens with probability u^k if there are k such edges. Thus the total probability is $1-\phi+\phi u^k$.

The value of k is distributed according to the excess degree distribution

$$q_k = \frac{(k+1)p_{k+1}}{\langle k\rangle} \tag{17.26}$$

(see Eq. (16.3)). Averaging over k we then arrive at a self-consistent expression for u thus:

$$u = 1-\phi+\phi\sum_{k=0}^{\infty} q_k u^k = 1-\phi+\phi g_1(u), \tag{17.27}$$

where g_1 is the probability generating function for the excess degree distribution, defined in Eq. (13.49). Equation (17.27) is the same as the corresponding equation for the site percolation case, Eq. (16.4), and has the same solutions.

The probability that a vertex of total degree k does not belong to the giant cluster is now simply u^k, and the average such probability over the whole network, which is equal to $1-S$, is calculated by averaging u^k over the degree distribution p_k giving

$$S = 1-\sum_{k=0}^{\infty} p_k u^k = 1-g_0(u). \tag{17.28}$$

This equation differs from the corresponding equation in the site percolation case, Eq. (16.2), by an overall factor of ϕ, but is otherwise the same. Thus the shape of the curve for S as a function of ϕ will be different from the site percolation case, but the position ϕ_c of the percolation transition, which is dictated by the solution of Eq. (17.27), will be the same. The solution of Eq. (17.27) was shown graphically in Fig. 16.2 and the position of the transition is given by Eq. (16.7) to be

$$\phi_c = \frac{1}{g_1'(1)} = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}. \tag{17.29}$$

This equation thus also gives us the position of the epidemic threshold in terms of the probability ϕ. If we prefer our solution in terms of the more fundamental parameters β and τ we can rearrange Eq. (17.25) to give

$$\beta\tau = -\ln(1-\phi_c) = \ln \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k^2 \rangle - 2\langle k \rangle}. \tag{17.30}$$

If $\beta\tau$ exceeds this value then there is the possibility of an epidemic, though not the certainty, since the initial carrier or carriers of the disease could by chance fall outside the giant cluster. If $\beta\tau$ is smaller than this value then an epidemic is impossible, no matter where the initial carrier falls. The probability of the epidemic, if one is possible, is given by S, Eq. (17.28), as is the size of the epidemic if and when one occurs.

Since the epidemic behavior of the model is controlled by the combination of parameters $\beta\tau$, the epidemic transition can be driven either by an increase in the infectiousness time τ, which is a property of the particular disease under study, or by an increase in the transmission rate β, which is a property both of the disease and of the behavior of members of the population. At the same time, the precise position of the transition in terms of these variables, as well as the probability and size of any epidemic that occurs, depend on the structure of the network via the moments $\langle k \rangle$ and $\langle k^2 \rangle$ of the degree distribution. This contrasts with the fully mixed model of Section 17.3, which incorporated no network effects.

Because of the close similarity between the site and bond percolation problems, we can easily translate a number of the results of Section 16.2.1 into the language of epidemics. For instance, a random graph with a Poisson degree distribution with mean c, which has $g_0(z) = g_1(z) = \mathrm{e}^{c(z-1)}$, has an epidemic threshold falls at $\phi_c = 1/c$ (Eq. (16.11)), or

$$\beta\tau = \ln \frac{c}{c-1}, \tag{17.31}$$

and the size of the epidemic, when there is one, is given by the solution to the equations

$$u = 1 - \phi + \phi e^{c(u-1)}, \qquad S = 1 - e^{c(u-1)}. \tag{17.32}$$

The first of these equations can be rearranged to read $1 - u = \phi(1 - e^{c(u-1)}) = \phi S$ and substituting into the second then gives

$$S = 1 - e^{-\phi c S}, \tag{17.33}$$

which has no simple closed-form solution,[7] but can easily be solved numerically by making an initial guess at the solution ($S = \frac{1}{2}$ seems to work well) and then iterating the equation to convergence.

Note that this equation is similar to Eq. (17.15) for the fully mixed model, but with different parameters. The similarity is not coincidental. In the fully mixed model an infected individual infects others chosen uniformly at random from the population, and in the Poisson random graph the network neighbors of any individual are also chosen uniformly at random. It is possible to show that there is a direct correspondence between the traditional fully mixed model and the network model on a random graph [30].[8]

Another important case is the scale-free network with its power-law degree distribution. As we saw in Section 16.2.1, if the exponent α of the power law in such a network lies in the usual range $2 < \alpha < 3$ then $\phi_c = 0$, because $\langle k^2 \rangle$ diverges while $\langle k \rangle$ remains constant and hence Eq. (17.29) goes to zero. Thus in the power-law case there is always an epidemic, no matter how small the probability of transmission of the disease, at least in the limit of infinite network size. (For finite networks, $\langle k^2 \rangle$ is not infinite, but very large, and ϕ_c is correspondingly very small, but not precisely zero.)

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[7] The solution can be written in closed form using the *Lambert W-function*, which is defined to be the solution of the equation $W(z)e^{W(z)} = z$. In terms of this function, the size of the epidemic is given by

$$S = 1 + \frac{W(-\phi c e^{-\phi c})}{\phi c}.$$

Alternatively, we can rearrange Eq. (17.33) to give ϕ as a function of S rather than the other way around:

$$\phi = -\frac{\ln(1-S)}{cS}.$$

This expression can be useful for making plots of S.

[8] The differences in parameters arise because we are considering a slightly different disease process (one in which each individual is infectious for the same amount of time, rather than the exponential distribution used in the fully mixed model), and also because in the network model β is the transmission rate per edge, rather than the rate for the whole network—this is what gives us the factor of c in the exponent of Eq. (17.33).

This statement is, however, slightly misleading since, as we saw in the previous chapter, the size of the giant cluster in a scale-free network becomes very small as we approach $\phi = 0$; it generally decays faster than linearly with ϕ. Thus although technically there may be an epidemic for all positive values of ϕ, it can be very small in practice, affecting only the tiniest fraction of the population. (On the other hand, the difference between non-epidemic behavior and epidemic behavior, even with a tiny value of S, will become very important when we look at models such as the SIS model that incorporate reinfection. In such models the epidemic threshold separates the regime in which the disease persists and the regime in which it becomes extinct, an important distinction even if the number of individuals infected is small.)

17.9 TIME-DEPENDENT PROPERTIES OF EPIDEMICS ON NETWORKS

The techniques of the previous section can tell us about the late-time properties of epidemics on networks, such as how many people will eventually be affected in an outbreak of a disease. If we want to know about the detailed progression of an outbreak as a function of time, however, then we need another approach that takes dynamics into account. Moreover, the techniques we have used so far cannot tell us about even the late-time behavior of models with reinfection, such as the SIS and SIRS models of Sections 17.4 and 17.5. For these models the equivalence between epidemics and percolation that we used above does not hold, and to understand their behavior, including at long times, we need to address the dynamics of the epidemic.

A number of approaches have been proposed for tackling the dynamics of epidemics on networks, some exact and some approximate. Of course, given a specific network, one can always perform computer simulations of epidemics and get numerical answers for typical disease outbreaks. Analytic approaches, however, offer more insight and some results are known, as discussed below, but they are mostly confined to specific classes of model network, such as random graphs and their generalizations. In the following sections we will look at some of the most straightforward and general approaches to epidemic dynamics on networks, starting with the simple SI model and progressing to the more complex (and interesting) models in later sections.

17.10 TIME-DEPENDENT PROPERTIES OF THE SI MODEL

The analytic treatment of the time-dependent properties of epidemic models revolves around the time evolution of the probabilities for vertices to be in specific disease states. One can imagine having repeated outbreaks of the same

disease on the same network, starting from the same initial conditions, and calculating for example the average probabilities $s_i(t)$ and $x_i(t)$ that vertex i is susceptible or infective at time t. Given the adjacency matrix of a network one can write down equations for the evolution of such quantities in a straightforward manner. Consider for instance the SI model.

An SI outbreak starting with a single randomly chosen vertex somewhere eventually spreads, as we have seen, to all members of the component containing that vertex. Our main interest is in epidemics occurring in the giant component of the network, since all other outbreaks will only affect a small component and then die out, so let us focus on the giant component case.

Consider a vertex i. If the vertex is not a member of the giant component then by hypothesis $s_i = 0$ at all times, since we are assuming the epidemic to take place in the giant component. For i in the giant component we can write down a differential equation for s_i by considering the probability that i becomes infected between times t and $t + \mathrm{d}t$. To become infected an individual must catch the disease from a neighboring individual j, meaning j must already be infected, which happens with probability $x_j = 1 - s_j$, and must transmit the disease during the given time interval, which happens with probability $\beta\,\mathrm{d}t$. In addition we also require that i be susceptible in the first place, which happens with probability s_i. Multiplying these probabilities and then summing over all neighbors of i, the total probability of i becoming infected is $\beta s_i \sum_j A_{ij} x_j$, where A_{ij} is an element of the adjacency matrix. Thus s_i obeys the coupled set of n non-linear differential equations:

$$\frac{\mathrm{d}s_i}{\mathrm{d}t} = -\beta s_i \sum_j A_{ij} x_j = -\beta s_i \sum_j A_{ij}(1 - s_j). \tag{17.34}$$

Note the leading minus sign on the right-hand side—the probability of being susceptible goes down when vertices become infected.

