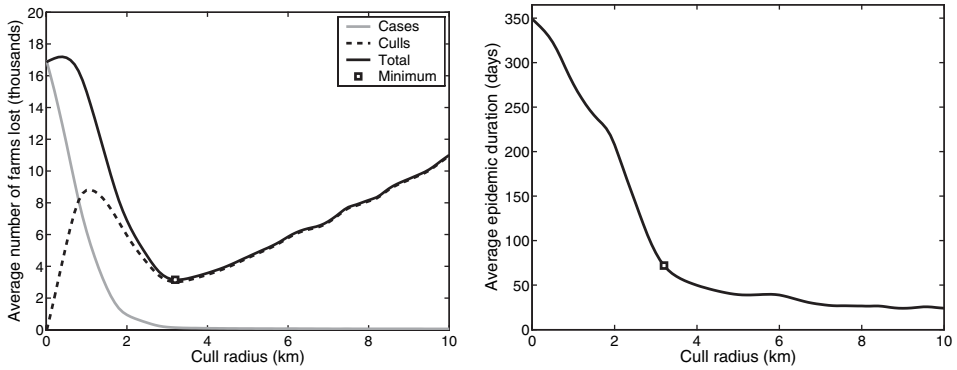


**Figure 7.17.** The simulated impact of ring-culling using the individual-based foot-and-mouth disease model, equation (7.18); with all other parameters as in Figure 7.16. The left-hand figure gives the average total loss of farms during an epidemic, composed of both reported infections and farms removed as part of a ring cull. There is a clear trade-off between minimizing the number of cases and not losing too many farms as part of the cull. For the same simulations the right-hand graph gives the average duration of the epidemic, with the cull size that minimized loss of farms marked. Results are from 1,250 simulations per radius value considered, and are smoothed using a local spline. Simulation results kindly provided by M. J. Tildesley.

## 7.6. NETWORKS

Networks provide a unified way to think about the interaction between individuals or populations, and are especially useful when each individual is in direct contact with only a small proportion of the population (Garnett and Anderson 1996; Morris 1997; Dunyak et al. 1998; Potterat et al. 1999; Klovdahl 2001; Rothenberg 2001; Sander et al. 2002; Potterat et al. 2002; Halloran et al. 2002; Liljeros et al. 2003; Rothenberg 2003; Szendroi and Csanyi 2004; Doherty et al. 2005; Keeling and Eames 2005). Networks tend to be very powerful tools for understanding the transmission of infection in human populations due to either social contacts (for airborne infections) or sexual contacts (for sexually transmitted diseases). In either case we expect that each individual will be in contact with only a small proportion of the population, and that the number of contacts will be highly heterogeneous—networks provide a simple means of capturing such interactions. We therefore see that the primary advantage of network models is their ability to capture complex individual-level structure in a simple framework.

To specify all the connections within a network, we can form a matrix from all the interaction strengths $\rho_{ij}$, which we expect to be sparse with the majority of values being zero. Usually, for simplicity, two individuals (or populations) are either assumed to be connected with a fixed interaction strength or unconnected (and therefore have an interaction strength of zero). In such cases, the network of contacts is specified by a graph matrix $\boldsymbol{G}$, where $\boldsymbol{G}_{ij}$ is 1 if individuals $i$ and $j$ are connected, or 0 otherwise. For the remainder of this section we will exclusively consider such networks where all the interaction strengths are identical; understanding networks with variable strength connections remains a challenge for the future (Sander et al. 2002). When $\boldsymbol{G}$ is symmetric ($\boldsymbol{G}_{ij} = \boldsymbol{G}_{ji}$) we define the network as undirected and infection can pass in both directions across a contact—this is the standard assumption for the vast majority of infectious diseases. However, there are a few special cases where a network is directed and infection

can pass only one way across a contact; examples of such directional transmission include infections transmitted through blood products, infections of livestock transmitted through artifical insemination, and transmission to populations (e.g., farms) through the movement of individuals (livestock).

