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Phase transitions in cellular automata models of spatial susceptible–infected–resistant–susceptible epidemics*

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Spatially explicit models have become widely used in today's mathematical ecology and epidemiology to study the persistence of populations. For simplicity, population dynamics is often analysed by using ordinary differential equations (ODEs) or partial differential equations (PDEs) in the one-dimensional (1D) space. An important question is to predict species extinction or persistence rate by mean of computer simulation based on the spatial model. Recently, it has been reported that stable turbulent and regular waves are persistent based on the spatial susceptible–infected–resistant–susceptible (SIRS) model by using the cellular automata (CA) method in the two-dimensional (2D) space [*Proc. Natl. Acad. Sci.* USA **101**, 18246 (2004)]. In this paper, we address other important issues relevant to phase transitions of epidemic persistence. We are interested in assessing the significance of the risk of extinction in 1D space. Our results show that the 2D space can considerably increase the possibility of persistence of spread of epidemics when the degree distribution of the individuals is uniform, i.e. the pattern of 2D spatial persistence corresponding to extinction in a 1D system with the same parameters. The trade-offs of extinction and persistence between the infection period and infection rate are observed in the 1D case. Moreover, near the trade-off (phase transition) line, an independent estimation of the dynamic exponent can be performed, and it is in excellent agreement with the result obtained by using the conjectured relationship of directed percolation. We find that the introduction of a short-range diffusion and a long-range diffusion among the neighbourhoods can enhance the persistence and global disease spread in the space.

Keywords: cellular automata, epidemiology, phase transitions, emergence of trade-offs

PACC: 0550, 0570F

1. Introduction

There is growing evidence to support the idea that space is the most important factor facilitating persistence of population in nature (see Refs.[1–4] and references therein). Estimation of the extinction risk for a population is, obviously, an important issue. It was shown in different models that a pronounced heterogeneity of environment often results in species survival, impossible otherwise, such as in host-pathogen/epidemics interactions systems[5−7] and predator-prey systems.[3,.8,.9] Specially, recently, Morozov and Li's studies[3] show that adding a second space coordinate into the one-dimensional space (1D) model results in a pronounced increase in size of the domain in the parametric space where predator-prey coexistence becomes possible. What enables some species to persist while others become extinct? This question has shaped the history of research on population dynamics and remains as a central issue for ecologists and epidemiologists.[10] Now, it is natural to ask whether the same phenomenon exists in the spread of epidemics or not. This motivates us to review some of the relevant history, and to understand the very fruitful interaction that is possible between ecologists and epidemiologists working on the population dynamics. The goal of our present work is to study the risk of epidemic extinction from the one-dimensional cellular automata (CA) model based on the spatiotemporal dynamics of susceptible-infected-resistant-susceptible (SIRS) type epidemics. The parameters are taken to be those corresponding to the persistence in the two-dimensional CA model.[11] Hence, the present paper is a continuation and an expansion of previous work[3,11] on population dynamics.

In the epidemiology, most studies concentrate on the local temporal development of disease and epidemics. Their geographical spread is less well understood, although important progress has been achieved in a number of case studies.[11−17] In modern societies, individuals can easily travel over a wide range of spatial and temporal scales. The interconnections of ar-

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eas and populations through various means of transport have an important influence on the geographical spread of epidemics. In particular, the structure and the different complexity levels of the diffusion and transport processes are responsible for the heterogeneous and seemingly erratic outbreak patterns observed in the worldwide propagation and persistence of diseases[10,11,18−20] and large-scale spatiotemporal patterns (e.g. spiral waves[11,21] and Turing patterns[22,23]) in measles, dengue fever,[24,25] severe acute respiratory syndrome (SARS),[14,26] and influenza.[27−30] In order to describe such a complex phenomenon and to obtain powerful numerical forecasting tools, different levels of description are possible, ranging from a simple global mean-field to a detailed individual-based simulation (see Refs.[31–35] and reference therein), cellular automata,[21,36−39] coupled map lattices,[40] etc. Note that although the degree distribution of the individuals is actually heterogeneous, it is assumed to be uniform in the present paper, for our results serve as a reference point for illustrating the divergence of Ballegooijen *et al*'s results[11] from the same approach. This is justified by the fact that they did not consider the heterogeneous environment.

On the other hand, on a large scale, such as the worldwide level or inter-city level, a very important role in modern epidemiology is the transportation processes in which passengers flow from one city to another. For instance, the passenger flow represents a major channel for the worldwide spread of infections diseases[14,18,41] and its complex diffusion features at various levels (such as degree distribution, traffic, population and economics). Thus, each individual can be in various states (healthy, infectious, recovered, etc), change the state through contact with other individuals and the short-range and the long-range diffusions on a grid structure. Theses processes can be considered as reaction-diffusion processes on a spatial structure,[42,43] which opens up very interesting prospects and comes up with some new issues.[12] These local reaction-diffusion models generally lead to epidemic wavefronts which were observed, for example, in the geotemporal spread of the black death in Europe from 1347 to 1350.[44,45]

Generally speaking, several generic points can be asked when the dynamics of epidemic spreading is studied. The first point is the explanation of the possible oscillations with respect to the temporal evolution of the densities of infection waves in homogeneous or heterogeneous realistic situation, as well as of the spatial resonance as a function of the control parameter of the problem under study.[39] Here, we refer to the oscillations that include the sustained oscillations via coherence resonance phenomenon in the epidemic models.[28,46] The second point is concerned with the study of the possible steady states at which a disease spreading eventually arrives as well as its arrival time.[33,46] The third point is about the study of a system in its spatiotemporal synchronization (or phase synchronization of recurrent epidemics, see Refs.[19, 20, 47]) to see the role of the parameters and space of the model in determining its fate. The fourth point, which is an important point, is whether such a simplification of the 2D or 3D spatial dynamics into 1D spatial dynamics is adequate for predicting epidemics persistence, in which many different mechanisms associated with several different control parameters can be taken into account, for instance, patterns of 2D persistence corresponding to extinction in the 1D system, (see Ref.[3] for predator-prey). We will explain why we need to simplify 2D dynamics into 1D dynamics when modelling the epidemics in Section 4.

In the present paper, we focus, in the framework of cellular automata models, on the issue of the local coupling of the nearest-neighbouring pairs of the agents and their diffusion processes with short- or long-range interactions. In the sense of the microscopic (individual) community, the spatial structure is especially important in the ecological and host-pathogen systems,[5−7,11,48] such as spatial patterns (spirals, spiral chaos and turbulence) emerging as well as power law. The emergence of spatial order structure in a range of spatial models of multi-species interactions is unsurprising, but it is an important phenomenon in the view of persistence and extinction of population.[49] The dynamics of epidemic model can also be regarded as multi-species interactions, such as the densities of the three populations: susceptible, infection, and resistant. Spatial models of multi-species systems often manifest very different behaviours from their mean-field counterparts. In particular, the individual-based models display some important general features. (i) They have a possibility of global persistence in spite of local extinctions and so are usually more stable than their mean-field counterparts, and have a tendency of spatial self-organization.[50,51] (ii) Spatial self-structure produces nonrandom spatial patterns on a scale much larger than the scale of interaction among individuals, even in a continu-

ous abiotic homogeneous environment. This structure is not explicitly coded, but it emerges from local interactions among individuals and local dispersal.[51] (iii) The population densities typically display erratic oscillations with amplitudes that vanish in the thermodynamic limit,[52] caused by persistent and recurrent population activity waves that form complex spatiotemporal structures.[11,37,53–57] Also, for the host-pathogen models with two types of pathogens, the invasion of the system by one pathogen (the other becoming extinct) through oscillatory behaviour was studied by using mean-field and pair-approximation treatments,[58–60] cellular automata, etc.