Similarly we can write an equation for x_i thus:

$$\frac{\mathrm{d}x_i}{\mathrm{d}t} = \beta s_i \sum_j A_{ij} x_j = \beta(1 - x_i) \sum_j A_{ij} x_j, \tag{17.35}$$

although the two equations are really the same equation, related to one another by $s_i + x_i = 1$.

We will use the same initial conditions as we did in the fully mixed case, assuming that the disease starts with either a single infected vertex or a small number c of vertices, chosen uniformly at random, so that $x_i = c/n$ and $s_i = 1 - c/n$ for all i. In the limit of large system size n, these become $x_i = 0$, $s_i = 1$, and we will use this large-n limit to simplify some of the expression derived in this and the following sections.

Equation (17.34) is not solvable in closed form for general A_{ij} but we can calculate some features of its behavior by considering suitable limits. Consider for example the behavior of the system at early times. For large n, and assuming initial conditions as above, x_i will be small in this regime. Working with Eq. (17.35) and ignoring terms of quadratic order in small quantities, we have

$$\frac{\mathrm{d}x_i}{\mathrm{d}t} = \beta \sum_j A_{ij} x_j \,, \tag{17.36}$$

or in matrix form

$$\frac{\mathrm{d}\mathbf{x}}{\mathrm{d}t} = \beta \mathbf{A}\mathbf{x}, \tag{17.37}$$

where $\mathbf{x}$ is the vector with elements x_i.

Now let us write $\mathbf{x}$ as a linear combination of the eigenvectors of the adjacency matrix:

$$\mathbf{x}(t) = \sum_{r=1}^{n} a_r(t) \mathbf{v}_r \,, \tag{17.38}$$

where $\mathbf{v}_r$ is the eigenvector with eigenvalue κ_r. Then

$$\frac{\mathrm{d}\mathbf{x}}{\mathrm{d}t} = \sum_{r=1}^{n} \frac{\mathrm{d}a_r}{\mathrm{d}t} \mathbf{v}_r = \beta \mathbf{A} \sum_{r=1}^{n} a_r(t) \mathbf{v}_r = \beta \sum_{r=1}^{n} \kappa_r a_r(t) \mathbf{v}_r \,. \tag{17.39}$$

Then, comparing terms in $\mathbf{v}_r$, we get

$$\frac{\mathrm{d}a_r}{\mathrm{d}t} = \beta \kappa_r a_r \,, \tag{17.40}$$

which has the solution

$$a_r(t) = a_r(0)\, \mathrm{e}^{\beta \kappa_r t}. \tag{17.41}$$

Substituting this expression back into Eq. (17.38), we then have

$$\mathbf{x}(t) = \sum_{r=1}^{n} a_r(0)\, \mathrm{e}^{\beta \kappa_r t}\, \mathbf{v}_r. \tag{17.42}$$

The fastest growing term in this expression is the term corresponding to the largest eigenvalue κ_1. Assuming this term dominates over the others we will get

$$\mathbf{x}(t) \sim \mathrm{e}^{\beta \kappa_1 t} \mathbf{v}_1. \tag{17.43}$$

So we expect the number of infected individuals to grow exponentially, just as it does in the fully mixed version of the SI model, but now with an exponential constant that depends not just on β but also on the leading eigenvalue of the adjacency matrix.

Moreover, the probability of infection in this early period varies from vertex to vertex roughly as the corresponding element of the leading eigenvector $\mathbf{v}_1$. The elements of the leading eigenvector of the adjacency matrix are the same quantities that in other circumstances we called the eigenvector centrality—see Section 7.2. Thus eigenvector centrality is a crude measure of the probability of early infection of a vertex in an SI epidemic.

At long times in the SI model the probability of infection of a vertex in the giant component tends to one (again assuming the epidemic takes place in the giant component). Thus overall we expect the SI epidemic to have a similar form to that seen in the fully mixed version of the model, producing curves qualitatively like that in Fig. 17.1 but with vertices of higher eigenvector centrality becoming infected faster than those of lower.

Reasonable though this approach appears to be, it is not precisely correct, as we can see by integrating Eq. (17.35) numerically. Figure 17.5a shows the results of such a numerical integration (the curve labeled "first-order") on a network generated using the configuration model (Section 13.2), compared against an average over a large number of simulated epidemics with the same β spreading on the same network (the circular dots). As the figure shows, the agreement between the two is good, but definitely not perfect.

The reason for this disagreement is an interesting one. Equation (17.34) may appear to be a straightforward generalization of the equivalent equation for the fully mixed SI model, Eq. (17.4), but there are some subtleties involved. The right-hand side of the equation contains two average quantities, s_i and x_j, and in multiplying these quantities we are implicitly assuming that the product of the averages is equal to the average of their product. In the fully mixed model this is true (for large n) because of the mixing itself, but in the present case it is, in general, not, because the probabilities are not independent. The quantity s_i measures a vertex's probability of being susceptible and x_j measures the probability of its neighbor being infected. It should come as no surprise that in general these quantities will be correlated between neighboring vertices. Correlations of this type can be incorporated into our calculations, at least approximately, by using a so-called pair approximation or moment closure method, as described in the following section.

17.10.1 PAIR APPROXIMATIONS

Correlations between the disease states of different vertices can be handled by augmenting our theory to take account of the joint probabilities for pairs of vertices to have given pairs of states. To handle such joint probabilities we will need to make our notation a little more sophisticated. Let us denote

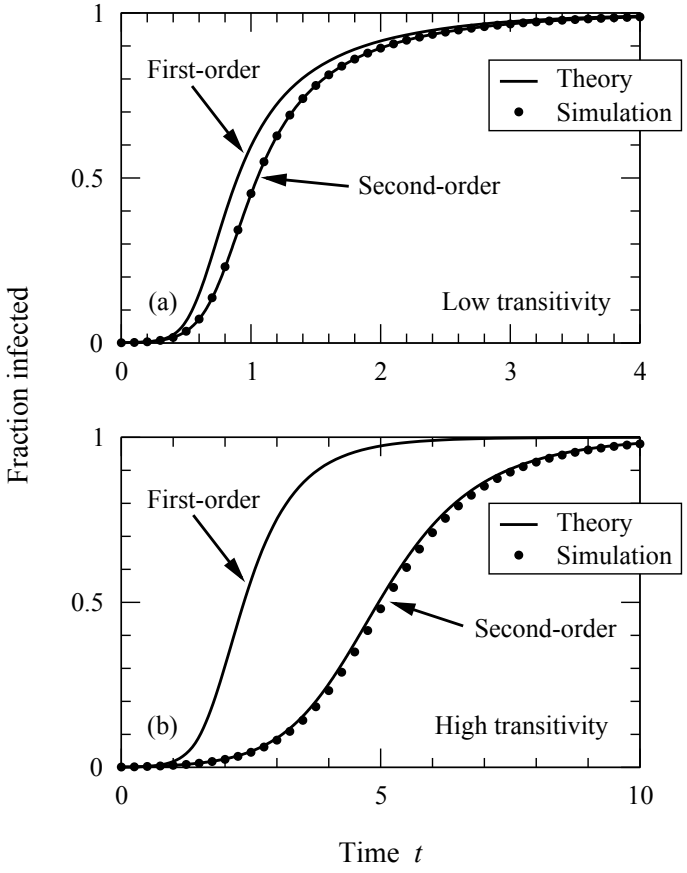

Figure 17.5: Comparison of theory and simulation for the SI model on two different networks. (a) The fraction of infected individuals as a function of time on the giant component of a network with low transitivity (i.e., low clustering coefficient), calculated by numerical solution of the differential equations for the first- and second-order moment closure methods, and by direct simulation. (b) The same comparison for a network with high transitivity. The networks have one million vertices each and the transmission rate is $\beta = 1$ in all cases. Simulation results were averaged over 500 runs.

by $\langle s_i \rangle$ the average probability that vertex i is susceptible. This is the same quantity that we previously called s_i, but, as we will see, it will be useful to indicate the average explicitly with the angle brackets $\langle \ldots \rangle$. If you like, you can think of $s_i(t)$ as now being a variable with value one if i is susceptible at time t and zero otherwise and $\langle s_i \rangle$ as being the average of this quantity over many different instances of disease outbreaks on the same network. Similarly $\langle x_i \rangle$ will be the average probability that i is infected. And $\langle s_i x_j \rangle$ indicates the average probability that i is susceptible *and* j is infected at the same time.

In this notation it is now straightforward to write down a truly exact version of Eq. (17.34), taking correlations into account. It is

$$\frac{\mathrm{d}\langle s_i \rangle}{\mathrm{d}t} = -\beta \sum_j A_{ij} \langle s_i x_j \rangle. \tag{17.44}$$

Equation (17.34) is an approximation to this true equation in which we assume that $\langle s_i x_j \rangle \simeq \langle s_i \rangle \langle x_j \rangle$.

