A Flexible Automata Model for Disease Simulation

Shih Ching Fu and George Milne
School of Computer Science and Software Engineering
The University of Western Australia
35 Stirling Highway, Crawley, 6009, WA, Australia
{scfu,george}@csse.uwa.edu.au

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Abstract. This paper presents an approach for capturing the behaviour of disease spread in a tractable model. More specifically, by embedding spatial population information into the cells of a cellular automaton, accurate representations of disease spread may be produced. Non-homogeneity is easily introduced into the implicitly discretized landscape of a cellular automaton, contributing to the accuracy of such models and overcoming some of the simplifying assumptions of homogeneity found in earlier models. The need to develop and test more effective disease containment measures inspires the search for new and more accurate models.

1 Introduction

As stated by Ferguson [1], mathematical modelling is the only way to analyse the effectiveness of different disease control strategies. Simulation is the obvious choice since real life experimentation is impractical. An epidemic spread model that provides life-like results and reconfigurable parameters is an invaluable tool for use in developing outbreak contingency plans. Given the costs of outbreaks as foot-and-mouth disease (FMD) in the United Kingdom [2, 3], it is desirable to know beforehand whether vaccination or culling interventions are worthwhile.

There have been four recent studies of the spread dynamics of the smallpox virus [1]. Each study set out to determine the best containment strategy for smallpox. Unfortunately, rather than providing a definitive answer, each study recommended a different policy for optimally controlling such outbreaks. This suggests that more research needs to be directed into the field of epidemic modelling and that there may be no general all-purpose containment strategy, rather distinct outbreaks need tailored policies. Virtual simulation of outbreaks therefore provide a practical means to discover and refine these policies. Of particular interest are disease spread models that capture host mobility, spatial population heterogeneity as well as disease biology.

Most existing epidemic models, particularly those using ordinary differential equations (ODEs) assume the host landscape is homogeneous [4]. That is, hosts are taken to be distributed evenly throughout the landscape and mixed in well with one another. This is a major oversight since hosts are rarely found to be

equidistantly spaced and unmoving. Partial differential equation models introduce diffusion terms to model heterogeneous mixing but still treat populations as continuous entities rather than comprising discrete interacting hosts. There is need for models which take into account the spatial conditions of a landscape to improve model accuracy. It is therefore claimed that by using a discretized approach, such as a cellular automaton, we can capture both the local interactions of hosts and the effects of a heterogeneous population landscape into a single more realistic disease spread model.

2 Our Model

The main focus of our model is to capture the effects of geography on an epidemic's emergent behaviour. Of particular interest is the uneven distribution of hosts over the landscape caused by topographic and demographic heterogeneity. In this section, we discuss how we have encoded this heterogeneity into an automaton's cells so as to build a model that more closely reflects nature. We have adopted the widely used SIR framework to describe the disease state of host individuals [5]. In this framework, hosts are designated susceptible, infective, or recovered depending on whether they are healthy, diseased, or immune respectively.

2.1 Cell definition

In contrast to traditional CA, our model allows each cell to accommodate a variable number of hosts rather than just one static host. This structure is similar to that found in lattice-gas cellular automata [6]. Consequently, the state of each cell is completely defined by its number of susceptible, infective, and recovered hosts. Each cell has another parameter, a maximum *carrying capacity*, which denotes the maximum number of hosts a cell can contain. The landscape is therefore discretized into equal-sized cells whose populations and carrying capacities may vary differentially from cell to cell. The local population density is therefore defined as a cell's population divided by its carrying capacity.

By encoding cell population densities into the cell definition we can use this information to modulate the rate of spread to neighbouring cells. Note that disease spread can be due to both direct disease transmission at cell boundaries and through increased infective host movement between cells. Our CA based model captures both disease and host dynamics.

As implied by the above description, the number of hosts in a particular cell can vary with time; the carrying capacity however, remains constant. Increases and decreases in local cell population which correspond to births, deaths, and movement of hosts over the landscape are also included in the model. Although it is possible to have differing population densities with a neighbourhood of cells, it is assumed that within each cell, the population is well-mixed. That is, during each time step, all the hosts sharing a particular cell will come into contact at least once.

The carrying capacity of a cell is used as a mechanism to limit the movement of hosts between cells. Overcrowding is prevented since the number of newborns per epoch, discussed later, is conditional on a cell not exceeding its carrying capacity. Although the effect of the land's carrying capacity is not directly enforced in nature, carrying capacities are a straightforward way to encourage or discourage the motion of individuals between cells.

