disease modeling, and may validate or refute the standard (pseudo) mass-action assumption in particular circumstances. Second, at a larger scale, the 2001 foot-and-mouth epidemic in the United Kingdom highlighted the difficulties with assessing the degree of spread between farms. In reality, this spatial spread is the combination of several factors (such as the movement of vehicles or wind-borne transmission), but due to the complexities of parameterization this spread was estimated and modeled as a single transmission kernel. Finally, metapopulation models—treating each town or city as a subpopulation—appear to be an ideal tool for modeling national epidemic patterns; however, it is difficult to assess the level of coupling between communities, especially when many of the links may be sporadic and social.

Spatial models will continue to be an area of high research activity for many years. The importance of local spatial interaction is only recently being appreciated, in terms of both understanding disease dynamics and local control of infection. We can expect to see spatial models increasingly used in public health scenarios, where control usually operates on a regional basis and where preventing infection reaching new populations is a key control aim.

7.10. SUMMARY

Seven different model formulations have been described in this chapter (Metapopulations, Coupled lattices, Cellular automata, Reaction-Diffusion, Integro-Differential, Individual-Based and Network), each with its own merits and disadvantages. The techniques needed to simulate these models and the data needed to parameterize them differ greatly; however, they share a common theme in that local interactions (transmission) generally dominate longer-range interactions, leading to a clustering of cases.

➤ The type of spatial model used is dependent on the host organism, our degree of knowledge about its behavior, and the scale we wish to consider.

➤ **Metapopulations** provide a powerful framework for modeling disease dynamics for hosts that can be naturally partitioned into spatial subunits.

➤ The force of infection within a subpopulation can be modeled as a weighted sum of the prevalence in all subpopulations.

➤ With stochastic metapopulations, the speed of the spread of infection between subpopulations is reduced compared to the equivalent deterministic model.

➤ For plants and other sessile hosts, coupling (or the strength of spatial interaction) generally decreases with distance, mimicking the effects of wind—or vector—dispersal. Adding an extra subpopulation generally increases R_0 because more pathogens can be intercepted by the additional hosts.

➤ Metapopulation models of animal diseases usually capture the transmission of infection by the permanent immigration and emigration of hosts. In these models, R_0 is generally independent of the coupling because each host transmits infection at a constant rate.

➤ The spread of human diseases in metapopulations is best captured by the rapid commuter movements of individuals from their home subpopulation to another subpopulation and back again—requiring us to model both the current location and home location of

individuals. When commuter movements are of short duration, this can be approximated by simple coupling. In these models, R_0 is independent of the coupling.

➤ The correlation between disease prevalence in two subpopulations increases sigmoidally with the strength of interaction between the populations. In general, the change from largely independent dynamics to synchrony occurs for interaction strengths from 10^{-3} to 0.1.

➤ Without interaction between the subpopulations, a spatially segregated metapopulation suffers a faster rate of stochastic extinction than its randomly mixed counter-part. When interaction between the subpopulations is included, the level of local (subpopulation-scale) and global (metapopulation-wide) extinctions is an emergent property of the dynamics and cannot be easily predicted from the parameters.

➤ **Levins' metapopulation** models ignore the internal dynamics within each subpopulation, and instead classify each subpopulation as either infected or disease free. Despite differences between the equilibrium-level results of Levins and full metapopulation models, the Levins model still remains a useful and simple tool for studying invasion dynamics.

➤ **Coupled lattice models** are specialized metapopulation models, where subpopulation are arranged on a grid and coupling is generally to the nearest neighbors only.

➤ The wave speed of an invading epidemic in a coupled-lattice model increases almost linearly with the initial growth rate of the infection, $\beta - \gamma - \nu$; increases nonlinearly with the level of coupling, ρ; and is slightly more rapid in deterministic compared to stochastic models.

➤ **Cellular automata** operate on a lattice of sites, with each site generally assumed to hold a single host. Interaction is usually stochastic and with the neighboring (four or eight) lattice sites.

➤ In many locally coupled spatial models (such as cellular automata), the depletion of the locally available susceptible population can reduce the early growth rate of the epidemic and the speed of the invading wave front.

➤ The forest-fire model typifies many stochastic spatial cellular automata models. The fact that transmission is faster than recovery, which is faster than births, which is faster than imports of infection, leads to power-law relationships for the frequency of epidemic sizes.

➤ **Reaction-diffusion models**, which use a PDE formulism, assume local transmission of infection and rely on spatial diffusion of hosts to spread the infection.

➤ For PDE models (in two dimensions with equal diffusion in all directions), infection spreads as a growing circular wave of near constant velocity.

➤ With **integro-differential equations**, the spatial spread of infection is via a transmission kernel that defines how transmission risk decays with distance. The shape of the tail of the transmission kernel determines the eventual spatial pattern of invasion, from wavelike spread, to scattered local foci, to highly probable extremely long-range jumps.

➤ **Individual-based models** account for the spatial interaction between individual hosts distributed on a spatial landscape. They can include a wide variety of complex (more

biologically realistic) behavior that often features a spatial component; this can lead to a huge number of parameters that can be difficult to determine from available data.

➤ **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.

➤ 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.

➤ In general, networks display slower epidemic dynamics compared to randomly mixed models. As a consequence, networks that are most like the random-mixing models—with short average-path length (Small-World, Random, and Scale-Free) and little clustering (Random and Scale-Free)—show the fastest epidemic growth rates for a given average number of contacts per individual.