2. Spatial SIRS epidemic model description

2.1. Local infection and couple dynamics

In order to test the importance of dimensionality of space and the persistence on epidemics by using cellular automata, we developed a spatial susceptible-infection-resistant (SIRS) model for the infectious disease along the lines of previous study.[11] The population with N individuals is categorized according to its infection status into three states, i.e. susceptibility (S), infectious (I), or resistant (R).[11,19,20,45,61] Within a subpopulation and neglecting the spatial effect, the dynamics for the flow of epidemic spreading among these categories is given by

$$\frac{\mathrm{d}s}{\mathrm{d}t} = r/\tau_R - \beta si,$$

$$\frac{\mathrm{d}i}{\mathrm{d}t} = \beta si - i/\tau_I,$$

and

$$\frac{\mathrm{d}r}{\mathrm{d}t} = i/\tau_I - r/\tau_R, \tag{1}$$

where $s = S/N$, $i = I/N$, and $r = R/N$ denote the relative numbers of susceptible, infected and resistant, respectively. The above SIRS model incorporates the underlined mechanism of transmission and shifting of cyclic states dynamics and can account for experimental data in a number of cases. However, the transmission of and the resistant to an infection are intrinsically stochastic, and the deterministic SIRS model does not consider fluctuations that are particularly important at the beginning of an epidemics when the number of infected individuals is very small.

In this case, a probabilistic description must be used. We consider a basic reaction scheme conserving the number of population that has been studied both in physics and mathematical epidemiology, namely the stochastic infection dynamic process identified by the following set of reactions:

$$S + I \xrightarrow{\beta} I + I, \tag{2a}$$

$$I \xrightarrow{\tau_I} R, \tag{2b}$$

and

$$R \xrightarrow{\tau_R} S. \tag{2c}$$

The first reaction (2a) reflects the fact that an infected host (I) can infect susceptible (S) neighbours with an infection rate (β); the second reaction (2b) indicates the acquisition of resistance: hosts are infectious for a fixed period (τ_I), afterwards they become resistant (R); and the third reaction Eq.(3) indicates the loss of resistance, i.e. after a fixed period τ_R, resistant hosts once again become susceptible. In the view of individual (local)-level model, infected hosts can infect adjacent susceptible hosts at an infection rate (β) (Here, the infection rate is unequal to the probability of infection, see Refs.[11, 97].). In the short-range case, the infectious neighbourhood comprises two/eight direct nearest-neighbours (NNs) in 1D/2D square lattices. The result obtained in our 1D case is different from that of the previous study in 2D case, (for further information, see Ref.[11]). In addition, some recent investigations have included long-range processes in one- and two-dimensional lattice models, reporting quite different results on whether the self-sustained oscillation, extinction and persistence exist,[62–66] as well as increasing coupling between subpopulation (subcommunity) in spatially structured environment can lead to population outbreaks.[67] Thus, an understanding of the joint effects of short- and long-range interactions and of the individuals (including the motion called the diffusion, hopping, or stirring) is desirable and relevant from the viewpoint of ecology and statistical physics.[68] In the present paper, we also further study the outbreak of disease inspired stochastic lattice epidemic model with a next-nearest-neighbour (NNN) interaction, both in the presence and absence of an NN diffusion (exchange) process. Infection lasts a fixed infection period (τ_I), after which the host becomes resistant. Resistant hosts return to susceptibility after a fixed duration (τ_R).

At any time an individual can belong to one of three classes: susceptible (S), infected (I) and resistant (R). At each time-step Δt, cell changes state according to the following rules,[11,19] which are given by Eqs.(2a)–(2c) correspondingly:

A susceptible cell can be infected by infected cells from its NN. The probability $p_{\rm in}$ of infection is calculated from the infection rate β as $p_{\rm in} = 1 - {\rm e}^{-\Delta t \beta n_{\rm i}}$, where $n_{\rm i}$ is the number of infected neighbours (There exists a typographical error of the word pin in Ref.[11]).

Infected cells remain infected for a fixed time period (τ_I) and then become resistant.

Resistant cells remain resistant for a fixed time period (τ_R) after which the cells become susceptible again.

Thus, if τ_R is infinitely long we retrieve the SIRS model. The SIRS formulation is the most appropriate for bacterial diseases such as gonorrhea, meningitis and streptococcal sore throat, and it is also a useful model for exploring ideas concerning antibiotic resistance.[69,70]

The general feature of these transition rules allows network nodes and lattices in plane to represent individual hosts, communities, schools, cities, and so forth. For simplicity and reality, we consider the case with a population of N. Each lattice (cell) represents an individual (susceptible, infectious, or resistant). With this in mind and on a relevant time scale, it is possible to construct the simplest model that can help us understand the global disease spread and the phase transition between extinction and persistence, and that contains the biological and physical ingredients listed above, namely stochastic variation within individual cells and short- and long-range interactions by using the three-state or rock-paper-scissors technology[71−75] (see Fig.1). The simplicity of the model makes relatively fast the numerical simulation and thus an extensive description of the phase transition and spatiotemporal pattern formation in question.

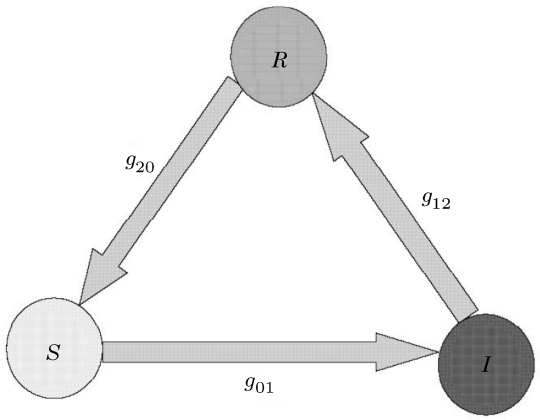


Fig.1. A diagram showing the shift of the cell state of system (2) described in a three-state way, where the arrows represent the direction of the shift at a spatial transition rate.

2.2. Three-state model of epidemic

Our starting point is to construct a tree-state unit model where cells each have transition rate g_{ij} and their states (see Fig.1) each are determined by the above rules (i)–(iii)). The spatial pattern used in this investigation comprises rectangular lattices of sites. For simplicity, each site is computerized by a value indicating its state and any given site on the lattice may be one of three states: 0 (susceptibility), 1 (infected), and 2 (resistant). The probability of going from the current state 1 to state 2 in an infinitesimal time Δt is $\Delta t g_{12}$. For a single individual, g_{ij} is simplified into a constant that represents the cyclic intrinsic frequency (or transition rate); for many individuals coupled together in the one- or two-dimensional space, we will allow g_{01} to be dependent on the neighbouring states in the spatial grid. The choice of coupling, specified below, is not unique.