**Networks provide a robust means to consider the individual nature of disease transmission. Two individuals are linked if they have sufficient contact to allow the infection to pass between them.**

**A matrix $G$ can be used to completely specify the network, indexing all possible transmission links between individuals.**

### 7.6.1. Network Types

Several types of networks are commonly used within the epidemiological literature (as well as by statistical physicists). Although many theoretical approaches to networks use the terms nodes and edges, we will generally refer to individuals and contacts. Below we describe the basic nature of these networks in terms of a few fundamental properties: the way the network is constructed, the heterogeneity in the number of contacts, the clustering of contacts, and the average path length (in terms of the number of steps it takes on average to link two randomly chosen individuals). The five common network types discussed below are illustrated in Figure 7.18.

#### *7.6.1.1. Random Networks*

Random networks ignore the actual spatial position of individuals and, as the name suggests, connections are formed at random (Islam et al. 1996; Andersson 1998; Diekmann et al. 1998; Newman et al. 2002; Neal 2003). In one of the most analytically tractable versions of the random network, each individual has the same number of contacts. The random network is therefore characterized by a lack of heterogeneity in the number of contacts and a lack of clustering. The average path length in a random network is low because a large number of "long-distance" contacts exist, effectively spanning the population. A range of analytical techniques can be applied to understanding the dynamics of diseases spreading through such networks (Diekmann et al. 1998; Keeling 1999); of greatest importance, these models show that the initial growth rate of a disease in a network is reduced compared to the random-mixing equivalent:

$$\text{Initial random network growth rate} = \tau(\overline{n} - 2),$$

$$\text{Initial random-mixing growth rate} = \beta = \tau\overline{n},$$

where $\tau$ is the transmission rate across a contact and $\overline{n}$ is the average number of contacts (or effective number of contacts in the random-mixing model). This reduction is due to the development of strong negative correlations between susceptible and infected individuals during the early phase of the epidemic.

#### *7.6.1.2. Lattices*

As explained in Section 7.3, lattices are associated with a regular grid of contacts and each individual has a fixed number of contacts (usually either 4 or 8) (Bak et al. 1990;

