Modelling Space-Time Pattern in Infectious Disease under Environmental Constraints to Infer Underlying Transmission Processes: Initial Results from a Simulation Model

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1. Introduction

Dynamic simulation models have emerged as important geographical tools to support geographical research (Ballas *et al.*, 2005). Examples include micro-simulation (MS) models (Holm *et al.*, 2000) and agent-based models (Gimblett et al, 2002) which usually represent space as continuous and cellular automata (CA) (Sirakoulis et al 2000, Situngkir 2004) which represent space as discrete (in particular, a raster).

Within epidemiological studies, much data, particularly on number of cases, are acquired at health facilities (e.g., hospitals, clinics, dispensaries). Such data are often acquired without reference to geographical coordinates, and even where they are, disclosure and confidentiality restrictions prevent their full use. The geographical reference for a health facility is not a point (x, y), but a catchment defined using some two-dimensional function. Catchments are often not equally weighted spatially and further may overlap. Often, catchments may be approximated using mathematical functions such that space time distributions of disease rate can be estimated from health facility data. However, such estimates are regularised (convolved) by the spatially varying catchments such that it is difficult to make inferences about the underlying processes of transmission.

The emergent space-time pattern of disease in a given region depends on the parameters of both the disease transmission model and the spatial and social network structures in place in the environment in which transmission takes place. In particular, it is expected that changes in the parameters of the (simulation) model will lead to observable changes in the space-time pattern of disease. Simulation models provide an important means for evaluating the sensitivity of emergent patterns and their space-time character to changes in model parameters.

The above introduction leads to two possible avenues for exploitation of the spatio-temporal information in emergent and possibly aggregated disease patterns:

- (i) it is hypothesised that statistical models fitted to the space-time patterns of aggregated disease data can be used to infer parameters of the underlying disease transmission process.
- (ii) it is hypothesised that specific changes in the spatial environment and social network structures in which transmission occurs will provide explanations for variations in disease dynamics from place to place (e.g., town to town).

Demonstration of the above hypothesised linkages would have important implications for a range of applications. For example, recent emergent diseases (e.g., SARS, bird flu, biological agents) can pose serious hazards to human health, with little known about their transmission characteristics. In such circumstances, it is extremely important to characterise their transmission properties early in an outbreak in order to plan early warning and containment strategies. To date, little use has been made of spatio-temporal information in this regard.

If the association between particular elements of the environment and social network structure and disease outcomes can be quantified then it should be possible to map the vulnerability of settlements to specific diseases. For example, it is well-known that the behaviour characteristics of individuals can be modified to reduce the likelihood of disease transmission. However, spatial elements such as the effects of public v. state school education for children and settlement structure (e.g., out-of-town supermarket v. local shop) are less well studied. Again, such knowledge would be useful in terms of planning containment strategies.

In this paper, we characterise the space-time pattern of disease occurrence for two common diseases (flu, mumps) using the (spatial) variogram computed as regular intervals in time. This information on spatial pattern is added to the global pattern as represented by the common SLIR curves. We then explore the sensitivity of spatial statistics to changes in the parameters of disease transmission and settlement spatial and social structures.

2. Simulation Model

To simulate infectious disease transmission, a model was created in which space is essentially discrete (raster) although the model is not a CA in the strict sense. The model was run with a diurnal time-step. The model included three separate sets of parameters: disease transmission, environment and population parameters.

The parameters of the disease were selected based on the well-established SLIR disease evolution model (Figure 1). Specifically, a parameter range was established for each component of the SLIR model from the literature.



Figure 1. SLIR model (Chen, S., 2001)

To keep the model simple in the first instance, 100 by 100 residences were distributed evenly over the raster space of 600 by 600 pixels. M=25 schools were distributed using a stratified (5 by 5 cells, in this case) random sampling scheme. N companies were distributed randomly.

Population parameters included both the population structure and individual behaviour. The structure of each household was based on published statistical data (http://www.statistics.gov.uk). Two age groups were established (adults, children) with

different behaviours (Figure 2). During the day, children attended their nearest school according to non-overlapping catchments as represented by the Thiessen polygon structure (Figure 3). This structure is not dissimilar to the state secondary school system in England. During the day, adults attended either their nearest or a randomly selected company in equal numbers per household. During the evening, some interaction (transmission) between neighbours was allowed.



Figure 2. Schematic representation of the social network used in the simulation model.



Figure 3. Thiessen polygons representing school catchments.

From one simulation run the entire space-time data cube was extracted for further analysis.

3. Analysis

Figure 4 shows the plot of total number of cases against day number.



Figure 4. Number of infected individuals by day.

The information in Figure 4 is typically all that is used to characterise diseases. However, further spatio-temporal information may be gleaned from geostatistical and similar analyses of the space-time patterns of occurrences. Figure 5 shows three variograms obtained for days

(a) 65, (b) 149 and (c) 308 (c.f. Figure 4). It is clear that the character of spatial variation changes through time in subtle ways. In particular, on day 65 there are two scales of spatial variation (3 pixels, 11 pixels; corresponding to local interactions between neighbours and the effect of school catchments. These patterns are evident in adults as well as children. On day 149, the local pattern has been subsumed by the more dominant pattern attributable to school catchments, and by day 308, a larger scale trend has emerged which subsumes the pattern induced by the initial school catchment structure (e.g., day 149). In this paper, such changes are modelled using geostatistical variogram functions and the differences between such models as a function of changes in simulation model parameters are explored.



Figure 5 Variograms for days (a) 65, (b) 149 and (c) 308 showing some of the changes in spatial structure which occur through simulation runs.

4. Conclusion

Dynamic simulation models of infectious disease provide an important experimental environment for evaluating and quantifying the effects of changes in disease transmission probabilities as well as environmental and social parameters on space-time disease outcomes. In this paper, such models are used to explore the extent to which geostatistical modelling of the space-time pattern of disease occurrence can help in early characterisation and control of emerging diseases and in quantifying the effect of changing environmental and social parameters.

5. References

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