For all sites, the component vector $X = \{S_1, I_1, R_1, \ldots, S_N,\ I_N,\ R_N\}$ specifies a stochastic state of the system, and the probability density function $p(X,t)$ obeys the following linear evolution equation:

$$\frac{\partial p(X,t)}{\partial t} = \sum_{X'} \{w(X|X')p(X',t) - w(X'|X)p(X,t)\}, \tag{3}$$

which is determined by the probability rate $w(X|X')$ of making a transition from state X' to X in one time step (gain term), $w(X'|X)$ that is the corresponding loss term, and the summation over all possible changes of X'. As done before, assume a constant closed population with no births or deaths. The normalization condition $p(S,t) + p(I,t) + p(R,t) = 1$ allows us to eliminate $p(R,t)$ and $p(R,t) = 1 - p(S,t) - p(I,t)$. Thus, for the process determined by Eqs.(2) and (3), the master equation reads explicitly

$$\frac{\partial p(S,t)}{\partial t} = \sum_i (S_i + 1)p(R_i^{\tau_R}, t) - \sum_i w(S_i'|S_i)p(S_i,t), \tag{4}$$

$$\frac{\partial p(I,t)}{\partial t} = \sum_i w(S_i'|S_i)p(S_i,t) - \sum_i (I_i+1)p(I_i^{\tau_I},t), \quad (5)$$

where $R_t^{\tau_R}$ denotes that the individual state reaches maximum resistance period τ_R and becomes the susceptible in the present time step at site i. $I_t^{\tau_I}$ represents the individual state reaches maximum infection period τ_I and becomes the resistance in the present time step at site i.

Along with an initial condition $p(X,\ t\ =\ 0)$, Eqs.(4) and (5) determine the temporal evolution of the process. The relation of the probabilistic master Eqs.(4) and (5) to the deterministic SIRS model (1) can be developed in the limit of a large but finite population of N, i.e. $N \gg 1$. In this limit, one can approximate the master equation by using the Fokker-Planck equation and performing an expansion in terms of conditional moments (see Refs.[14, 72, 76]).

What we are interested in is the behaviour that emerges when individuals are coupled to each other by allowing the transition probability of a given individual to be dependent on the states of its nearest neighbours in the spatial grid. More specifically, we assume that each individual state may transit to the state ahead or remain in its current state, depending on the state of its nearest neighbours with short- and long-range diffusions. For the susceptibility state, we choose the transition probability to the infected state, given as

$$p_{\rm in} = 1 - {\rm e}^{-\beta n_1 \Delta t}, \quad (6)$$

where $n_{\rm i}$ denotes the number of nearest neighbours in state 1. To make things simple, suppose that susceptible individuals after being infected keep in the infected state for a period τ_I and subsequently τ_R spend one unit in the resistant state (τ_R=1). After a period of $(\tau_I + \tau_R)$, i.e. $\tau = \tau_I + \tau_R$, individuals then return to the susceptible state. Then, we use the infection rate and the resistant period to study the system. The length of the updated cellular automata time-step Δt reduces until the system behaviour converges. We use $\Delta t = 0.01$ in simulation.

3. Simulation and results

3.1. Local interaction

When the individuals are at rest on a local site, then the infection only happens to the NNs, which is referred to as the local interaction. Specially, we study the dynamics of such a spatial extension model in one-dimensional space by defining appropriate microscopic rules. Table 1 shows the explicit transition rates in one dimension, where the susceptibility state 0 is in an isotropic space.

Table 1. The transition rates in one dimension.

neighbour	transition probability	neighbours	transition probability
1, 0, 1	1-e$^{-2\beta\Delta t}$	0, 0, 0	0
1, 0, 0 or 0, 0, 1	$1-{\rm e}^{-\beta\Delta t}$	2, 0, 1 or 1, 0, 2	$1-{\rm e}^{-\beta\Delta t}$
0, 0, 2 or 2, 0, 0	0	2, 0, 2	0

In the one-dimensional CA model the state space is a row of L ($L = N$) cells with periodic boundary conditions. Transition rules are applied, synchronously being updated at each generation. Here, initial conditions are set by both infected sites and infected period τ_I at random. To avoid possible implicit errors from random number generators,[77] the data are checked by 50 independent runs each. The random number generator itself may be different if the Linux is not used, but it does not cause any qualitative difference. The random number generator we use here is called random, which on our computer (running Linux) returns a random 32-bit integer. Moreover, the size L of the system was chosen to be sufficiently large, specifically it ranged from 1000 to 10000, but the results were insensitive to the size, so in the present paper we consider a size of 1000. Our computational approach is applicable to the study of the spatiotemporal evolution with and without the diffusion, separately. Both extinction and persistence phases in the one-dimensional case are shown in Figs.2 and 3. For the case without the diffusion, one can see that the disease outbreaks are only localized rather than globalized in the one-dimensional space. Here the persistence phase is achieved by increasing infection period τ_I and infection rate β. Our results show that the spatial pattern of persistence phase reveals different characteristics by increasing the infection period and

the infection rate separately (see Figs. 2 and 3). For the fixed values of β and τ_R, the epidemics can persist only when the infection period is above the critical value $\tau_I^{\rm c}$. In this case, the crossed stripe pattern can survive at random and covers the lattices partially. However, only the parallel stripe pattern can survive at random by increasing the infection rate up to above the critical rate ($\beta_{\rm c}$). Figure 4 shows the phase transition diagram between the extinction and the persistence of disease against β and τ_I parameters respectively, in which the epidemic will be extinct in region II but persistently spread in region I. Note that there appears a trade-off of the phase transition between extinction and persistence with respect to β and τ_I. One can see that the persistence of epidemic is more sensitive to infection rate β for the low infection period τ_I. (see Fig.4(a)). In addition, the trade-off is monodescending with infection period and infection rate. We discuss an important difference in this property between one-dimensional and two-dimensional cases in Section 4.