The trouble with Eq. (17.44) is that we cannot solve it directly because it contains the unknown quantity $\langle s_i x_j \rangle$ on the right-hand side. To find this quantity, we need another equation for $\langle s_i x_j \rangle$, which we can deduce as follows. To reach the state in which i is susceptible and j is infected in an SI model it must be the case that both i and j are susceptible to begin with and then j becomes infected. Even though i and j are neighbors j cannot be infected by i, since i is not infected, so j must be infected by some other neighboring vertex k, which itself must therefore be infected. In our new notation, the probability for the configuration in which i and j are susceptible and k is infected is $\langle s_i s_j x_k \rangle$. If we have this configuration, then j will become infected via k with rate β. Summing over all neighbors k except for i, the total rate at which j becomes infected is then $\beta \sum_{k(\neq i)} A_{jk} \langle s_i s_j x_k \rangle$.

Unfortunately, this is not the end of the story because $\langle s_i x_j \rangle$ can also *decrease*—it decreases if i becomes infected. This can happen in two different ways. Either i can be infected by its infected neighbor j, which happens with rate $\beta \langle s_i x_j \rangle$, or it can be infected by another neighbor $l \neq j$ that happens to be infected, which happens with rate $\beta \langle x_l s_i x_j \rangle$. Summing the latter expression over all neighbors l other than j gives a total rate of $\beta \sum_{l(\neq j)} A_{il} \langle x_l s_i x_j \rangle$.

Putting all of these terms together, with minus signs for those that decrease the probability, we get a final equation for $\langle s_i x_j \rangle$ thus:

$$\frac{\mathrm{d}\langle s_i x_j \rangle}{\mathrm{d}t} = \beta \sum_{k(\neq i)} A_{jk} \langle s_i s_j x_k \rangle - \beta \sum_{l(\neq j)} A_{il} \langle x_l s_i x_j \rangle - \beta \langle s_i x_j \rangle. \tag{17.45}$$

In theory this equation will now allow us to calculate $\langle s_i x_j \rangle$. In practice, however, it involves yet more terms that we don't know on the right-hand side, the three-variable averages $\langle s_i s_j x_k \rangle$ and $\langle x_l s_i x_j \rangle$. We can write down further equations for these averages but, as you can no doubt guess, those equations involve still higher-order (four-variable) terms, and so forth. The succession of equations will never end—in the jargon of mathematics, it doesn't *close*—and so looks as though it will be of no use to us.[9]

In fact, however, we can still make progress by approximating our three-variable averages with appropriate combinations of one- and two-variable averages, which allows us to close the equations and get a set we can actually solve. This process is called *moment closure* and the method described in this section is called a *moment closure method*. The moment closure method at the

[9] On a finite network with n vertices the equations will in fact close once we get all the way up to combinations of n variables, but this limit is not useful in practice as the equations will become unmanageably numerous and complicated long before we reach it.

level of two-variable averages that we discuss here is also called a *pair approximation method*.

In fact, our first attempt at writing equations for the SI model on a network, Eq. (17.34), was itself a simple moment-closure method. We approximated the true equation, Eq. (17.44), by writing $\langle s_i x_j \rangle \simeq \langle s_i \rangle \langle x_j \rangle$, closing the equations at the level of one-variable averages. By going a step further and closing at the pair approximation level of two-variable averages, we can make our equations more precise because we will be taking two-variable correlations into account. In fact, as we will see, this "second-order" moment closure approach is exact for some networks, although only approximate for others. Even in the latter case, however, the method gives a remarkably good approximation. The approximation can be further improved by going to third order, but the equations rapidly become complicated and researchers have rarely used moment closure methods beyond the second-order, pair approximation level.

The pair approximation is relatively straightforward however. Starting with Eq. (17.45) our goal is to approximate the three-variable averages on the right-hand side with lower-order ones. We do this by making use of Bayes theorem for probabilities thus:

$$\langle s_i s_j x_k \rangle = P(i,j \in S, k \in I) = P(i,j \in S) P(k \in I | i,j \in S), \tag{17.46}$$

where $P(i \in S)$ means the probability that vertex i is in the set S of susceptible vertices. We know that i and j are neighbors in the network and that j and k are neighbors, and our approximation involves assuming that the disease state of k doesn't depend on the disease state of i. This is a good approximation—indeed not an approximation at all—if the only path in the network from i to k is through j. In that case, given that we know j to be susceptible, there is no way that the disease state of i can affect that of k because there is no other path by which the disease could spread from i to k. On the other hand, if there is another path from i to k that avoids vertex j then the disease can spread along that path, which will introduce correlations between i and k and in that case our approximation is just that—an approximation—although as we will see it may be a very good one.

Assuming the state of k to be independent of the state of i, we have

$$P(k \in I | i, j \in S) = P(k \in I | j \in S) = \frac{P(j \in S, k \in I)}{P(j \in S)} = \frac{\langle s_j x_k \rangle}{\langle s_j \rangle}, \tag{17.47}$$

where we have used Bayes theorem again in the second equality. Putting Eqs. (17.46) and (17.47) together, we then have

$$\langle s_i s_j x_k \rangle = \frac{\langle s_i s_j \rangle \langle s_j x_k \rangle}{\langle s_j \rangle}. \tag{17.48}$$

We can write a similar expression for the other three-variable average appearing in Eq. (17.45):

$$\langle x_l s_i x_j \rangle = \frac{\langle x_l s_i \rangle \langle s_i x_j \rangle}{\langle s_i \rangle}, \tag{17.49}$$

and, substituting both into Eq. (17.45), we then get the pair approximation equation

$$\frac{\mathrm{d}\langle s_i x_j \rangle}{\mathrm{d}t} = \beta \frac{\langle s_i s_j \rangle}{\langle s_j \rangle} \sum_{k(\neq i)} A_{jk} \langle s_j x_k \rangle - \beta \frac{\langle s_i x_j \rangle}{\langle s_i \rangle} \sum_{l(\neq j)} A_{il} \langle s_i x_l \rangle - \beta \langle s_i x_j \rangle. \tag{17.50}$$

This equation now contains only averages over two variables at a time. It does also contain a new average $\langle s_i s_j \rangle$ that we have not encountered before, but this can easily be rewritten as $\langle s_i s_j \rangle = \langle s_i (1 - x_j) \rangle = \langle s_i \rangle - \langle s_i x_j \rangle$ and so our equation becomes

$$\frac{\mathrm{d}\langle s_i x_j \rangle}{\mathrm{d}t} = \beta \frac{\langle s_i \rangle - \langle s_i x_j \rangle}{\langle s_j \rangle} \sum_{k(\neq i)} A_{jk} \langle s_j x_k \rangle - \beta \frac{\langle s_i x_j \rangle}{\langle s_i \rangle} \sum_{l(\neq j)} A_{il} \langle s_i x_l \rangle - \beta \langle s_i x_j \rangle. \tag{17.51}$$

This equation is more complex than Eq. (17.34) but it can be simplified by rewriting it as follows. We define p_{ij} to be the conditional probability that j is infected given that i is not:

$$p_{ij} = P(j \in I | i \in S) = \frac{P(i \in S, j \in I)}{P(i \in S)} = \frac{\langle s_i x_j \rangle}{\langle s_i \rangle}. \tag{17.52}$$

Then the time evolution of p_{ij} is given by

$$\begin{aligned}
\frac{\mathrm{d}p_{ij}}{\mathrm{d}t} &= \frac{\mathrm{d}}{\mathrm{d}t}\left(\frac{\langle s_i x_j \rangle}{\langle s_i \rangle} \right) \\
&= \frac{1}{\langle s_i \rangle} \frac{\mathrm{d}\langle s_i x_j \rangle}{\mathrm{d}t} - \frac{\langle s_i x_j \rangle}{\langle s_i \rangle^2} \frac{\mathrm{d}\langle s_i \rangle}{\mathrm{d}t} \\
&= \beta \left(1 - \frac{\langle s_i x_j \rangle}{\langle s_i \rangle} \right) \sum_{k(\neq i)} A_{jk} \frac{\langle s_j x_k \rangle}{\langle s_j \rangle} - \beta \frac{\langle s_i x_j \rangle}{\langle s_i \rangle} \sum_{l(\neq j)} A_{il} \frac{\langle s_i x_l \rangle}{\langle s_i \rangle} \\
&\quad - \beta \frac{\langle s_i x_j \rangle}{\langle s_i \rangle} + \beta \frac{\langle s_i x_j \rangle}{\langle s_i \rangle} \sum_l A_{il} \frac{\langle s_i x_l \rangle}{\langle s_i \rangle} \\
&= \beta (1 - p_{ij}) \sum_{k(\neq i)} A_{jk} p_{jk} - \beta p_{ij} \sum_{l(\neq j)} A_{il} p_{il} - \beta p_{ij} + \beta p_{ij} \sum_l A_{il} p_{il},
\end{aligned} \tag{17.53}$$

where we have used Eqs. (17.44) and (17.51) in the third line. All but one of the terms in the two sums over l now cancel out, leaving us with the relatively simple equation

$$\frac{\mathrm{d}p_{ij}}{\mathrm{d}t} = \beta (1 - p_{ij}) \left[-p_{ij} + \sum_{k(\neq i)} A_{jk} p_{jk} \right], \tag{17.54}$$

where we have used the fact that $A_{ij} = 1$ (since i and j are neighbors). We can also rewrite Eq. (17.44) in terms of p_{ij} thus:

$$\frac{\mathrm{d}\langle s_i\rangle}{\mathrm{d}t} = -\beta\langle s_i\rangle \sum_j A_{ij} p_{ij}, \tag{17.55}$$

which has the solution

$$\langle s_i(t)\rangle = \langle s_i(0)\rangle \exp\biggl(-\beta \sum_j A_{ij} \int_0^t p_{ij}(t')\,\mathrm{d}t'\biggr). \tag{17.56}$$

Between them, Eqs. (17.54) and (17.56) now give us our solution for the evolution of the epidemic. Note that there are two equations of the form (17.54) for each edge in the network, since p_{ij} is not symmetric in i and j.