The cells are tessellated in a square grid with straight, non-penetrable boundaries. These boundaries are likened to physical boundaries found in nature such as oceans or mountain ranges, or political boundaries over which hosts can 'immigrate' out of the landscape in question. The immigration and emigration of hosts is combined with the increases and decreases in host populations determined by birth and death probability parameters. Such births and deaths are completely independent of disease related parameters such as pathogen morbidity and affect susceptible, infective, and recovered hosts alike. It is assumed that there is no vertical *vectoring* of disease, that is, parents do not pass the disease directly to their offspring at birth.

Our model also uses two distinct neighbourhood radii for host and pathogen movement respectively; both are 8-connected Moore neighbourhoods. In our model, the two ways a cell can become contaminated are if an infective host moves in from an adjacent cell, or the pathogen, by its own spread mechanisms outside a carrier, infects a formerly susceptible host. In this way, we have modelled two kinds of heterogeneity: the spatial heterogeneity of the population due to host movement, and also the biological heterogeneity due to variations in disease presence among hosts.

2.2 Epidemic spread parameters

As discussed previously, there are two kinds of epidemic spread factors that we have incorporated into our model: those related to the disease pathogen, and those related to host demographics. Disease parameters include infectiousness, morbidity, and immunity; demographic parameters include population density and host movement. These parameters have featured separately in some earlier epidemic studies [7, 8], but we have aimed to capture several key aspects of disease spread into one composite model.

2.3 Cell update algorithm

Similar to the LGCA model proposed by Fukś and Lawniczak [6], the cell update algorithm is performed in two phases: the infection phase and the randomization phase. On a cell-by-cell basis, the encoded data relating to factors such as morbidity, mobility, contagiousness, and immunity is used to update the SIR characteristics of the CA lattice. Interleaved between the infection phases are randomization phases which model the movement of hosts between CA cells. Such movement is limited by the size of the CA interaction neighbourhood and cell carrying capacities. The interaction neighbourhood can be of any configuration; we have chosen the traditional 8-connected Moore neighbourhood. Specific

information about the interactions between cells, such as cyclic host movement, can be very easily encoded as different interaction neighbourhood [9]. Therefore in contrast to the uniform mixing found in ODE models, we can model localized host movement.

Currently, our model does not take into account other parameters such as latency or incubation time [10]. Latency is the lag between being infected and becoming infective, and incubation is the delay between becoming infected and becoming symptomatic. These times can be implemented by introducing further states, say 'E', to represent those hosts exposed and infected with the pathogen but not yet infective or transmissive. Including this or other states into our model is straightforward since such a change implemented by simply modifying the finite state automata populating the CA lattice.

3 Experimental Scenarios

In this section we describe two experimental scenarios and examine some of the results that our composite epidemic model produced under simulation. The purpose of these scenarios is to show how a cellular automata approach can be used to accurately simulate disease spread, needing only to define localized cell interactions. It is also interesting that due to the intrinsically graphical nature of cellular automata, no further processing of results is needed in order to visualize the resultant disease spread patterns.

3.1 Corridors of spread

This experiment tries to emulate a real life landscape with imaginary town centres and transport links. Towns and roads are human constructed features and attract high and patterned population densities. Of particular significance is that rather than having flat and uniform population density profiles, settlements around cultural features generally have a directed or linear shape.

In this scenario, each cell has been initialized to have a carrying capacity of 1000, with three 'towns' set to this maximum. Two of these towns, on in the northwest and another in the southeast, start with a 1:9 infective to susceptible ratio. There are also 'transport links' comprising a dense line of susceptibles running between each of the 'towns'. Cells on this line have an initial susceptible population of 100, whilst the cells on either side have an initial susceptible population of 75. The rest of the landscape comprises cells with 10 susceptibles in them. The town in the southwest corner contains 1000 susceptibles and no infectives.

Parameter settings The primary aim of this scenario is to highlight the fact that disease spread generally occurs faster in regions of high host density. Consequently, to isolate the effects of the heterogeneous landscape, host movement is turned off for this scenario. This is done to preserve the population density profile for the duration of this experiment. The probability of transmission between

hosts has been set to one. This means that there is 100% chance of a susceptible host becoming infective after sharing a cell with at least one infective host during a time step. This has been done to accelerate the rate of infection spread and magnify the effect of population density on epidemic dynamics for illustrative purposes.

Results The results of executing our model with the above-mentioned parameters are presented in the time lag map shown in Fig. 1. The lag map is a series of snapshots taken at $t = 0, 20, 40, 60, 80,$
$100, 200, 300$. Each cell is represented by a square: black squares contain at least one infective host and white squares contain only susceptible and recovered hosts. Figure 1 shows the tendency of the epidemic to follow the lines of population density to produce the 'fuzzy cross' pattern.

After 300 epochs, the top left outbreak has reached all four edges of the map but the bottom right outbreak is yet to reach any. Notice that the epidemic spreads outward along the arms or 'roads' before filling up the space between the roads. This example illustrates how disease spreads over areas of higher population density more rapidly than unpopulated areas, as expected from knowledge of known disease spread. Consequently, we can conclude that we have satisfactorily modelled the relationship between propagation delay of a disease and local population density.