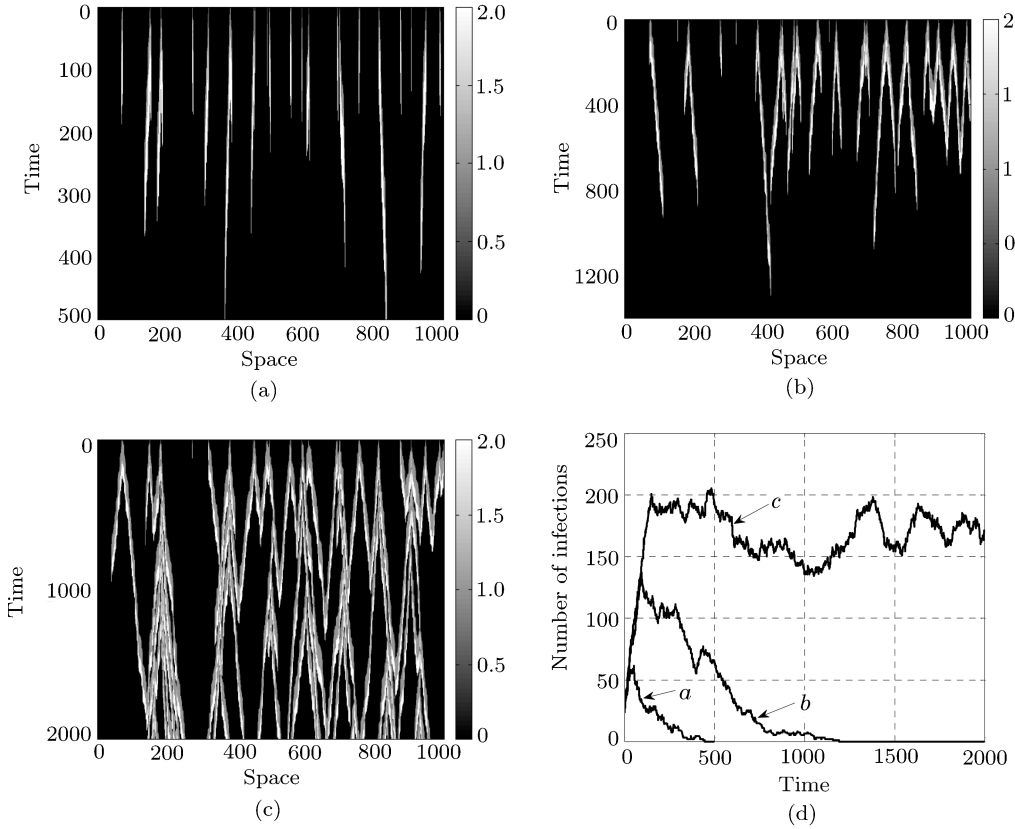


Fig.2. Spatiotemporal diagram and time series in the one-dimensional cellular automaton in the absence of diffusion. Space goes from left to right and time goes from top to bottom. Here shown are three possible states: susceptible cells (0), infected (1), and resistant (2), separately. The parameters are L=1000, β = 3.5, and only 20 infected initially. In all results presented, τ_R is set to be unity. By increasing the infection period, the epidemic is able to achieve persistence. Panel (a) is for τ_I = 0.5, $R_0 = 2\beta\tau_I$=3.5; panel (b) for τ_I = 0.95, $R_0 = 2\beta\tau_I$=6.7; panel (c) for τ_I = 1.5, $R_0 = 2\beta\tau_I$=10.5; panel (d) for the time series showing cases (a), (b) and (c) labelled by *a*, *b*, and *c* respectively.

The previous mean-field analysis on system (1) shows that the key parameter describing the infection is the basic reproduction number $R_0 = \tau_I/\tau_R$, which is the average number of secondary infections transmitted by an infected individual in the noninfected population. Similarly, in the cellular automata model the infection is also described by reproduction number R_0 = number of neighbours × infection rate × infection period.[11] The mean-field method also shows that the extinction or the persistence of disease can be predicted by R_0 and the initial relative number of susceptible individuals (required to be bigger than a critical value $s_{\rm c}$=$1/R_0$). But here our results show that the R_0 has no such a trait in the one-dimensional cellular automata model. Namely, the absorbing state of extinction or persistence is reached independently of the initial conditions (see Figs.2, 3, and 4). For example, comparing Fig.2(b) with Fig.7 in Appendix A,

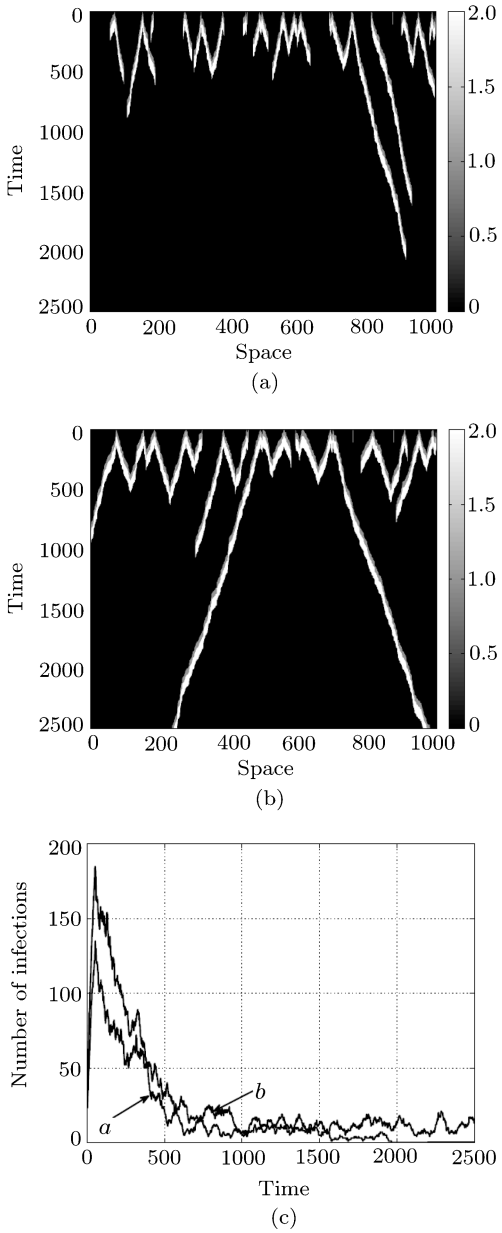


Fig.3. Same caption as that of Fig.2 except for the parameters used here: L=1000, $\tau_I = 0.95$, only 20 infected initially, β=6.5 (a) and 10.5 (b). Increasing the infection rate causes the epidemic to be able to achieve persistence.

one can see that the phase transition is independent of the initial condition. However, the change in phase transition can be predicted by the one-dimensional cellular automata model, which is consistent with the result obtained by using the mean-field method as shown in Fig.4(b), which displays the phase transition about stationary number of infections with τ_I increasing. The critical infection period (τ_I^c) with $\beta = 3.5$ and $\tau_R = 1$ is found to be about 10, i.e. $\tau_I^c \approx 1.0$. Furthermore, near the extinction threshold, we evaluate the fundamental order parameter critical exponent by using the directed percolation theory with fitting the data. The expected power-law behaviour is obtained for the stationary population level as shown in Fig.4(b), where the order parameter critical exponent is determined via $I(t \to \infty) \to (\tau_I - \tau_I^c)^\nu$ with $\nu = 369.53$.[78–80]

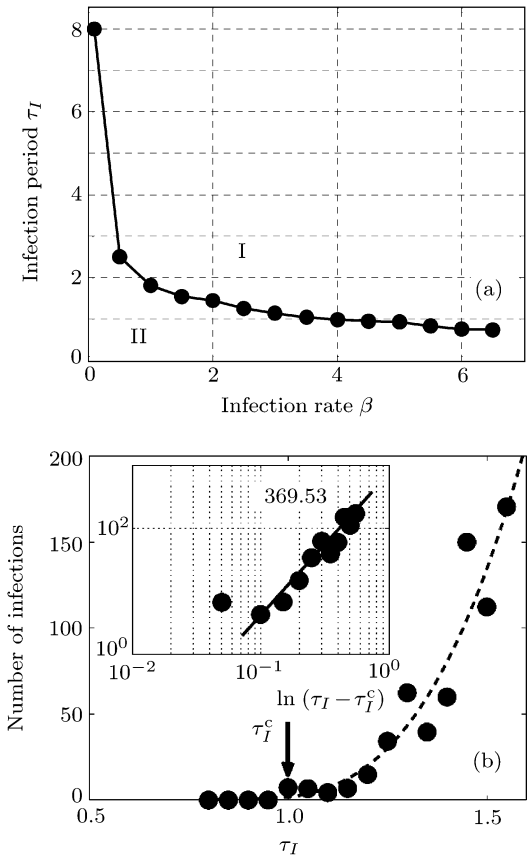


Fig.4. Phase diagram showing the cellular automata model in one-dimensional space. Panel (a) shows the phase transition between the extinction and the persistence in $\beta - \tau_I$ parameter space. The epidemic will die out in region II and propagate persistently in region I. Panel (b) displays that the average stationary density of infections is obtained as a mean value by running 50 times in simulation. There exists a directed percolation like a phase transition at $\tau_I^c \approx 1$ (for β=3.5) with an exponent of 369.53 shown in the inset. The parameters are the same as those in Figs.2 and 3, except for the parameters used here: L=1000, and only 20 infected initially. Each data point is obtained through over 3000 runs in simulation.

