Dynamics in a lattice epidemic model

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Abstract

We present a lattice-based epidemic model of a nonfatal communicable disease in a mobile host population in which novel dynamical behaviour is observed. The model exhibits temporal and spatial fluctuations over a wide range of time scales with a power spectrum of $1/f^2$ form. The consequences of this scaling behaviour for prevalence of endemic disease is discussed.

In the biological context we are often confronted with populations which appear to be fluctuating randomly in time, or dispersed in space in a seemingly structureless way. Insight from a nonlinear dynamics perspective has shed light on the connection between spatial and temporal dynamics which is clearly appealing if we are to fully understand the population biology of spatially distributed species. Traditionally systems of coupled ODEs, based on Lotka-Volterra type equations, have been used to model interacting populations. In particular, when studying disease spread, the mass-action assumption is most commonly used where the rate of appearance of new infections is proportional to the population of susceptibles and infectives, i.e. $dl/dt = BSI$. This type of model is a mean-field approach where spatial heterogeneity in the population is ignored [1]. More recently, in an attempt to account for spatially distributed populations, diffusion terms have been added to the basic population models, examples being the spread of rabies in fox populations [2] and the spread of the plague through Medieval Europe [3]. In this paper we present a lattice-based method for dealing with a spatially distributed host population. It facilitates the incorporation of spatial distribution in a natural way and the possibility of motion, and hence mixing, of the population over time. Also, discrete stochastic local disease transmission events can be modelled when susceptibles are in the vicinity of infectives on the lattice. Recent work on lattice based models of interacting populations [4,5] has shown the utility of this approach. Deeper insight into spatial processes has been gained from the use of methods traditionally deployed in the study of critical phenomena.

In this paper we concern ourselves with a modified version of the recently introduced model of Boccara and Cheong [4] which represents the spread of a nonfatal communicable disease through a spatially extended homogeneous host population. The principle purpose of previous studies using this model was to investigate the effects of population density and motion (mixing) on the dynamics and persistence of the disease. Though the model has not been used for applied epidemiological modelling it does show many of the characteristics of disease spread in populations and is a good starting point for the addition of more
realistic refinements [6].

A square lattice defines the points in space which may be occupied by the host population. Only one individual may occupy any given lattice site at a time. The population can be subdivided into any number of classes according to their epidemiological status. Typically three classes are used: susceptibles (those capable of receiving infection from a nearby infective), infectives (those currently carrying infection) and recovereds (those who in the past were infective but are not any longer and are immune to further infection). At the start of the simulation a number of susceptibles are randomly distributed over the lattice. Then a seed of one or more infectives is introduced. At each successive time step the individuals can hop to any one of the four nearest neighbour sites. A set of rules then determines what changes occur to the epidemiological status of each individual:

(i) If a susceptible is the nearest neighbour of an infective then the susceptible can be infected with a probability $p$.

(ii) Infectives recover and move into the recovered class with a probability $p_r$.

(iii) Recovereds can be returned to the susceptible class with a probability $p_s$.

The last two rules imply that infectives remain infectious for an average of $1/p_r$ time steps and recovereds become susceptibles after an average $1/p_s$ time steps. Recycling of recovereds into the susceptible class allows us to simulate birth and death whilst keeping the population constant. Periodic boundary conditions are applied. The model can be defined in three dimensions as well but we confine our attention to the two-dimensional case as is traditional when thinking about spatial epidemic processes. In all the following the fundamental unit of time is a single time step and the unit of length is one lattice spacing.

At each time step the total number of infectives on the lattice is recorded. Fig. 1 shows a typical time series generated by a population of 1600 individuals distributed on a $100 \times 100$ lattice. At first sight the time series of infectives shows no particular structure, but the power spectrum, $S(f)$, in Fig. 2 has a clear $1/f$ profile indicating fluctuations over a very wide range of time scales. The crossover to white noise at very low frequencies is a finite-size effect. Also at high frequencies there will be a breakdown of the scaling law because of the discrete time step nature of the simulation. The average over 100 simulations each of length $2^{14}$ time steps is taken to obtain this result. A best fit line is shown, taken over the full frequency spectrum, which gives $S(f) \sim f^{1.2}$. Clearly there is no overall frequency dominating the dynamics here, neither is this the result of a noisy equilibrium state.

The first of the scaling relationships we investigate is the distribution of fluctuation lifetimes, $D(T)$. This is measured directly from the time series, in the way defined in Ref. [7], by recording the length of deviations of the time series away from its average value. Fig. 3 shows the number of fluctuations of lifetime $T$ against $T$ in log-log form, indicating that $D(T) \sim T^{-\alpha}$ with $\alpha \approx 1.25$.

The second scaling relation addresses the distribution of fluctuation sizes, $D(S)$. In this case we sum the fluctuations away from the mean value giving a measure of the number of fluctuations of size $S$. From Fig. 4 a scaling law, $D(S) \sim T^{-\gamma}$, is observed with $\gamma \approx 1.43$.