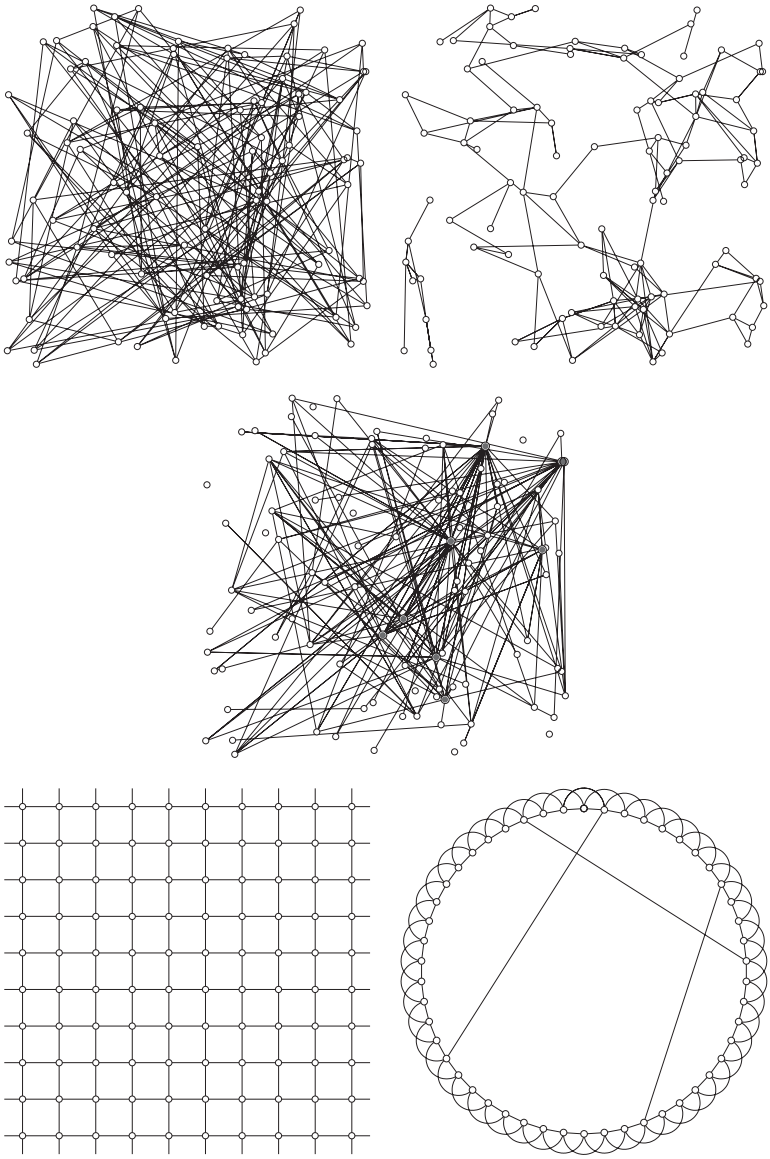

**Figure 7.18.** Five distinct network types containing 100 individuals. These are from left to right: Random, Spatial (top row ), Scale-free (middle row), Lattice, and Small-World (bottom row). The Random, Spatial, and Scale-Free networks all use the same position of individuals—although for the Random and Scale-Free network, the position of the individuals is irrelevant for forming connections. In all five graphs, the average number of contacts per individual is approximately 4. For the scale-free network, individuals with high numbers of contacts are shaded gray.

Rhodes and Anderson 1997). In contrast to random networks, lattices possess far stronger clustering because contacts are localized in space. This higher level of clustering further reduces the initial growth rate of a disease in a lattice compared to the random network, although exact analytical results are no longer available. Given that all the connections are local, the average path length is very long because the only way to transverse the lattice from one side to the other is by steps of a single grid size.

#### *7.6.1.3. Small World Networks*

Small world networks are based upon a lattice structure, with a small number of "long-range" connections added. Figure 7.18 shows the classical one-dimensional small-world model (Watts and Strogatz 1998) where each individual is connected to its four nearest neighbors together with three long-range contacts across the entire population. Locally (from the perspective of an individual), small world networks look very much like lattices; they are highly clustered and have little heterogeneity in the number of neighbours—therefore, transmission of infection is predominantly localized so that the strong saturation effects and wavelike spread observed in the lattice models still occur. However, the presence of the few long-range connections provides shortcuts across the network, vastly reducing the average path length and allowing a spreading infection to jump to new susceptible areas (Newman and Watts 1999; Moore and Newman 2000). In practice, it may be difficult to estimate the number of long-range contacts, but small-world networks have highlighted their profound importance for disease dynamics (Boots and Sasaki 1999; Kuperman and Abramson 2001).

#### *7.6.1.4. Spatial Networks*

Spatial networks are one of the most flexible forms of networks, and are related to the individual-based models discussed in Section 7.5. In spatial networks, a kernel is often used to calculate the probability of any two individuals being connected depending on the distance between them (Watts 1999; Read and Keeling 2003; Keeling 2005a). By changing the distribution of individuals and the connection kernel, it is possible to generate a wide variety of networks from highly clustered lattices to small world arrangements and globally connected random networks. Spatial networks generally show a reasonably high degree of heterogeneity, with the number of neighbors often being approximately Poisson distributed. In addition, when the connection kernel preferentially links nearby individuals, we can regain the spatial wavelike spread of infection that characterizes lattice models.