3.2. Short- and long-range diffusion

In fact, in a real system, an individual is in a motion state. How does individual mobility, in addition to nonlinearity, affect the system behaviour? In this section we analyse the dynamic behaviours of the system by considering spatial degrees of freedom in this process. For simplicity, we consider a simple process with two types of exchanges (diffusions) of the NN and NNN among the individuals, namely at rate ε all

individuals each can exchange their position with a nearest neighbour. These exchange processes lead to an effective diffusion of individuals. The diffusion of the infection needs considering only in the epidemic model, for the diffusion of the susceptible does not affect dynamics of the disease spreading. In the present paper, the following two types of diffusions are referred to:

Diffusion type I:

$$XI \xrightarrow{\varepsilon} IX. \tag{7a}$$

Diffusion type II:

$$XXI \xrightarrow{\varepsilon} IXX, \tag{7b}$$

where $X \in \{S,\ I,\ R\}$. It should be noted that both the diffusion process and the stirring process are not taken into account at the mean-field rate equation level. Choosing the linear dimension of the lattice as the basic length unit, then the macroscopic diffusion constant D of individuals will be shown to stem from the exchange process described as $D = \varepsilon L^{-2}$[81] in one-dimensional space.

We perform cellular automata simulation of Eq.(2) (i.e. rules (i)–(iii)) together with the diffusion processes described by Eqs.(7a) and (7b). The space and time steps are chosen to be the same as those in the foregoing section. We set the parameters β and τ_I in the domain of extinction and persistence for different fixed values of exchange rate (ε). In our simulation, the relationship between the exchange rate and the probability of exchange is used in the Gillespie algorithm.[82,83] First, we assume the parameters τ_I=0.90 and β=3.5 in domain II of extinction in the absence of diffusion (see Fig.4). Figure 5 shows the space–time diagram in the one-dimensional cellular automaton model with type I diffusion, with the diffusion coefficient ε (or D) increasing from left to right. Our results indicate that the type I diffusion does not have some effects on the emergence of trade-off and the critical point of the phase transition on system (2), but the spatial pattern changes when diffusion takes place. Figures 5(a), 5(b), and 5(c) show the scenarios of epidemic extinction and persistence for β=3.5, 3.5 and 5.0, respectively. For example, for β=3.5, the corresponding critical threshold value $\tau_I^{\rm c}$ is 1.0, i.e. $\tau_I^{\rm c}$ =1.0, in the 1D case (see Fig.4(b)). We find that the epidemic still dies out with the values of exchange rate ε increasing up to ε 50 under different initial conditions (see Appendix B) when the values of parameters τ_I and β lie in domain II (extinction region). However, the epidemic will persist in spreading when β is higher than the critical point $\beta_{\rm c}$ under the same values of ε and τ_I (see Fig.5(c), in which (β, τ_I) are in domain I).

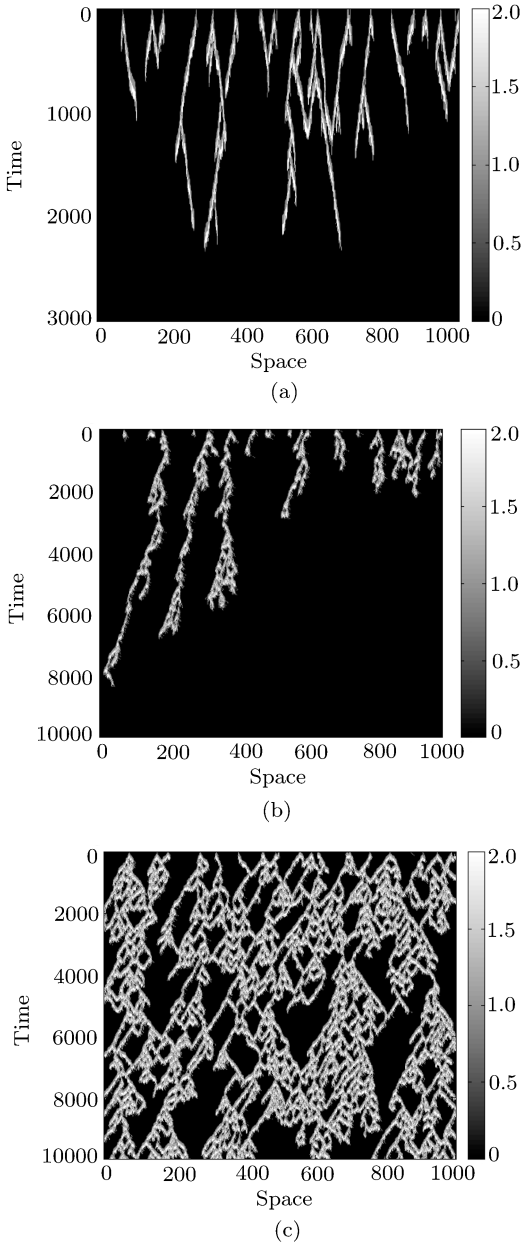


Fig.5. Space–time diagram in the one-dimensional cellular automaton with type I diffusion. Space goes from left to right and time goes from top to bottom. Here shown are three possible states i.e. susceptible (0), infected (1), and resistant (2). The parameters are L=1000, τ_I=0.90, and only 20 individuals that are infected initially for ε=5.6 and β=3.5 (a); ε=10.6 and β=3.5 (b); ε=5.6 and β=5.0 (c).

Comparing Fig.3 with Fig.5, we find the spatial pattern very different when the diffusion takes place. First, the smooth front of travelling waves becomes rough. Second, the persistence of the cross stripe pattern (wave front) becomes possible. Last, the disease outbreak quickly spreads over the entire area in a short time from the local areas as shown in Fig.5(c).