Figure 17.5a shows results from a numerical solution of these equations (the curve marked "second-order"), again on a configuration model network and, as the figure shows, the calculation now agrees very well with the simulation results represented by the dots in the figure. By accounting for correlations between adjacent vertices we have created a much more accurate theory.

This near-perfect agreement, however, is something of a special case. Configuration model networks are locally tree-like, meaning they have no short loops, and, as discussed above, our second-order moment closure approximation is exact when non-adjacent vertices i and k have only a single path between them through some intermediate j. When there are no short loops in our network this is true to an excellent approximation—the only other way to get from i to k in such a network is by going around a long loop and the length of such loops dilutes any resulting correlations between the states of i and k, often to the point where they can be ignored. The network used in the simulations for Fig. 17.5a was sufficiently large (a million vertices) and the resulting loops sufficiently long that the pair approximation equations are an excellent approximation, which is why the agreement is so good in the figure.

Unfortunately, as we saw in Section 7.9, most real social networks have a lot of short loops, which raises the question of how well our method does on such networks. Figure 17.5b shows a comparison between the predictions of our equations and direct simulations for a network with many short loops,[10] for both the simple first-order moment closure, Eq. (17.34), and for our more sophisticated second-order approach. As the plot shows, the first-order calculation agrees quite poorly with the simulations, its predictions being inaccurate enough to be of little use in this case. The second-order equations, however,

[10]The network was generated using the clustered network model of Ref. [240].

still do remarkably well. Their predictions are not in perfect agreement with the simulations, but they are close.

Thus the pair approximation method offers a significant improvement on networks both with and without short loops, providing a usefully accurate approximation in the former case and being essentially exact in the latter.

17.10.2 DEGREE-BASED APPROXIMATION FOR THE SI MODEL

The analysis of the previous section gives exact equations for the dynamics of the SI model on a network with few short loops and an excellent approximation in other cases. Unfortunately those equations cannot in general be solved analytically, even for simple networks such as those of the configuration model. The solutions presented in Fig. 17.5 were derived by integrating the equations numerically.

In this section we describe an alternative approximate approach that gives good, though not perfect, results in practice and produces equations that can be solved analytically. Moreover, the method can, as we will see, be generalized to other epidemic models such as the SIR model. The method was pioneered by Pastor-Satorras and coworkers [32, 33, 263, 264], though it has precursors in earlier work by May and others [199, 212]. It takes its simplest form when applied to networks drawn from the configuration model and so it is on this model that we focus here, although in principle the method can be extended to other networks.

Consider a disease propagating on a configuration model network, i.e., a random graph with a given degree distribution p_k, as discussed in Chapter 13. As before we focus on outbreaks taking place in the giant component of the network, this being the case of most interest—outbreaks in small components by definition die out quickly and do not give rise to epidemics.

An important point to notice is that the degree distribution of vertices in the giant component of a configuration model network is not the same as the degree distribution of vertices in the network as a whole. As shown in Section 13.8, the probability of a vertex of degree k belonging to the giant component goes up with vertex degree. This means that the degree distribution of vertices in the giant component is skewed towards higher degrees. (For a start, notice that there are trivially no vertices of degree zero in the giant component, since by definition such vertices are not attached to any others.) We will, as before, denote the degree distribution and the excess degree distribution in our calculations by p_k and q_k, but bear in mind that these are for vertices in the giant component, which means they are not the same as the distributions for the network as a whole.

The approximation introduced by Pastor-Satorras *et al.* was to assume that all vertices of the same degree have the same probability of infection at any given time. Certainly this *is* an approximation. The probability of infection of a vertex of degree, say, five situated in the middle of the dense core of a network will presumably be larger than the probability for a vertex of degree five that is out on the periphery. Nonetheless, if the distribution of probabilities for vertices of given degree is relatively narrow it may be a good approximation to set them all equal to the same value. And in practice, as we have said, the approximation appears to work very well.

Returning, for the sake of simplicity, to our earlier notation style, let us define $s_k(t)$ and $x_k(t)$ to be the probabilities that a vertex with degree k is susceptible or infected, respectively, at time t. Now consider a susceptible vertex A. To become infected, A has to contract the infection from one of its network neighbors. The probability that a particular neighbor B is infected depends on the neighbor's degree, but we must be careful. By hypothesis vertex A is not infected and so B cannot have caught the disease from A. If B is infected it must have caught the disease from one of its remaining neighbors. In effect this reduces the degree of B by one—B will have the same probability of being infected at the current time as the average vertex with degree one less. To put that another way, B's probability of infection depends upon its excess degree, the number of edges it has other than the edge we followed from A to reach it. B's probability of infection is thus x_k, but where k indicates the excess degree, not the total degree.

The advantage of the degree-based approach now becomes clear: the probability of B being infected depends, in this approach, only on B's excess degree and not on A's degree. By contrast, the conditional probability p_{ij} in our earlier formalism was a function of two indices, making the equations more complicated. To derive the equations for the degree-based approximation, consider the probability that vertex A becomes infected between times t and $t + \mathrm{d}t$. To become infected it must catch the disease from one of its neighbors, meaning that neighbor must be infected. The probability of a neighbor being infected is x_k where k is the excess degree of the neighbor, and the excess degree is distributed according to the distribution q_k of Eq. (13.46), which means that the average probability that the neighbor is infected is

$$v(t) = \sum_{k=0}^{\infty} q_k x_k(t). \tag{17.57}$$

If the neighbor is infected then the probability that the disease will be transmitted to vertex A in the given time interval is $\beta\,\mathrm{d}t$. Then the total probability of transmission from a single neighbor during the time interval is $\beta v(t)\,\mathrm{d}t$ and

the probability of transmission from any neighbor is $\beta k v(t)\,\mathrm{d}t$, where k is now the number of A's neighbors. In addition we also require that A itself be susceptible, which happens with probability $s_k(t)$, so our final probability that A becomes infected is $\beta k v s_k\,\mathrm{d}t$. Thus the rate of change of s_k is given by

$$\frac{\mathrm{d}s_k}{\mathrm{d}t} = -\beta k v s_k. \tag{17.58}$$

This equation can be solved exactly. We can formally integrate it thus:

$$s_k(t) = s_0 \exp\biggl(-\beta k \int_0^t v(t')\,\mathrm{d}t'\biggr), \tag{17.59}$$

where we have fixed the integration constant so that all vertices have probability s_0 of being susceptible at $t = 0$. Although we don't yet know the form of the function $v(t)$ this expression tells us that s_k depends on k as a simple power of some universal k-independent function $u(t)$:

$$s_k(t) = s_0[u(t)]^k, \tag{17.60}$$

where in this case

$$u(t) = \exp\biggl(-\beta \int_0^t v(t')\,\mathrm{d}t'\biggr). \tag{17.61}$$

Writing $x_k = 1 - s_k$ and substituting into Eq. (17.57) we then get

$$v(t) = \sum_{k=0}^{\infty} q_k(1 - s_k) = \sum_{k=0}^{\infty} q_k(1 - s_0 u^k) = 1 - s_0 g_1(u), \tag{17.62}$$

where $g_1(u)$ is the generating function for q_k and we have made use of $\sum_k q_k = 1$. Substituting Eq. (17.60) into Eq. (17.58) then gives us

$$\frac{\mathrm{d}u}{\mathrm{d}t} = -\beta u v = -\beta u\bigl[1 - s_0 g_1(u)\bigr]. \tag{17.63}$$

This is a straightforward linear differential equation for u that, given the degree distribution, can be solved by direct integration.

Finally, to calculate the total fraction $x(t)$ of infected individuals in the network we average over k thus:

$$x(t) = \sum_{k=1}^{\infty} p_k x_k(t) = \sum_{k=1}^{\infty} p_k(1 - s_0 u^k) = 1 - s_0 g_0(u). \tag{17.64}$$

Notice that the sums here start at $k = 1$ because there are no vertices of degree zero in the giant component.

Equations (17.63) and (17.64) between them give us an approximate solution for the SI model on the giant component of a configuration model network with any degree distribution.