3.2 Barriers to spread

This experimental scenario depicts how a CA model can used to simulate the effects barrier containment to control disease spread. As seen in the foot-and-mouth disease (FMD) epidemic in Great Britain during 2001, a key to slowing down disease spread is restricting host movement [11]. Culling of livestock to create barrier areas over which FMD cannot spread was the main eradication technique. These measures are simulated by incorporating 'no spread zones' in the initial state of the cellular automaton's lattice.

The starting distribution contains two 'hot spots' which have been segregated from the rest of the landscape. One hot spot has a four square wide barrier surrounding it, whilst the other has a one square wide barrier surrounding it. Barriers are implemented as cells with zero carrying capacity. In Fig. 2, barriers are represented by black squares and all other (blank) squares contain an equal number of hosts. The grey squares depict the two sources of infectives, both of which confined by 'buffers'. The barriers restrict host movement and provide no hosts for pathogens to infect and escape.

4 Conclusion

Accurate disease spread models are necessary for the testing of disease containment measures in the hope of reducing the economic and health impacts of disease outbreaks. We have presented an epidemic model which captures in

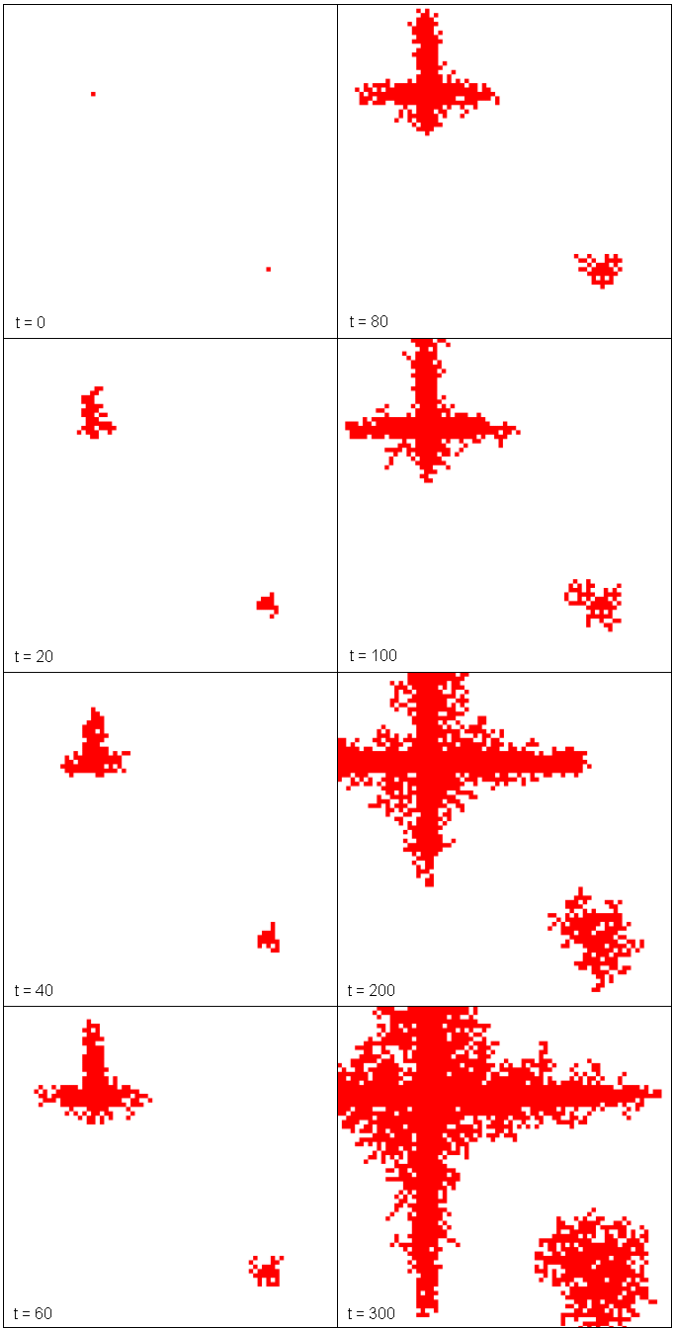

Fig. 1. A lag map showing the state of the epidemic at $t = 0, 20, 40, 60, 80, 100, 200, 300$. Notice that the outbreak to the north-west is able to cover a greater distance than the outbreak in the southeast because it has access to the road link and the population associated with that link. Notice that the spread from $t = 20$ in the top left of the map appears asymmetrical. This is probably an artifact of the stochastic nature of this model.