Figure 6 shows the typical space–time diagram for type II diffusion, which is obtained by using the same parameters as those in Fig.5 and the same starting distribution at the beginning. Note that for the parameters from Fig.5(b), now the epidemic is persistent in the case of type II diffusion, but it dies out with type I diffusion correspondingly. Moreover, as we can see in Figs.6(b) and 6(c), the epidemic propagates much faster and has a high density of infections. From this we conclude that the long-range diffusion considerably affects the persistence of the epidemic and disease outbreaks in the global area. Our results are obtained from the one-dimensional spatial CA model, however, these conclusions have been confirmed by the recent results obtained from the two-dimensional[12,14,18,27,33,46] network model and the CA model.[38]

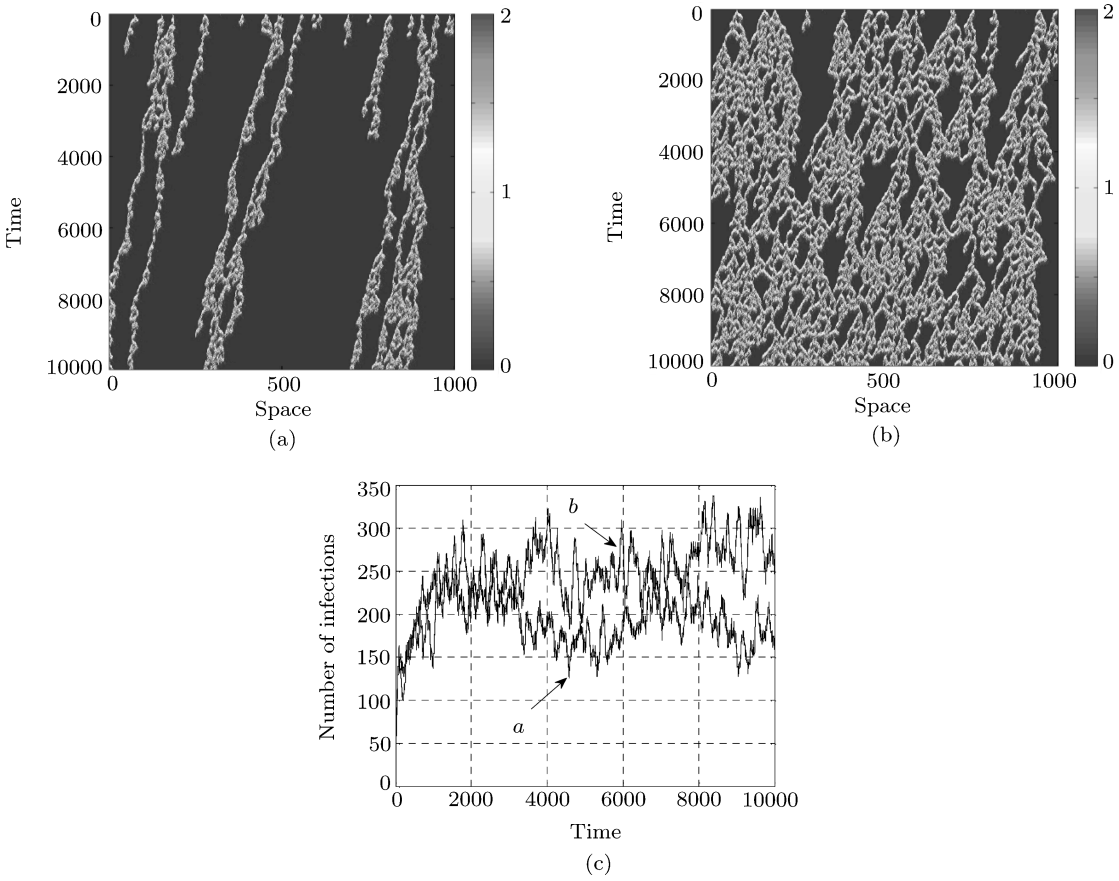


Fig.6. Same as Fig.5, except for type II diffusion appearing here, with the parameters being ε=1.6 and β=3.5 (a); ε=5.6 and β=5.0 (b); time series showing two cases labelled by a and b corresponding to Figs.5(c) and Figs.6(b), 6(c).

4. Conclusions and discussion

In the present paper, we investigate a spatial SIRS model, in which the degree distribution of the individuals is assumed to be uniform, i.e. each individual is assumed to have the same number of neighbours on the lattices. Although this assumption may be unreasonable in reality and we should take a certain heterogeneous distribution due to the existence of heterogeneous spatial structure and the social structure in the population as reported in an extensive literature, it still improves upon the mean-field method that does not take into account the effects of spatially localized transmission by several orders of magnitude.[84] A fundamental challenge to mathematical epidemiology is to determine how the structure of a population exerts

an influence on the disease transmission. One important aspect is the spatial structure. For instance, Ballegooijen *et al* [11] studies showed that stable spiral waves, turbulent and regular waves appeared in the 2D case under the parameters for extinction in the 1D case; namely, the persistence of epidemic always took place for the 2D spatial case, no matter how the initial conditions were chosen. Notably, Ballegooijen *et al* observed the emergence of a trade-off between infection period and infection rate (the emergent trade-off corresponds roughly to the transition region between turbulent waves and regular waves.). Unlike the classical trade-offs, that relationship had not been determined before, yet its emergence promoted the formation of the evolutionary dynamics of the system (2) in 2D case.[11] However, we need to take the 1D simplification of 2D dynamics in some cases when we do the modelling of epidemics. For instance, in the early stage of epidemic invasion in a spatial population, infected individuals are clustered together in areas of the lattice where the first infected cases were introduced. We refer to these infected areas as invading clusters, since their spatial extent grows with time. More specifically, we focus on estimating the speed of wavefront propagation in the contact process for one-dimensional lattices.[54,85−87] Moreover, the invading clusters actually determine the final success in or failure of the prevalence. Hence, it is important to study the spatial epidemic models in one-dimensional space. Our results show that a similar trade-off is also emergent between the infection period and the infection rate wish respect to the extinction and persistence phase transition in the 1D case. The extinction of epidemic becomes possible in the 1D case contrary to the persistence in the 2D case. Here we do not consider the 2D dynamics because this case has been investigated in Ref.[11].

On the other hand, the majority of epidemic models are formulated in terms of differential equations, such as system (1). Until recently most mathematical models of spread of epidemics have described interactions among a large number of individuals in aggregate form and often these model have neglected the spatial distribution of populations, the importance of which has been addressed in Refs.[88, 89]. Models that ignore the spatial structure can lead to inaccuracy in the prediction of population dynamics. The present paper deals with 1D cellular automata SIRS model for the spread of infectious disease with and without a population of moving individuals. The emphasis is placed on the influence of motion. This factor is usually neglected in the epidemic models. However, it is proved that it is very important in global disease spread.[12,14,18,27,33,36,38,46,90,91] For instance, the SARS epidemic spread to 12 countries in a few weeks. A spatial explicit model is necessary to evaluate the efficacy of movement controls.[92,93]

In the present paper, we take the infection period τ_I and the resistant period τ_R both as a fixed duration. An interesting point in the context of the current work is to investigate how a further phase transition to the probability model of infection period and the probability model of spatial removal will influence the space mediated epidemic persistence predicted by the 1D and the 2D models. We should note that these probability three-state spatial CA models can be compared with the stochastic aspects of introduced model that is based on stochastic differential equations, in particular in the context of the approach given in Refs.[14,81–83]. It is worth noting that the emergence of stochastic (noisy) pattern has such a feature shared with crossdisciplines in many complex systems that are characterized by their nonequilibrium nature and nonlinear interactions. The relevant examples range from the well-known Belousov–Zhabotinsky reaction[94] and many other chemical reactions[95] to epidemic outbreaks (travelling waves),[24,44] excitable media[95,96] and so forth.