Although the solution is elegant in principle, in most practical cases we cannot integrate Eq. (17.63) in closed form. Even without completing the integral, however, we can already see the basic form of the solution. First of all, at time $t = 0$ we have $u = 1$ by Eq. (17.61). Since $v(t)$ is, by definition, positive and non-decreasing with time, the same equation also implies that $u(t)$ always decreases and tends to zero as $t \to \infty$. This implies that at long times Eq. (17.63) becomes

$$\frac{\mathrm{d}u}{\mathrm{d}t} = -\beta u\bigl[1 - s_0 g_1(0)\bigr] = -\beta u(1 - s_0 p_1/\langle k\rangle), \tag{17.65}$$

and hence $u(t)$ decays exponentially as $\mathrm{e}^{-\beta(1-s_0 p_1/\langle k\rangle)t}$. Assuming the infection starts with only one or a handful of cases, so that $s_0 = 1 - c/n$ for some constant c, we have $s_0 \to 1$ in the limit of large n and

$$u(t) \sim \mathrm{e}^{-\beta(1-p_1/\langle k\rangle)t}. \tag{17.66}$$

Note that the long-time behavior is dictated by the fraction p_1 of vertices with total degree one. This is because these are the last vertices to be infected—individuals with only one contact are best protected from infection, although even they are guaranteed to become infected in the end. In networks where the fraction p_1 is zero or very small we have $u(t) \sim \mathrm{e}^{-\beta t}$ and the functional form of the long-time behavior depends only on the infection rate and not on the network structure.

At short times we can write $u = 1 - \epsilon$ and to leading order in ϵ Eq. (17.63) becomes

$$\frac{\mathrm{d}\epsilon}{\mathrm{d}t} = \beta\bigl[x_0 + (g_1'(1) - 1)\epsilon\bigr], \tag{17.67}$$

where $x_0 = 1 - s_0$ is the initial value of x_k. This has solution

$$\epsilon(t) = \frac{\beta x_0}{g_1'(1) - 1}\bigl[\mathrm{e}^{\beta(g_1'(1)-1)t} - 1\bigr], \tag{17.68}$$

where we have made use of the initial condition $\epsilon = 0$. Equivalently we can write[11]

$$u(t) = 1 - \epsilon = 1 - \frac{\beta x_0}{g_1'(1) - 1}\bigl[\mathrm{e}^{\beta(g_1'(1)-1)t} - 1\bigr]. \tag{17.69}$$

[11] This equation will diverge if $g_1'(1) = 1$. However, since we are performing the calculation on the giant component of the network, and since the giant component only exists if $g_1'(1) > 1$—see Section 13.8—we can safely rule out this possibility.

Given the short- and long-time behavior and the fact that $u(t)$ is monotonically decreasing, we can now guess that $u(t)$ has a form something like Fig. 17.6. Then, since g_0 is a monotonically increasing function of its argument, $x(t)$ in Eq. (17.64) has a similar shape but turned upside down, so that it looks qualitatively similar to the curve for the fully mixed version of the model shown in Fig. 17.1, although quantitatively it may be different.

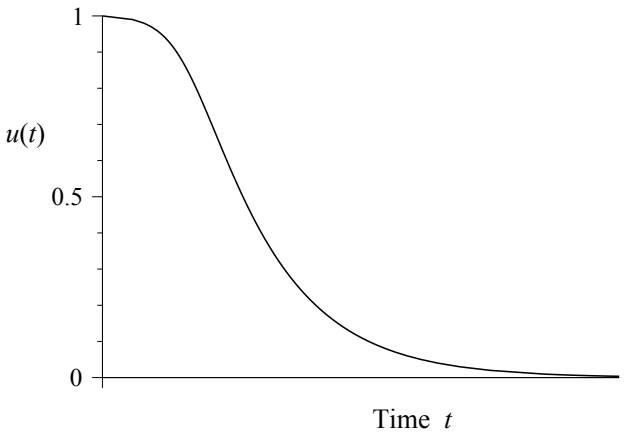

Figure 17.6: The function $u(t)$ in the solution of the SI model. Generically we expect $u(t)$ to have the form sketched here: it is monotonically decreasing from an initial value of 1 and has an exponential tail at long times.

The initial growth of $x(t)$ can be calculated by putting $u = 1 - \epsilon$ in Eq. (17.64) to give $g_0(1-\epsilon) \simeq 1 - g_0'(1)\epsilon$ and

$$\begin{aligned} x(t) &= 1 - s_0 + s_0 g_0'(1)\epsilon \\ &= x_0\left[1 + \frac{\beta g_0'(1)}{g_1'(1)-1}\left[e^{\beta(g_1'(1)-1)t} - 1\right]\right], \end{aligned} \tag{17.70}$$

where we have again set $s_0 = 1$. Thus, as we would expect, the initial growth of infection is roughly exponential.

The appearance of $g_1'(1)$ in Eq. (17.70) is of interest. As we saw in Eq. (13.68), $g_1'(1)$ is equal to the ratio c_2/c_1 of the average number of second neighbors to first neighbors of a vertex and hence is a measure of how fast the network branches as we move away from the vertex where the disease first starts. It should be not surprising therefore (though it's still satisfying) to see that this same quantity—along with the transmission rate β—controls the rate at which the disease spreads in our SI model.

Another interesting feature of the model is the behavior of the quantities $s_k(t)$ that measure the probability that a vertex of a given degree is susceptible. Since these quantities are all proportional to powers of $u(t)$—see Eq. (17.60)—they form a family of curves as shown in Fig. 17.7. Thus, as we might expect, the vertices with highest degree are the ones that become infected first, on average, while those with low degree hold out longer.

17.11 TIME-DEPENDENT PROPERTIES OF THE SIR MODEL

It is relatively straightforward to extend the techniques of Section 17.10 to the more complex (and interesting) SIR model. Again we concentrate on outbreaks taking place in the giant component of the network and we define s_i, x_i, and r_i to be the probabilities that vertex i is susceptible, infected, or recovered respectively. The evolution of s_i is (approximately) governed by the same equation

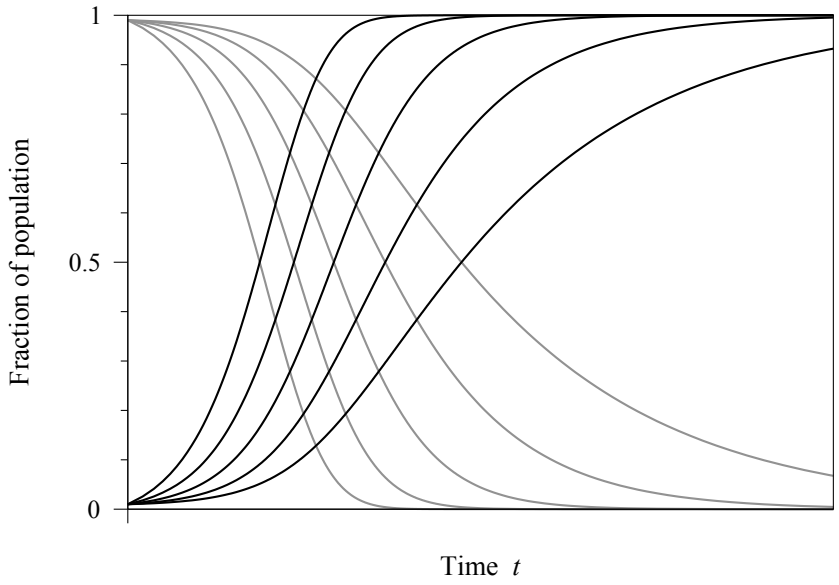

Figure 17.7: Fractions of susceptible and infected vertices of various degrees in the SI model. The various curves show the fraction of vertices of degree k that are susceptible (gray) and infected (black) as a function of time for $k = 1$, 2, 4, 8, and 16. The highest values of k give the fastest changing (leftmost) curves and the lowest values the slowest changing. The curves were calculated by integrating Eq. (17.63) numerically with $\beta = 1$ and a Poisson degree distribution with mean degree four.

as before:

$$\frac{\mathrm{d}s_i}{\mathrm{d}t} = -\beta s_i \sum_j A_{ij} x_j, \tag{17.71}$$

while x_i and r_i obey

$$\frac{\mathrm{d}x_i}{\mathrm{d}t} = \beta s_i \sum_j A_{ij} x_j - \gamma x_i, \tag{17.72}$$

$$\frac{\mathrm{d}r_i}{\mathrm{d}t} = \gamma x_i, \tag{17.73}$$

where, as previously, γ is the recovery rate, i.e., the probability per unit time that an infected individual will recover.[12]

[12]This contrasts with the approach we took in Section 17.8 where all vertices remained infected for the same amount of time and then recovered. Thus the model studied in this section is not exactly the same as that of Section 17.8, being more similar to the traditional SIR model of Section 17.3. We will see some minor consequences of this difference shortly.

We can choose the initial conditions in various ways, but let us here make the same assumption as we did for the SI model, that at $t = 0$ we have a small number c of infected individuals and everyone else is susceptible, so that $s_i(0) = 1 - c/n$, $x_i(0) = c/n$, and $r_i(0) = 0$.

