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Five challenges for spatial epidemic models

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ABSTRACT

Infectious disease incidence data are increasingly available at the level of the individual and include high-resolution spatial components. Therefore, we are now better able to challenge models that explicitly represent space. Here, we consider five topics within spatial disease dynamics: the construction of network models; characterising threshold behaviour; modelling long-distance interactions; the appropriate scale for interventions; and the representation of population heterogeneity.

Introduction

There have been many important ecological and public health questions related to the transmission of infectious disease that neither need, nor would benefit from, a mechanistic model in which space is represented explicitly. In many instances, the concept of the average behaviour of a large population is sufficient to provide genuinely useful insight and to extract good information from the data that are available.

However, the importance of the spatial component of many transmission systems is being increasingly recognised. When there is a need to consider spatially heterogeneous interventions, it is clearly essential to represent the location of hosts and the pattern of transmission. Sometimes the location of the hosts in space is clearly defined and easily measured – such as for plant systems and some livestock systems. However, for humans and wild animals, the single location assigned to a host represents the best average from the complex social behaviour of each individual.

If it is thought that the aggregate characteristics of epidemic incidence are being driven by spatial aspects of transmission (such as waves), it is difficult to investigate data from these systems with models that do not represent space in some way. Also, and perhaps most importantly for future modelling work, where data are provided with high spatial resolution, even when the primary hypotheses of interest for a given phenomenon does not relate directly to spatial effects, it is often necessary to account for spatial processes in order to discount plausible alternate explanations for an observed feature in the data.

Mechanistic spatial models are usually described as being; an individual-based simulation, a metapopulation model or a network model. Individual-based models explicitly represent every individual host within a simulation algorithm and usually assume a highly variable – but non-zero – probability that any infectious host can infect any susceptible host. Metapopulation models do not represent individuals. Rather, they keep track of the number of individuals at different locations who are in each state of the natural history. Often, they also assume that each location (patch) is connected to all others, but, again, with highly variable strengths of connection. Network models typically define each node to be an individual host and assume that each host is connected to only a small subset of other hosts. Also, usually, the strengths of connection along each arc in a network epidemic model are assumed to be equal.

Here we consider five broad challenges for theoretical infectious disease dynamics.

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1. How can network models best be constructed to reflect spatial population structure?

The three types of spatial model outlined above do not form disjoint sets. We can think of the network formulation as a potential unifying framework within which the other two can be nested. Individual-based simulations are very dense fully connected networks with highly variable edge weights. Similarly, metapopulation models become network models as the average number of individuals represented in each patch approaches 1. Therefore, given that it has proven difficult to obtain analytical results for metapopulation models and individual-based simulations, it may be possible to make more analytical progress in our ability to describe complex spatial phenomena by basing analysis on network formulations that mimic these other model structures.

There is a long history of using regular lattices as a basis for infection spread (Mollison and Kuulasmaa, 1985), often in the context of plant populations. Random geometric graphs (Penrose, 2003) provide another, less highly structured, way to represent a spatial process by a simple graph. They are constructed by starting from a spatial Poisson point process, which need not necessarily be homogeneous. Pairs of points (nodes) that are within some critical distance are connected by an edge to form a graph, after which the underlying spatial structure is ignored. The conditional independence properties of Poisson processes mean that the analytic properties of such graphs are well understood. When they form the underlying contact structure for epidemic processes (Isham et al., 2011), random geometric graphs provide a nice way of escaping the lack of local correlation and clustering that are implicit properties of the configuration graphs often used to explore epidemic dynamics.

The spatial construction of the random geometric graph leads naturally to the question of how transmission is affected when the hosts move in space, so that edges are continuously broken and created. This scenario has direct application to computer viruses spreading on wifi computer/phone networks (e.g. Rhodes and Nekovee, 2008). In other applications, it may be appropriate to model the creation and annihilation of nodes and edges. Network dynamics is discussed in section “How do we define a threshold parameter for spatial models?” of the chapter on Networks (this volume).