Appendix A: Independent of initial condition

In this Appendix, we provide a supporting figure (Fig.7) to prove that the absorbing state of extinction is reached independently of the initial conditions.

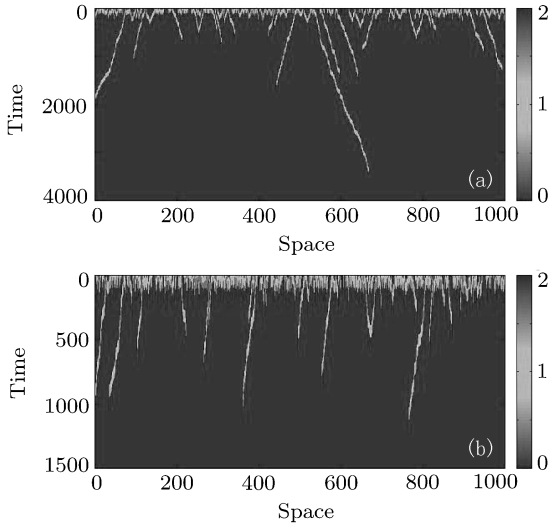


Fig.7. Space–time diagram in the one-dimensional cellular automaton in the absence of diffusion. Space goes from left to right and time goes from top to bottom. Here shown are three possible states: susceptible (0), infected (1), and resistant (2), separately. The parameters are L=1000, β=3.5, and τ_I=0.95. In all results presented, τ_R is set to be unity. By increasing the infection number at the beginning, the epidemic is still extinct when the parameter in domain II. Panel (a) is for 200 infected initially;nd panel (b) for 500 infected initially.

Appendix B: Supporting information with type I diffusion

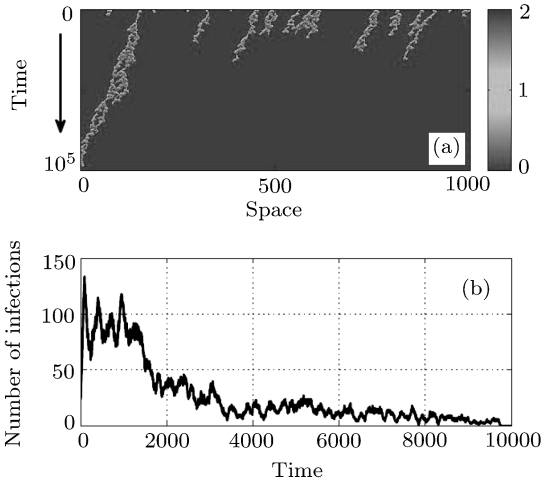


Fig.8. Space–time diagram and time evolution in the one-dimensional cellular automaton with type I diffusion. The parameters are L=1000, τ_I=0.90, and only 20 infected initially. Panel (a) is for ε=50 and β=3.5, where space goes from left to right and time goes from top to bottom and three possible states are shown: susceptible (0), infected (1), and resistant (2), separately. Panel (b) is for the time series corresponding to panel (a).

References

[1] Comins H N and Hassell M P 1996 *J. Theor. Biol.* **183** 19

[2] Maron J L and Harrison S 1997 *Science* **278** 1619

[3] Morozov A and Lia B L 2007 *Theor. Popul. Biol.* **71** 278

[4] Petrovskii S, Li B L and Malchow H 2004 *Ecol. Complexity* **1** 37

[5] Hassell M P, Comins H N and May R M 1994 *Nature* **370** 290

[6] Wilson W G, Harrison S P, Hastings A and Mccann K 1999 *J. Ani. Ecol.* **68** 94

[7] Wood S N and Thomas M B 1996 *Proc. R. Soc. Lond.* B **263** 673

[8] Gurney W S C, Veitch A R, Cruickshank I and McGeachin G 1998 *Ecology* **79** 2516

[9] Petrovskii S V, Morozov A Y and Venturino E 2002 *Ecol. Lett.* **5** 345

[10] Ear D J D, Rohani P and Grenfell B T 1998 *Proc. R. Soc. Lond.* B **265** 7

[11] van Ballegooijen W M and Boerlijst M C 2004 *Proc. Natl. Acad. Sci. USA* **101** 18246

[12] Colizza V, Pastor-Satorras R and Vespignani A 2007 *Nat. Phys.* **3** 273

[13] Colizza V and Vespignani A 2007 *Phys. Rev. Lett.* **99** 148701

[14] Hufnagel L, Brockmann D and Geisel T 2004 *Proc. Natl. Acad. Sci. USA* **101** 15124

[15] Keeling M J, Woolhouse M E J, May R M, Davies G and Grenfell B T 2003 *Nature* **421** 136

[16] Keeling M J, Woolhouse M E J, Shaw D J, Matthews L, Chase-Topping M, Haydon D T, Cornell S J, Kappey J, Wilesmith J and Grenfell B T 2001 *Science* **294** 813

[17] Smith D L, Lucey B, Waller L A, Childs J E and Real L A 2002 *Proc. Natl. Acad. Sci. USA* **99** 3668

[18] Colizza V, Barrat A, Barthelemy M and Vespignani A 2006 *Proc. Natl. Acad. Sci. USA* **103** 2015

[19] He D and Stone L 2003 *Proc. R. Soc. Lond.* B **270** 15919

[20] Stone L, Olinky R and Huppert A 2007 *Nature* **446** 533

[21] Liu Q X, Jin Z and Liu M X 2006 *Phys. Rev.* E **74** 031110

[22] Liu Q X and Jin Z 2007 *J. Stat. Mech.* p05002

[23] Sun G, Jin Z, Liu Q X and Li L 2007 *J Stat. Mech.* p11011

[24] Cummings D A T, Huang N E, Endy T P, Nisalak A, Ungchusak K and Burke D S 2004 *Nature* **427** 344

[25] Vecchio A, Primavera L and Carbone V 2006 *Phys. Rev.* E **73** 031913

[26] Colizza V, Barrat A, Barthelemy M and Vespignani A 2007 *BMC Medicine* **5** 34

[27] Colizza V, Barrat A, Barthelemy M, Valleron A J and Vespignani A 2007 *PloS Medicine* **4** e13

[28] Dushoff J, Plotkin J B, Levin S A and Earn D J D 2004 *Proc. Natl. Acad. Sci. USA* **101** 16915

[29] Viboud C, Bjornstad O N, Smith D L, Simonsen L, Miller M A and Grenfell B T 2006 *Science* **312** 447

[30] Viboud C, Miller M A, Grenfell B T, Bjornstad O N and Simonsen L 2006 *PLoS Medicine* **3** e503

[31] Eubank S, Guclu H, Kumar V S A, Marathe M V, Zoltan Toroczkai A S and Wang N 2004 *Nature* **429** 180

[32] Ferguson N M, Keeling M J, John Edmunds W, Gani R, Grenfell B T, Anderson R M and Leach S 2003 *Nature* **425** 681