A further quantity of interest is the distribution of infectives on the lattice. We illustrate a typical distribution of infectives on the lattice in Fig. 5 showing that the spatial distribution infectives are strongly correlated with each other. The susceptibles and recovereds are not shown for clarity. The overall population of $S + I + R$ remains uniformly distributed on the lattice at each time step. We were unable to find evidence of a fractal distribution of infectives.

All the results above are typical of the lattice epidemic simulation over a wide range of parameters and lattice sizes and are suggestive of the emergence of a self-organised critical (SOC) state. We are seeing fluctuation sizes and times on a wide range of scales though not a fractal distribution of infectives as would be expected in SOC dynamics. Our investigations of lattice size effects, whilst showing the same sort of behaviour as described above, was inconclusive due to computational limitations. The behaviour seen here should be contrasted with the more regular noisy periodic behaviour observed in lattice models with static populations [5]. Though our model does not fall strictly into the class of models that exhibit SOC, those features that is does have in common provide us with a new insight into the dynamics of disease in a mobile host population. Clearly, an epidemic model exhibiting SOC could be derived from a reinterpretation of the forest fire with sparks model of
Fig. 1. Time series of the number of infectives in the lattice epidemic simulation. We use $p = 0.5$, $p_r = 0.1$ and $p_s = 0.05$.

Fig. 2. Power spectrum (log-log) of the time series showing $1/f$ behaviour.
Fig. 3. Distribution function (log-log) of the fluctuation lifetimes. The average over 100 simulations is taken.

Fig. 4. Distribution function (log-log) of the fluctuation sizes. The average over 100 simulations is taken.
Drossel and Schwabl [9]. Also, much work has been undertaken to diagnose irregular and possibly chaotic dynamics in biological models [10], however there has been little discussion of why this might arise in real systems. Chaotic fluctuations have been shown to reduce extinction rates in certain metapopulation models [11]. The analysis we present here might go some way to explaining the biological advantage of such seemingly structureless fluctuations.

From our model, which is specified at the local level of the individual, we can observe large scale emergent behaviour. Endemic disease and its host population can be regarded as a spatially extended dynamical system. The simulation illustrates the connection of localised transmission events between individuals with the overall macroscopic dynamics of an epidemic.

Given that fluctuations occur on a wide range of time and size scales it is instructive to consider why this happens and the biological consequences. To do this it is useful to think about the case of the sandpile [5]. The analogy is not exact, but the comparison is illuminating. The sand builds up until the side of the pile reaches an equilibrium angle of repose. Moving the sand above the angle of repose will induce a predominance of large avalanches until the angle is once again restored, whereas a sandpile below the angle of repose will be subject to predominantly small avalanches until the critical angle is reached. Exactly at the critical angle a small perturbation can result in anything from a single grain movement to a large avalanche. In the epidemic model the overall average prevalence of infection is akin to the angle of repose. If we artificially increase the prevalence (by instantaneously infecting a large number of susceptibles, keeping the population constant) the system will not maintain this new elevated prevalence and it relaxes back to its previous equilibrium level. Conversely, a sudden lowering of the number of infectives (by administering an instantaneous “cure”) pushes the prevalence down, but in time it too will recover to the equilibrium level. In effect, the critical state is acting as an attractor for the dynamics in this model. By attaining this critical prevalence the disease would appear to be ensuring that it reaches a state where small fluctuations have the potential to induce cascades of infection on a wide range of length scales and a wide range of time scales. This, in effect, maximises possibility of persistence by ensuring all spatial and temporal scales are accessed. Having the system constantly in a state where only large cascades of infection occur would expose the pathogen to the risk of “burning itself out” before the stock of susceptibles is suitably replenished. Like-
wise, if the system was in a state where only small avalanches existed the disease will not maximise its prevalence. By fluctuating on many length and time scales the disease maximises the possibility of persistence (by minimising the risk of extinction) and maximises its prevalence in the host community. Hence, in this model of communicable disease, persistence and prevalence are intimately linked.

We are used to regarding an epidemic as a large positive fluctuation away from the average endemic level of disease. In a homogeneous uniform host population, such an upsurge in case notifications could only be due to a suddenly increased population density or a seasonal effect, boosting the reproductive ability of the relevant pathogen. The simulations above demonstrate that there need not be any particular seasonal or behavioural reason for these epidemics and that they can arise spontaneously out of the underlying spatial dynamics. Consequently we have to expect large epidemics from time to time but, on the whole, smaller departures from the endemic level will occur more frequently.

The presence of wide-scale fluctuations in this simple SIR model presents us with a different view of disease in a host population. Usually we regard disease as an assembly of independent infected hosts. The viral colonies in each infected host are subject to the local biological laws determined by the host immune system. This determines epidemiological parameters such as the incubation period and the period of infectiousness. Also if an infected individual is in the vicinity of a susceptible then there is a certain probability that infection will be passed. However, the presence of the scaling of the fluctuations implies that out of this independent localised viral activity there emerges a general overall dynamic which ensures persistence of the disease and maximises the prevalence.

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References