In most metapopulation and network models, the group or network structure of the host population is fixed. The actual contacts between hosts in which transmission takes place are not explicitly represented; implicitly one might imagine some local spatial movement that brings the two hosts in contact. In contrast, in an alternative modelling approach, hosts move between a set of discrete spatial locations that form the nodes of a graph, and infection is only possible between hosts in the same location. Thus, in a simple model, hosts might perform independent random walks on the graph (Draief and Ganesh, 2011; Abdullah et al., 2011).

Work is needed to develop other network models that reflect spatial structure and, when that network is not fully connected, to explore how well the properties of an epidemic running on the network approximate the full spatial dynamics.

2. How should we model contact structure in spatially heterogeneous populations?

Human populations are never distributed uniformly in space. Hence, the movement of people to achieve their daily tasks in life is driven strongly by the distribution of population density around them. In rural areas, people must travel further on average to shop compared with urban areas; while they may travel less far to socialise. The movement of hosts is clearly an important feature of spatially explicit infectious disease models (Riley, 2007). It is also an important aspect of human behaviour for the study of other social phenomena: urbanisation, disaster planning, transport planning, and many others. There has been considerable interest in developing parsimonious models of human movement in recent years in order to support these different studies (González et al., 2008; Wang et al., 2009; Simini et al., 2012).

Most quantitative descriptions of human movement are based on the concept of a gravity model: that the flux of individuals from area dA_1 to area dA_2 is proportional to the product of the populations of the two areas n_1 and n_2 and inversely proportional to the distance between them r_1, raised to some power (Viboud et al., 2006). If the analogy with Newtonian gravity is direct, movement between areas is assumed to be proportional to $n_1 n_2 / r^2$. With only minor refinements, for some systems, this formulation describes observations extremely well. For example, the number of people travelling between Germany and 28 other European cities by air can be well estimated with simple gravity-based models (Grosche et al., 2007).

However, spatial models of infectious disease are often defined for an individual (as well as for linked metapopulations). Therefore flux models must be refined so as to be consistent with simulated infections between individuals. This is usually achieved by assuming that the infectious contacts of individuals are determined by a mobility kernel: the probability that an individual at location r_1 will make contact with an individual at location r_2. The kernel itself can be defined only up to a constant of proportionality, with the number of infection events determined by a separate parameter (Riley and Ferguson, 2006). Effectively, individual mobility becomes relative to available opportunities.

The discovery of flexible and accurate movement models is a current challenge for infectious disease dynamics, with high interest in the recently proposed radiation flux model. In the radiation model, the degree of flow between two populations is driven by their population sizes, the distance between them and also by the total number of people who live the same distance away from each population (or closer) (Simini et al., 2012). Thus, the intervening population absorbs journeys in the same way that radiation is absorbed as it passes through a media. Although the radiation model as currently proposed has no free parameters and is attractive in its simplicity, it is not yet clear to what degree previously proposed gravity-like mobility kernels can achieve similar or better fits to observed patterns by estimating two or three key parameters.

One obvious way forward is for the underlying movement assumptions of spatial models of infectious disease to be compared using spatially resolved social contact data (Read et al., 2014).

3. How do we define a threshold parameter for spatial models?

The basic reproductive number R_0 is most commonly understood to be the average number of infections generated by one infectious individual in an otherwise susceptible population. Therefore, for simple non-spatial homogeneous mixing models, the critical or threshold value of a straightforward R_0 parameter is unity: that is, when $R_0 \leq 1$, the expected outbreak size is small; when $R_0 > 1$, there is a significant probability of a large outbreak.

Where the population includes individuals of different infectious types, a more sophisticated approach defines R_0 as the largest eigenvalue λ_* of the next generation operator for those types (Diekmann and Heesterbeek, 2000; Heesterbeek, 2001). This is appropriate for most non-spatial models, for which branching process approximations can be applied (Ball, 1983; Davis et al., 2008), showing that early growth is exponential, with the nth generation of infectives $\propto \lambda_*^n$, and with infectious numbers of each type in