[33] Gautreau A, Barrat A and Barthelemy M 2007 *J Stat. Mech.* L09001

[34] Keeling M J 1999 *Proc. R. Soc. Lond.* B 266 859

[35] Pastor-Satorras R and Vespignani A 2001 *Phys. Rev. Lett.* **86** 3200

[36] Boccara N and Cheong K 1992 *J. Phys.* A: *Math. and Gen.* **25** 2447

[37] Boccara N, Roblin O and Roger M 1994 *Phys. Rev.* E **50** 4531

[38] Fuks H, Lawniczak A T and Duchesne R 2006 *Eur. Phys. J.* B **50** 209

[39] Ramani A, Carstea A S, Willox R and Grammaticos B 2004 *Physica A* **333** 278

[40] Sherratt J A, Eagan B T and Lewis M A 1997 *Philos. Trans. R. Soc. Lond.* B **352** 21

[41] Colizza V, Barrat A, Barthelemy M and Vespignani A 2006 *Bull. Math. Biol.* **68** 1893

[42] Albert R and Barabasi A L 2002 *Rev. Mod. Phys.* **74** 47

[43] Boccalettia S, Latorab V, Morenod Y, Chavez F M and Hwang D U 2006 *Phys. Rep.* **424** 175

[44] Grenfell B T, Bjornstad O N and Kappey J 2001 *Nature* **414** 716

[45] Murray J D 1993 *Mathematical Biology* (Berlin: Springer)

[46] Gautreau A, Barrat A and Barthelemy M 2008 *J. Theor. Biol.* **251** 509

[47] Blasius B, Huppert A and Stone L 1999 *Nature* **399** 354

[48] Pekalski A and Droz M 2006 *Phys. Rev.* E **73** 021913

[49] Hassell M P, Comins H N and May R M 1994 *Nature* **370** 290

[50] Dieckmann U, Law R and Metz J A J 2000 *The Geometry of Ecological Interactions: Simplifying Spatial Complexity* (Cambridge: Cambridge University Press)

[51] Johnson C R 1997 *Frontiers in Ecology*: *Building the Links* In chap: Self-organizing in spatial competition systems (Elsevier Science) p245–263

[52] Provata A, Nicolis G and Baras F 1999 *J. Chem. Phys.* **110** 8361

[53] Droz M and Pekalski A 2001 *Phys. Rev.* E **63** 051909

[54] Jeltsch F, Muller M S, Grimm V, Wissel C and Brandl R 1997 *Proc. R. Soc. Lond.* B 264 495

[55] Keeling M J, Brooks S P and Gilligan C A 2004 *Proc. Natl. Acad. Sci. USA* **101** 9155

[56] Savill N J, Rohani P and Hogeweg P 1997 *J. Theor. Biol.* **188** 11

[57] Tainaka K I 1994 *Phys. Rev.* E 50 3401

[58] de Aguiar M A M, Rauch E M and Bar-Yam Y 2003 *Phys. Rev.* E **67** 047102

[59] de Aguiar M A M, Rauch E M and Bar-Yam Y 2004 *J. Stat. Phys.* 114 1417

[60] Rauch E M, Sayama H and Bar-Yam Y 2002 *Phys. Rev. Lett.* **88** 228101

[61] Anderson R M and May R M 1991 *Infectious Diseses of Humans* (Oxford: Oxford University Press)

[62] Antal T and Droz M 2001 *Phys. Rev.* E 63 056119

[63] Antoniazzi A, Fanelli D, Ruffo S and Yamaguchi Y Y 2007 *Phys. Rev. Lett.* **99** 040601

[64] Julian Adamek A S Michael Keller and Hinrichsen H 2005 *J. Stat. Mech.* P09002

[65] Rozenfeld A F and Albano E V 2004 *Phys. Lett.* A **332** 361

[66] Tessone C J, Cencini M and Torcini A 2006 *Phys. Rev. Lett.* 97 224101

[67] Petrovskii S and Li B L 2001 *J. Theor. Biol.* **212** 549

[68] Mobilia M, Georgiev I T and TÄauber U C 2006 *Phys. Rev.* E **73** 040903

[69] Girvan M, Callaway D S, Newman M E J and Strogatz S H 2002 *Phys. Rev.* E **65** 031915

[70] Hethcote H W and Levin S A 1989 *Applied Mathematical Ecology* chap. Periodicity in epidemiological models. (Berlin: Springer) p193–211

[71] Kerr B, Riley M A, Feldman M W and Bohannan B J M 2002 *Nature* **418** 171

[72] Reichenbach T, Mobilia M and Frey E 2006 *Phys. Rev.* E 74 051907

[73] Reichenbach T, Mobilia M and Frey E 2007 *Nature* 448 1046

[74] Szabo G, Santos M A and Mendes J F F 1999 *Phys. Rev.* E **60** 3776

[75] Wood K, den Broeck C V, Kawai R and Lindenberg K 2006 *Phys. Rev. Lett.* **96** 145701

[76] Kuske R, Gordillo L F and Greenwood P 2007 *J. Theor. Biol.* 245 459

[77] Ferrenberg A M, Landau D P and Wong Y J 1992 *Phys. Rev. Lett.* **69** 3382

[78] Chen C C, Park H and den Nijs M 1999 *Phys. Rev.* E **60** 2496

[79] Hinrichsen H 2000 *Adv. Phys.* **49** 815

[80] Janssena H K and TaÄuber U C 2005 *Ann. Phys.* **315** 147

[81] Reichenbach T, Mobilia M and Frey E 2007 *Phys. Rev. Lett.* 99 238105

[82] Gillespie D T 1977 *J. Phys. Chem.* **81** 2340

[83] Gillespie D T 2000 *J. Phys. Chem.* **113** 297

[84] Bauch C T 2005 *Math. Biosci.* **198** 217

[85] Pearce I G, Chaplain M A J, Schofield P G, Anderson A R A and Hubbard S F 2007 *J. Math. Biol.* **55** 365

[86] Schofield P G, Chaplain M A J and Hubbard S F 2005 *J. Math. Biol.* **50** 559

[87] White K A J and Gilligan C A 1998 *Phil. Trans. R. Soc. Lond.* B **353** 543

[88] Durrett R 1999 *SIAM Rev.* **41** 677

[89] Durrett R and Levin S A 1994 *Theor. Popu. Biol.* **46** 363

[90] Boccara N and Cheong K *J. Phys.* A: *Math. and Gen.* **26** 3707

[91] Reluga T C, Medlock J and Galvani A P *Bull. Math. Biol.* **68** 401

[92] Eubank S, Guclu H, Anil, Marathe M V, Srinivasan A, Toroczkai Z and Wang N 2004 429 *Nature* 180

[93] Riley S, Fraser C, Donnelly C A, Ghani A C, Abu-Raddad L J, Hedley A J, Leung G M, Ho L M, Lam T H, Thach T Q, Chau P, Chan K P, Lo S V, Leung P Y, Tsang T, Ho W, Lee K H, Lau E M C, Ferguson N M and Anderson R M 2003 *Science* 300 1961

[94] Zaikin A N and Zhabotinsky A M 1970 *Nature* **225** 535

[95] Kapral R and Showalter K (Eds.) 1995 *Chemical Waves and Patterns* (Dordrecht: Kluwer)

[96] Muratov C B, Vanden-Eijnden E and Weinan 2007 *Proc. Natl. Acad. Sci. USA* **104** 702

[97] Brauer F and Castillo-Chaavez C 1996 *Mathematical Models in Population Biology and Epidemiology* (New York: Springer)