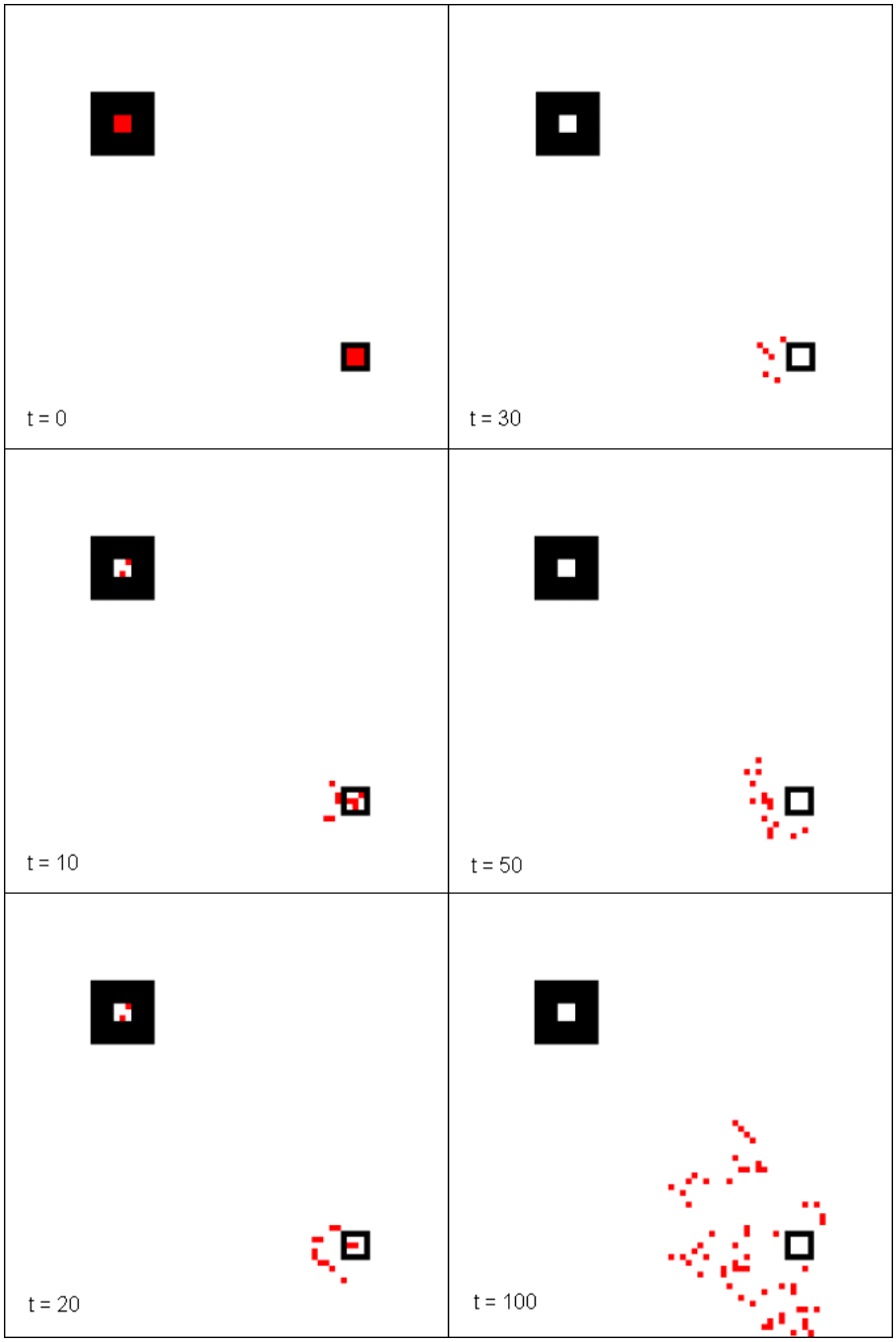

Fig. 2. This lag map shows that buffer zones that are too narrow provide no resistance to the spread of the pathogen.

a CA-like discrete framework, realistic patterns of disease spread. More specifically, a cellular automaton approach has been used to model discrete areas of landscape and non-homogeneous automata states are used to capture the effects of spatial heterogeneity. Such heterogeneity is due to variations in local population density compounded by host movements. Few existing models encode spatial heterogeneity into their mechanics; our approach shows promise for the development of accurate and tractable cellular automata based disease spread models suitable for simulation.

Two scenarios have been presented which demonstrate how a cellular automata model can be initialized with a specific configuration to run hypothetical "what if?" games. The first scenario demonstrated the successful encoding of population density effects into the epidemic model whereby disease spread is accelerated in areas of high density and slowed in sparsely populated regions. The second scenario depicts how our cellular automata model simulates the effectiveness of different disease containment strategies. These two scenarios have reproduced known patterns of spread, and thus contribute to determining the efficacy of a cellular automata modelling approach to disease spread.

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