As with the SI model we cannot solve these equations exactly, but we can extract some useful results by examining their behavior at early times. In the limit $t \to 0$, x_i is small and $s_i = 1 - c/n$, which tends to 1 as n becomes large, so Eq. (17.72) can be approximated as

$$\frac{\mathrm{d}x_i}{\mathrm{d}t} = \beta \sum_j A_{ij} x_j - \gamma x_i = \sum_j (\beta A_{ij} - \gamma \delta_{ij}) x_j, \tag{17.74}$$

where δ_{ij} is the Kronecker delta. This can be written in matrix form as

$$\frac{\mathrm{d}\mathbf{x}}{\mathrm{d}t} = \beta \mathbf{M} \mathbf{x}, \tag{17.75}$$

where $\mathbf{M}$ is the $n \times n$ symmetric matrix

$$\mathbf{M} = \mathbf{A} - \frac{\gamma}{\beta} \mathbf{I}. \tag{17.76}$$

As before we can write $\mathbf{x}$ as a linear combination of eigenvectors, though they are now eigenvectors of $\mathbf{M}$ rather than of the simple adjacency matrix as in the case of the SI model. But now we notice a useful thing: since $\mathbf{M}$ differs from the adjacency matrix only by a multiple of the identity matrix, it has the same eigenvectors $\mathbf{v}_r$ as the adjacency matrix:

$$\mathbf{M}\mathbf{v}_r = \mathbf{A}\mathbf{v}_r - \frac{\gamma}{\beta}\mathbf{I}\mathbf{v}_r = \left(\kappa - \frac{\gamma}{\beta}\right)\mathbf{v}_r. \tag{17.77}$$

Only the eigenvalue has been shifted downward by γ/β.

The equivalent of Eq. (17.42) is now

$$\mathbf{x}(t) = \sum_{r=1}^{n} a_r(0) \mathbf{v}_r \mathrm{e}^{(\beta\kappa_r - \gamma)t}. \tag{17.78}$$

Note that the exponential constant now depends on $\beta\kappa_r - \gamma$ and so is a function not only of the adjacency matrix and the infection rate but also of the recovery rate, as we would expect—the faster people recover from infection the less chance they have to spread the disease and the slower it will spread.

Again the fastest growing term is that corresponding to the most positive eigenvalue κ_1 of the adjacency matrix and individuals having the highest eigenvector centrality get infected first. Note, however, that it is now possible for γ to be sufficiently large that the exponential constant in the leading

term becomes negative, meaning that the term decays exponentially rather than grows. And if the leading term decays, so necessarily do all other terms, and so the total number of infected individuals will decay over time and the disease will die out without causing an epidemic.

The point at which this happens is the epidemic threshold for our model and it occurs at $\beta\kappa_1 - \gamma = 0$, or equivalently

$$\frac{\beta}{\gamma} = \frac{1}{\kappa_1}. \tag{17.79}$$

Thus the position of the epidemic threshold depends on the leading eigenvalue of the adjacency matrix. If the leading eigenvalue is small, then the probability of infection β must be large, or the recovery rate γ small, for the disease to spread. In other words a small value of κ_1 makes it harder for the disease to spread and a large value easier. This makes intuitive sense, since large values of κ_1 correspond to denser adjacency matrices and smaller values to sparser ones.

As in the case of the SI model, Eqs. (17.71–17.73) are only approximate, because they neglect correlations between the states of adjacent vertices. And as before we can allow for these correlations by using a pair approximation, but here we take a different approach and consider instead the equivalent of the methods of Section 17.10.2 for the SIR model.[13]

17.11.1 DEGREE-BASED APPROXIMATION FOR THE SIR MODEL

As with the SI model, let us make the approximation that all vertices with the same degree behave in the same way. Again we concentrate on the example of the configuration model [229] and on outbreaks taking place in the giant component of the network. We define $s_k(t)$, $x_k(t)$, and $r_k(t)$ to be the probabilities that a vertex with degree k is susceptible, infected, or recovered, respectively, at time t. Then we consider the state of a vertex B that is the neighbor of a susceptible vertex A. For such a vertex to be infected it must have contracted the disease from one of its neighbors other than A, since A is susceptible. That means, as before, that B's probability of being infected is given by x_k, but with k equal to the excess degree, which is one less than the total degree. And the

[13] We can see that the approach of this section cannot be exactly correct from the behavior of Eq. (17.79) on very sparse networks. On a vanishingly sparse network, with only a very few edges and no giant component, κ_1 becomes very small, though still non-zero. On such a network Eq. (17.79) implies that we could, nonetheless, have an epidemic if β is very large or γ very small. Clearly this is nonsense—there can be no epidemic in a network with no giant component. Thus the equation cannot be exactly correct.

probability that B is recovered depends only on the probability that it was previously infected, which is given by r_k where k is the excess degree, and the probability s_k of being susceptible can be derived from $s_k + x_k + r_k = 1$.

Armed with these observations, we can now write down an appropriate set of equations for the epidemic. The rate at which the probability of being susceptible decreases is given by the same equation as before, Eq. (17.58):

$$\frac{\mathrm{d}s_k}{\mathrm{d}t} = -\beta k v s_k, \tag{17.80}$$

where $v(t)$ is the average probability that a neighbor is infected:

$$v(t) = \sum_{k=0}^{\infty} q_k x_k(t), \tag{17.81}$$

and the equations for x_k and r_k are

$$\frac{\mathrm{d}x_k}{\mathrm{d}t} = \beta k v s_k - \gamma x_k, \tag{17.82}$$

$$\frac{\mathrm{d}r_k}{\mathrm{d}t} = \gamma x_k. \tag{17.83}$$

We can solve these equations exactly by a combination of the methods of Sections 17.3 and 17.10. We define the average probability that a neighbor is recovered thus:

$$w(t) = \sum_{k=0}^{\infty} q_k r_k(t). \tag{17.84}$$

Then, using Eqs. (17.81) and (17.83), we find

$$\frac{\mathrm{d}w}{\mathrm{d}t} = \sum_{k=0}^{\infty} q_k \frac{\mathrm{d}r_k}{\mathrm{d}t} = \gamma \sum_{k=0}^{\infty} q_k x_k = \gamma v, \tag{17.85}$$

which we use to eliminate v from Eq. (17.80), giving

$$\frac{\mathrm{d}s_k}{\mathrm{d}t} = -\frac{\beta}{\gamma} k \frac{\mathrm{d}w}{\mathrm{d}t} s_k \,. \tag{17.86}$$

This equation can be integrated to give

$$s_k = s_0 \exp\left(-\frac{\beta}{\gamma} k w\right), \tag{17.87}$$

where we have fixed the constant of integration so that at $t = 0$ all vertices have the same probability s_0 of being susceptible and there are no recovered vertices ($w = 0$).

Equation (17.87) implies that s_k is again proportional to a power of a universal function:

$$s_k(t) = s_0\big[u(t)\big]^k, \tag{17.88}$$

where in this case

$$u(t) = \mathrm{e}^{-\beta w/\gamma}. \tag{17.89}$$

Then, using Eq. (17.87), we find

$$\begin{aligned} v(t) &= \sum_k q_k x_k = \sum_k q_k(1 - r_k - s_k) = 1 - w(t) - s_0 \sum_k q_k u^k \\ &= 1 + \frac{\gamma}{\beta} \ln u - s_0 g_1(u), \end{aligned} \tag{17.90}$$

and Eq. (17.85) becomes

$$\frac{\mathrm{d}u}{\mathrm{d}t} = -\beta u \left[1 + \frac{\gamma}{\beta} \ln u - s_0 g_1(u)\right]. \tag{17.91}$$

This is the equivalent for the SIR model of Eq. (17.63), and indeed differs from that equation only by the new term in $\ln u$ on the right-hand side.

As before, Eq. (17.91) is a first-order linear differential equation in u and hence can, in principle, be solved by direct integration, although for any given degree distribution the integral may not have a closed-form solution. Once we have $u(t)$ the probability s_k of a vertex being susceptible is given by Eq. (17.88), or we can write the total fraction of susceptibles as

$$s(t) = \sum_k p_k s_k = s_0 \sum_k p_k u^k = s_0 g_0(u). \tag{17.92}$$

Solving for x_k and r_k requires a little further work but with perseverance it can be achieved.[14] Figure 17.8 shows the equivalent of Fig. 17.7 for vertices of a range of degrees. As we can see, the solution has the expected form, with the number of infected individuals rising, peaking, then dropping off as the system evolves to a final state in which some fraction of the population is recovered from the disease and some fraction has never caught it (and never will). Among vertices of different degrees the number infected goes up sharply with degree, as we would expect.

Even in cases where the integral in Eq. (17.91) cannot be performed, our solution can still shed light on features of the epidemic. Consider for example

[14] We observe that

$$\frac{\mathrm{d}}{\mathrm{d}t}\left(\mathrm{e}^{\gamma t} x_k\right) = \mathrm{e}^{\gamma t}\left(\frac{\mathrm{d}x_k}{\mathrm{d}t} + \gamma x_k\right) = \mathrm{e}^{\gamma t} \beta k v s_k,$$

where we've used Eq. (17.82) in the second equality. Integrating and using Eqs. (17.81) and (17.88),