#### *7.6.1.5. Scale-Free Networks*

In the vast majority of networks that have been studied, the number of contacts per individual is very heterogeneous, with most individuals having a relatively small number and a few have many contacts (Albert et al. 1999; Barabási and Albert 1999; Jeong et al. 2000; Lilijeros et al. 2001). Because the most connected individuals are likely to be disproportionately important in disease transmission (see Chapter 3), networks that can capture this heterogeneity are therefore vital in understanding the spread of real infections—scale-free networks incorporate these heterogeneities. Scale-free networks are generally created dynamically, adding new individuals to a network one at a time with a connection mechanism that mimics the natural formation of social contacts. Each new

individual that is added to the population connects preferentially with individuals that already have a large number of contacts; in a social setting, this corresponds to everyone wanting to be friends of the most popular people. The resultant network has a power-law distribution for the probability of having a given number of contacts, $\mathbb{P}(\text{contacts} > n) \approx n^{-\alpha}$. This power-law property was first observed for the World Wide Web connections and has also been recorded in power grid networks and graphs of actor collaborations. The same type of heterogeneities are likely to be present in the social contacts that permit the spread of infection.

**Many different types of network structure are possible. These differ in the amount of heterogeneity, clustering, and average path length, thus reflecting the different transmission routes for various infections.**

### 7.6.2. Simulation of Epidemics on Networks

Networks have many similarities with individual-based spatial models (see Section 7.5 and Section 6.3.5), in that spatial interactions can be defined in terms of a kernel. However, in networks, contacts tend to be of equal strength and limited in number. This can be used to considerable advantage in simulations:

$$\begin{aligned} Rate(\text{Infected individual } j \text{ recovers}) &= \gamma, \\ Rate(\text{Susceptible individual } i \text{ infected}) &= \tau \times \text{number of infectious contacts} \\ &= \tau \sum_j G_{ji} I_j = \lambda_i, \end{aligned}$$

where $\tau$ is the rate of transmission across a contact and $I_j$ is one if individual $j$ is infectious or zero otherwise. One immediate implication of the network structure is that the force of infection $\lambda_i$ depends on the state of only a few individuals. This means that the force of infection does not need to be calculated anew at every iteration, providing huge computational savings. Instead, we can store the force of infection for each individual; when an individual first becomes infectious, the force of infection of all its contacts is increased by $\tau$, and when an individual recovers the force of infection for all its contacts is likewise increased by $\tau$. Hence, each event impinges on the state of only its neighborhood of contacts. Further computational savings can be achieved if the contacts of each individual are stored in a list (such that $C_1^i, C_2^i, \ldots C_{n_i}^i$ are the $n_i$ contacts of individual $i$) because loops and summations need to be over only the $n_i$ neighbors, which is generally much faster than summing over the total number of individuals. For example:

$$Rate(\text{Susceptible individual } i \text{ infected}) = \tau \sum_{j=1}^{n_i} I_{C_j^i}.$$

This is online program 7.7

Figure 7.19 shows examples of the epidemic dynamics on the network types illustrated in Figure 7.18, giving both the individual epidemic curves (gray lines) and the average of all major epidemics (black line). Clearly the random network (which in many ways is closest to the nonspatial mass-action models) generates the fastest epidemic growth rate and has the highest proportion of infectious individuals at the maximum. Surprisingly, there appears to be little difference between the dynamics of epidemics on the Spatial and Scale-Free networks; this may be attributable to the variance in the number of contacts that is present in both networks. For infinitely large population sizes, Scale-Free