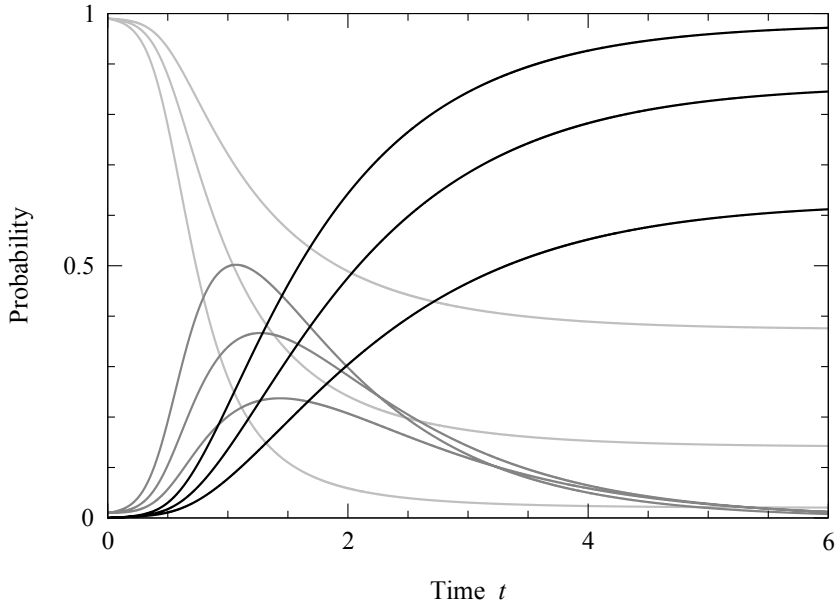

Figure 17.8: Fractions of susceptible, infected, and recovered vertices of various degrees in the SI model. The fraction of vertices of degree k that are susceptible (light gray), infected (darker gray), and recovered (black) as a function of time for $k = 1$, 2, 4 and $\beta = \gamma = 1$ on a network with an exponential degree distribution (Eq. (13.129)) with $\lambda = 0.2$. The highest values of k give the fastest growing numbers of infected and recovered vertices and the lowest values the slowest growing.

the long-time behavior. In the limit of long time we expect that the number of infected individuals will vanish leaving some individuals recovered and some who have never caught the disease. At $t = \infty$ the total fraction $r(t)$ of recovered individuals measures the overall size of the outbreak of the disease and is given by

$$r(\infty) = 1 - s(\infty) = 1 - g_0(u(\infty)). \tag{17.93}$$

where we have set $s_0 = 1$ as before on the assumption that the system is large and the number of initially infected individuals small.

We can find the stationary value of u by setting $\mathrm{d}u/\mathrm{d}t = 0$ in Eq. (17.91) to

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we then have

$$x_k(t) = \mathrm{e}^{-\gamma t}\biggl[x_0 + \beta k s_0 \int_0^t \mathrm{e}^{\gamma t'}[u(t')]^k \Bigl(1 + \frac{\gamma}{\beta}\ln u(t') - s_0 g_1(u(t'))\Bigr)\,\mathrm{d}t'\biggr],$$

and $r_k = 1 - s_k - x_k$.

give

$$1 + \frac{\gamma}{\beta} \ln u - g_1(u) = 0. \tag{17.94}$$

In the special case where the outbreak is small, so that the final value of u is close to 1, we can expand $\ln u = \ln[1 + (u-1)] \simeq u - 1$ and Eq. (17.94) becomes

$$u \simeq 1 - \frac{\beta}{\gamma} + \frac{\beta}{\gamma} g_1(u). \tag{17.95}$$

Equations (17.93) and (17.95) are similar in form to Eqs. (17.27) and (17.28) which give the final size of the outbreak in our treatment of the SIR model using percolation theory. The reason why Eq. (17.95) is only approximate in the present case where Eq. (17.28) was exact is that the model treated in this section is slightly different from the one treated earlier, having (as discussed in footnote 12 on page 662) a constant probability γ per unit time of recovery from disease for each infected individual as opposed to a fixed infection time for the model of Section 17.8.1.

We can also examine the early-time behavior of the outbreak by looking at the behavior of Eq. (17.91) close to $u = 1$. Writing $u = 1 - \epsilon$ and keeping terms to leading order in ϵ we get

$$\frac{\mathrm{d}\epsilon}{\mathrm{d}t} = \bigl[\beta g_1'(1) - \gamma\bigr]\epsilon, \tag{17.96}$$

assuming $s_0 = 1$ again, which means that

$$u(t) = 1 - \epsilon(t) = 1 - \mathrm{e}^{(\beta g_1'(1) - \gamma)t}. \tag{17.97}$$

This is similar to Eq. (17.69) for the SI model, except for the inclusion of the term in γ. The fraction of susceptible degree-k vertices is given by

$$s_k(t) = u^k = \bigl[1 - \mathrm{e}^{(\beta g_1'(1) - \gamma)t}\bigr]^k \simeq 1 - \mathrm{e}^{k(\beta g_1'(1) - \gamma)t}, \tag{17.98}$$

and total cases of the disease, infected and recovered, which is just $1 - s_k$, grows exponentially as $\mathrm{e}^{k[\beta g_1'(1) - \gamma]t}$.

The epidemic threshold for the model is the line that separates an initially growing number of cases of the disease from an initially decreasing one and is given in this case by the point at which the exponential constant in Eq. (17.98) equals zero, which gives

$$\frac{\beta}{\gamma} = \frac{1}{g_1'(1)}. \tag{17.99}$$

This result is similar in form to Eq. (17.79) for the epidemic threshold on a general network,[15] but with the leading eigenvalue of the adjacency matrix κ_1 replaced with $g_1'(1)$. It also looks similar to Eq. (17.29) for the percolation threshold for bond percolation, but this similarity is somewhat deceptive. In fact, the result most nearly corresponding to this one in the percolation treatment is Eq. (17.30). If we equate our recovery rate γ with the reciprocal of the infectiousness time τ in that previous treatment, then the two are roughly equivalent when the epidemic threshold is low, meaning either that β is small or that γ is large. If the threshold is higher then the match between the two models is poorer, which is again a result of the fact that the models are defined in slightly different ways.

17.12 TIME-DEPENDENT PROPERTIES OF THE SIS MODEL

It is straightforward to extend our methods to the SIS model also. By analogy with Eqs. (17.71–17.73) we have

$$\frac{\mathrm{d}s_i}{\mathrm{d}t} = -\beta s_i \sum_j A_{ij} x_j + \gamma x_i, \tag{17.100a}$$

$$\frac{\mathrm{d}x_i}{\mathrm{d}t} = \beta s_i \sum_j A_{ij} x_j - \gamma x_i \tag{17.100b}$$

for the SIS model. Caveats similar to those for previous models apply here: these equations ignore correlations between the states of adjacent vertices and hence are only an approximation.

Equations (17.100a) and (17.100b) are not independent since $s_i + x_i = 1$, so only one is needed to form a solution. Taking the second and eliminating s_i we get

$$\frac{\mathrm{d}x_i}{\mathrm{d}t} = \beta(1 - x_i) \sum_j A_{ij} x_j - \gamma x_i. \tag{17.101}$$

At early times, assuming as before that $x_i(0) = x_0 = 1 - c/n$ for all i and constant c, we can drop terms at quadratic order in small quantities to get

$$\frac{\mathrm{d}x_i}{\mathrm{d}t} = \beta \sum_j A_{ij} x_j - \gamma x_i, \tag{17.102}$$

which is identical to Eq. (17.74) for the SIR model at early times. Hence we can immediately conclude that the early-time behavior of the model is the same,

[15] And like Eq. (17.79) it is also clearly wrong on sparse networks for the same reasons—see footnote 13 on page 664.

with initially exponential growth and an epidemic threshold given by

$$\frac{\beta}{\gamma} = \frac{1}{\kappa_1}. \tag{17.103}$$

(See Eq. (17.79).) Also as in the SIR model the probability of infection of a given vertex at early times will be proportional to the vertex's eigenvector centrality.

At late times we expect the probability of infection to settle to a constant endemic level, which we can calculate by setting $\mathrm{d}x_i/\mathrm{d}t = 0$ in Eq. (17.101) and rearranging, to give

$$x_i = \frac{\sum_j A_{ij} x_j}{\gamma/\beta + \sum_j A_{ij} x_j}. \tag{17.104}$$

Typically we cannot derive a closed-form solution for x_i from this expression, but we can solve it numerically by iteration starting from a random initial guess. We can also see the general form the solution will take by considering limiting cases. If β/γ is large, meaning that we are well above the epidemic threshold given in Eq. (17.103), then we can ignore the term γ/β in the denominator and $x_i \simeq 1$ for all i, meaning that essentially all vertices will be infected all the time. This makes good sense since if β/γ is large then the rate of infection is very high while the rate of recovery is negligible.

Conversely, if β/γ is only just above the epidemic threshold level set by Eq. (17.103) then x_i will be small—the disease only just manages to stay alive—and we can ignore the sum in the denominator of Eq. (17.104) so that

$$x_i \simeq \frac{\beta}{\gamma} \sum_j A_{ij} x_j, \tag{17.105}$$

or

$$\kappa_1 x_i \simeq \sum_j A_{ij} x_j, \tag{17.106}$$

where we have used Eq. (17.103). This implies that x_i is proportional to the leading eigenvector of the adjacency matrix or, equivalently, proportional to the eigenvector centrality. (Note that this is at late times so this result is distinct from the finding above that x_i is proportional to eigenvector centrality at early times.)

Thus the long-time endemic disease behavior of the SIS model varies from a regime just above the epidemic threshold in which the probability of a vertex being infected is proportional to its eigenvector centrality, to a regime well above the threshold in which essentially every vertex is infected at all times.

17.12.1 DEGREE-BASED APPROXIMATION FOR THE SIS MODEL

We can also write down approximate equations for the evolution of the SIS model in which, as in Sections 17.10.2 and 17.11.1, we assume that the probability of infection is the same for all vertices with a given degree. Focusing once again on configuration model networks, the equivalent of Eqs. (17.80–17.82) is

$$\frac{\mathrm{d}s_k}{\mathrm{d}t} = -\beta k v s_k + \gamma x_k, \tag{17.107a}$$

$$\frac{\mathrm{d}x_k}{\mathrm{d}t} = \beta k v s_k - \gamma x_k, \tag{17.107b}$$

where the variables s_k and x_k are as before, and again

$$v(t) = \sum_{k=0}^{\infty} q_k x_k(t). \tag{17.108}$$

As before Eqs. (17.107a) and (17.107b) are not independent and only one is need to form a solution. Let us take the second and rewrite it using $s_k = 1 - x_k$ to give

$$\frac{\mathrm{d}x_k}{\mathrm{d}t} = \beta k v(1 - x_k) - \gamma x_k. \tag{17.109}$$

Unfortunately, there is no known complete solution to this equation but we can once again find its behavior at early and late times.

Assuming, as previously, that our epidemic starts off with only a single case or a small number of cases, the probability x_k of being infected at early times is c/n for constant c and hence small in the limit of large n. Dropping terms of second order in small quantities then gives us the linear equation

$$\frac{\mathrm{d}x_k}{\mathrm{d}t} = \beta k v - \gamma x_k, \tag{17.110}$$

which can be rewritten using an integrating factor to read

$$\frac{\mathrm{d}}{\mathrm{d}t}\left(\mathrm{e}^{\gamma t} x_k\right) = \mathrm{e}^{\gamma t}\frac{\mathrm{d}x_k}{\mathrm{d}t} + \gamma \mathrm{e}^{\gamma t} x_k = \beta k \mathrm{e}^{\gamma t} v, \tag{17.111}$$

and hence integrated to give

$$x_k(y) = \beta k \mathrm{e}^{-\gamma t} \int_0^t \mathrm{e}^{\gamma t'} v(t')\,\mathrm{d}t'. \tag{17.112}$$

Thus $x_k(t)$ for short times takes the form

$$x_k = k u(t), \tag{17.113}$$

where $u(t)$ is some universal, k-independent function. Substituting into Eqs. (17.108) and (17.110), we then have

$$v(t) = u(t) \sum_{k=0} k q_k = g_1'(1) u(t), \tag{17.114}$$

and

$$\frac{\mathrm{d}u}{\mathrm{d}t} = \bigl[\beta g_1'(1) - \gamma\bigr] u(t). \tag{17.115}$$

Thus we have exponential growth or decay of the epidemic at early times, with the epidemic threshold separating the two falling at the point where $\beta g_1'(1) - \gamma = 0$, or

$$\frac{\beta}{\gamma} = \frac{1}{g_1'(1)}, \tag{17.116}$$

just as for the SIR model (see Eq. (17.99)).

At late times the disease to settles down into an endemic state in which some constant fraction of the population is infected. We can solve for this endemic state by setting $\mathrm{d}x_k/\mathrm{d}t = 0$ for all k in Eq. (17.109) to give

$$x_k = \frac{kv}{kv + \gamma/\beta}. \tag{17.117}$$

Substituting this expression into Eq. (17.108), we then find that

$$\sum_{k=0}^{\infty} \frac{k q_k}{kv + \gamma/\beta} = 1. \tag{17.118}$$

In general there is no closed-form solution to this implicit equation for v, although it can typically be solved numerically for any given q_k, and given the value we can then get x_k from Eq. (17.117).

What we can tell from Eq. (17.118) is that, given the degree distribution, v at late times is a function solely of β/γ (or γ/β if you prefer) and hence x_k is solely a function of β/γ and k. Moreover, in order for Eq. (17.118) to be satisfied v must be an increasing function of β/γ—as β gets larger or γ smaller, v must increase in order to keep the sum in the equation equal to one. This means that x_k will also be an increasing function of β/γ. (Equation (17.117) implies that it is an increasing function of k as well.) Thus the equations give us a qualitative picture of the behavior of the SIS model, although quantitative details require a numerical solution.

We have in this chapter only brushed the surface of what is possible in the modeling of epidemics spreading across networks. We can extend our studies

to more complicated network structures, such as networks with degree correlations, networks with transitivity, networks with community structure, and even epidemics on empirically observed networks. More complicated models of the spread of infection are also possible, such as the SIRS model mentioned in Section 17.5, as well as models that incorporate birth, death, or geographic movement of individuals [17, 156]. In recent years, scientists have developed extremely sophisticated computer models of disease spread using complex simulations of the behavior patterns of human populations, including models of entire cities down to the level of individual people, cars, and buildings [110], and models of the international spread of disease that incorporate detailed data on the flight patterns and timetables of international airlines [79]. These developments, however, are beyond the scope of our necessarily brief treatment in this chapter.

PROBLEMS

17.1 Consider an SIR epidemic on a configuration model network with exponential degree distribution $p_k = (1 - \mathrm{e}^{-\lambda})\mathrm{e}^{-\lambda k}$.

a) Using the results of Section 16.2.1 write down an expression for the probability u appearing in Eq. (17.27) in terms of ϕ and λ.

b) Hence find an expression for the probability that a vertex is infected by the disease if it has degree k.

c) Evaluate this probability for the case $\lambda = 1$ and $\phi = 0.9$, for $k = 0$, 1, and 10.

17.2 Consider the spread of an SIR-type disease on a network in which some fraction of the individuals have been vaccinated against the disease. We can model this situation using a joint site/bond percolation model in which a fraction ϕ_s of the vertices are occupied, to represent the vertices not vaccinated, and a fraction ϕ_b of the edges are occupied to represent the edges along which contact takes place.

a) Show that the fraction S of individuals infected in the limit of long time is given by the solution of the equations

$$S = \phi_s[1 - g_0(u)], \qquad u = 1 - \phi_s\phi_b + \phi_s\phi_b g_1(u),$$

where $g_0(z)$ and $g_1(z)$ are the generating functions for the degree distribution and excess degree distribution, as usual.

b) Show that for a given probability of contact ϕ_b the fraction of individuals that need to be vaccinated to prevent spread of the disease is $1 - 1/[\phi_b g_1'(1)]$.

17.3 We have been concerned in this chapter primarily with *epidemic* disease outbreaks, meaning outbreaks that affect a finite fraction of all individuals in a network. Consider, by contrast, a small SIR outbreak—an outbreak that corresponds to one of the non-giant percolation clusters in the bond percolation approach of Section 17.8—occurring on a configuration model network with degree distribution p_k.

a) What is the probability of such an outbreak occurring if the disease starts at a vertex chosen uniformly at random from the whole network (including vertices both within and outside the giant component)?

b) Show that if the probability of transmission along an edge is ϕ then the generating function $h_0(z)$ for the probability π_s that the outbreak has size s is given by the equations

$$h_0(z) = z g_0(h_1(z)), \qquad h_1(z) = 1 - \phi + \phi z g_1(h_1(z)),$$

where $g_0(z)$ and $g_1(z)$ are the generating functions for the degree distribution and excess degree distribution respectively.

c) What is the mean size of such an outbreak?

17.4 Consider an SI-type epidemic spreading on the giant component of a k-regular random graph, i.e., a configuration model network in which all vertices have the same degree k. Assume that some number c of vertices, chosen at random, are infected at time $t = 0$.

a) Show using the results of Section 17.10 that the probability of infection of every vertex increases at short times as $e^{\beta k t}$.

b) Show that within the first-order moment closure approximation of Eq. (17.35) the average probability of infection x of every vertex is the same and give the differential equation it satisfies.

c) Hence show that

$$x(t) = \frac{c e^{\beta k t}}{n - c + c e^{\beta k t}}.$$

d) Find the time at which the "inflection point" of the epidemic occurs, the point at which the rate of appearance of new disease cases stops increasing and starts decreasing.

17.5 Consider a configuration model network containing vertices of degrees 1, 2, and 3 only, such that the fractions of vertices of each degree in the giant component are $p_1 = 0.3$, $p_2 = 0.3$, and $p_3 = 0.4$.

a) Find an expression for the excess-degree generating function $g_1(z)$ appearing in Eq. (17.63).

b) Hence, by solving Eq. (17.63), find an expression for t as a function of u for an SI epidemic on the giant component of the network, assuming that $s_0 \simeq 1$, and with initial condition $u(0) = 1 - \epsilon$, where ϵ is small.

c) Show that in the limit of long times the number of susceptibles falls off in proportion to $e^{-21\beta t/2}$.

17.6 Consider the spread of an SIR-type disease in a network in which some fraction of the individuals have been vaccinated against the disease. We can model this situation using a joint site/bond percolation model in which a fraction ϕ_s of the vertices are occupied, to represent the vertices not vaccinated, and a fraction ϕ_b of the edges are occupied to represent the edges along which contact takes place.'

a) Show that the fraction S of individuals infected in the limit of long time is given by the solution of the equations

$$S = \phi_s[1 - g_0(u)], \qquad u = 1 - \phi_s\phi_b + \phi_s\phi_b g_1(u),$$

where $g_0(z)$ and $g_1(z)$ are the generating functions for the degree distribution and excess degree distribution, as usual.

b) Show that for a given probability of contact ϕ_b the fraction of individuals that need to be vaccinated to prevent spread of the disease is $1 - 1/[\phi_b g_1'(1)]$.