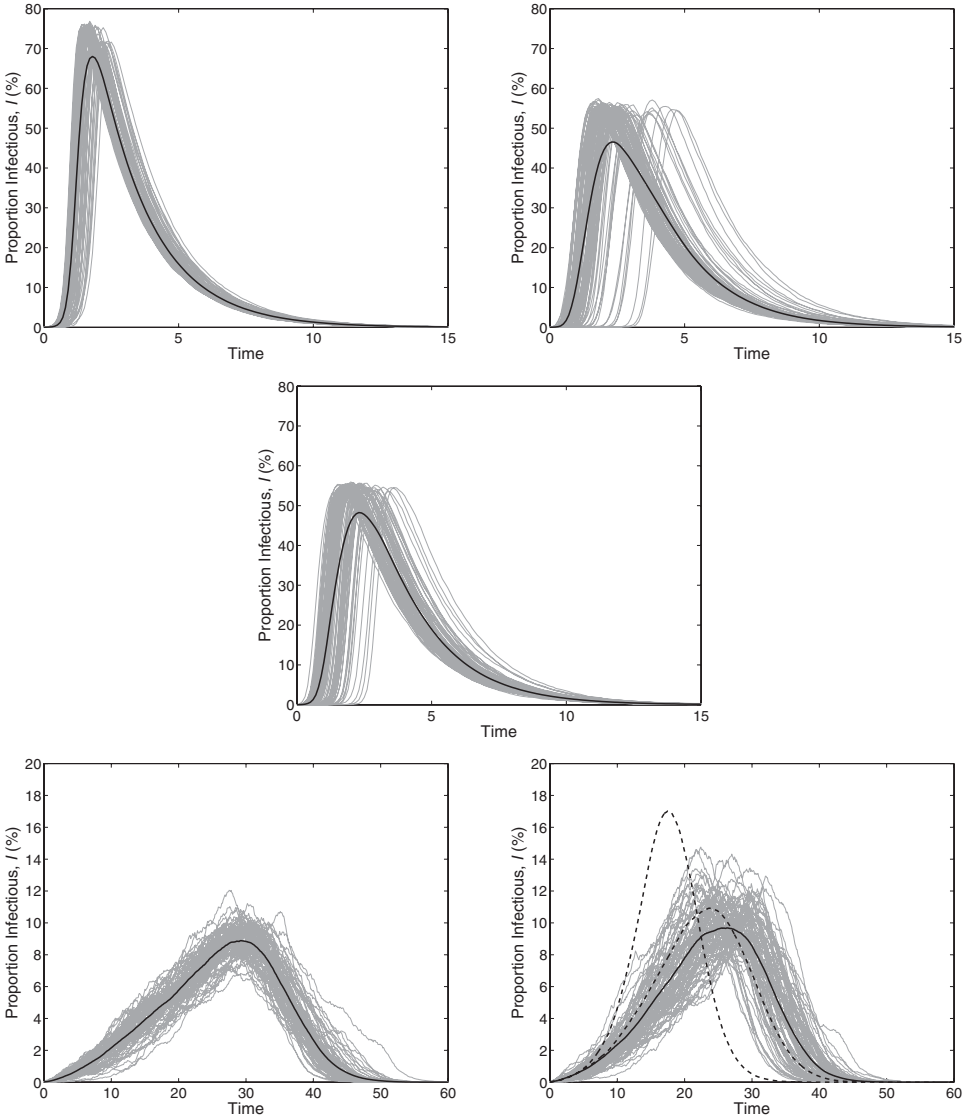


**Figure 7.19.** Typical epidemics of the five network types described above. These are from left to right: Random, Spatial (top row), Scale-Free (middle row), Lattice, and Small-World (bottom row). Each graph shows 100 epidemic curves (gray), together with the average for all major epidemics (black) for a single example of each network type—therefore, all variablity within each graph is due to the stochastic nature of transmission and not variation in the network. All five networks contain 10,000 individuals, although all individuals are not necessarily interconnected as part of a giant component. For the Spatial and Scale-Free networks, approximately 88% and 74% are part of the giant component and can therefore potentially become infected; for these networks, the proportion of infectious individuals have been rescaled as a fraction of the giant component. For all other types, the entire network is interconnected. In all networks, the average number of contacts per individual is approximately 4, although the Scale-Free network has considerable heterogeneity, with one individual having 85 contacts. For consistency, the Small-World network is formed from a two-dimensional lattice (Watts 1999) (not a one-dimensional circle as shown in Figure 7.18) with 10 additional random "long-range" contacts. The dashed lines show the effect on the mean epidemic of increasing the number of long-range contacts to 20 and 100. ($\tau = 1$, $\gamma = 0.5$.)