Persistence, extinction and spatio-temporal synchronization of SIRS spatial models

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Abstract. Spatially explicit models are widely used in today’s mathematical ecology and epidemiology to study persistence and extinction of populations as well as their spatial patterns. Here we extend the earlier work on static dispersal between neighboring individuals to the mobility of individuals as well as multi-patch environments. As is commonly found, the basic reproductive ratio is maximized for the evolutionarily stable strategy for disease persistence in mean field theory. This has important implications, as it implies that for a wide range of parameters the infection rate tends to a maximum. This is opposite to the present result obtained from spatially explicit models, which is that the infection rate is limited by an upper bound. We observe the emergence of trade-offs of extinction and persistence for the parameters of the infection period and infection rate, and show the extinction time as having a linear relationship with respect to system size. We further find that higher mobility can pronouncedly promote the persistence of the spread of epidemics, i.e., a phase transition occurs from the extinction domain to the persistence domain, and the wavelength of the spirals increases with the mobility ratio enhancement and will ultimately saturate at a certain value. Furthermore, for the multi-patch case, we find that lower coupling strength leads to anti-phase oscillation of the infected fraction, while higher coupling strength corresponds to in-phase oscillation.

Keywords: cellular automata, self-organized criticality (experiment), pattern formation (theory), interacting agent models
1. **Introduction**

In recent years, there has been increasing awareness of the threats posed by newly emerging and high profile infectious diseases, such as SARS, the H5N1 strain of avian influenza, HIV, ebola, whooping cough, dengue fever and the spread of influenza. These diseases exhibit large scale spatial contagion and long term spatial patterns [19,35,36,52,53,77,102,105,106].

At present, there is a great deal of interest in the role of spatial structure in both ecology and epidemics. All systems are to some degree spatially extended; however, the classical theory of the dynamics of epidemics ignores spatial effects with its assumption of homogeneous mixing. It is likely that local processes will play an important role in the majority of infectious disease interactions, particularly when an infection occurs through the direct contact of infected and susceptible individuals [39,82]. As a consequence, there have been some attempts to take spatial structure into account explicitly when examining the spreading of parasites [21,40], [57]–[59], [66,102]. For example, recent models show that interactions between local populations may generate complex spatial patterns by dispersal [40,48,66,102,105]. In modern societies, individuals can easily travel over a wide range of spatial scales. The interconnections of areas and populations through various means of transport have important effects on the geographical spread of epidemics [105]. In particular, the spatial structure and the different levels of diffusion and transport processes are responsible for the heterogeneity, which is an erratic outbreak pattern observed in the worldwide propagation and persistence of diseases [25,36,52,97,102], recently documented synchronization and waves [19,48,52,61,97,105], and large scale spatial patterns (e.g. spiral waves [55,66,102] and Turing patterns [65,98]) in measles, dengue fever, SARS, and influenza. For describing such a complex phenomenon and develop powerful, numerical forecasting tools, different levels of description are possible, ranging from a simple global mean field to detailed...
individual-based simulation (see [31, 37, 38, 41, 58, 70, 80] and references therein), cellular automata [14–16], [33, 40, 55, 66, 102], coupled map lattices [95], etc.

Dispersing individuals may react in a complex manner to local ecological and epidemiological situations [55, 64, 94, 105, 106], but rare long distance dispersal events may be important in Nature [1, 2, 55, 77]. The modeling methods that summarize dispersal in a diffusion coefficient in the classical theory—using partial differential equations—cannot give insight into the importance of rare events [21]. For humans and other social animals in which hosts are distributed in heterogeneous patches on a large scale, there are two critical sides to transmission. The first is local transmission among individuals within patches or communities (cities, towns, and villages). The second is the transmission between patches. With this point of view, there is a clear conceptual link between ecological and epidemiological theory; a body of recent works have focused on the analogies of the spatial dynamics between infectious diseases and ecological metapopulations [18, 36, 49, 106]. The twofold issues are dissecting how infection processes at the local scale determine spatio-temporal patterns of epidemics and understanding how these patterns are affected by the spatial spread between neighborhoods or patches—for instance, the epidemic wavefronts observed in the spatio-temporal spread of the Black Death in Europe from 1347 to 1350 [48, 73, 78] and West Nile virus [63].

Generally speaking, several generic questions can be asked when studying the dynamics of epidemic spreading. One is that of the explanation of the possible oscillations with respect to the temporal evolution of the densities of infection waves in a homogeneous or heterogeneous realistic situation, as well as the spatial resonance problem. Another concerns the study of the possibility that a disease spread eventually arrives at a steady state and the study of the time that it needs to arrive there [41, 42]. In addition, our model can also investigate what enables some species to persist while others become extinct. This question has shaped the history of research on population dynamics and remains a central issue for ecologists and epidemiologists [36].

Although the spatial structure has been developed for susceptible–infected–resistant (SIR) models in previous studies, the modulation of such effects in combination with the mobility of individuals is also unknown. We aim to bridge this gap in this paper. Specifically, we aim to elucidate the phase space for extinction and persistence, and contrast it with the prediction obtained for the mean field, as well as the effect of mobility on persistence criteria. We also examine how the spatial pattern depends on the mobility and coupling strength within the multi-patch case.

2. Model and methods

2.1. Cellular automata model

In a recent report, van Ballegooijen and Boerlijst (for short, vBB) showed that there were three types of spatial pattern in a spatial susceptible–infected–resistant model for disease dynamics by using a grid structured contact network, also called a cellular automata model [102]. They were localized disease outbreaks with self-limitation in size, turbulent waves, and stable spiral waves. Furthermore, they predicted that there existed a trade-off between the parameters of infection period and infection rate, which emerges from

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The sustained oscillations include the coherence resonance phenomenon in the epidemic models [3, 34, 62, 96].

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the evolutionary dynamics of the system in the stable spiral waves, and referred to this relationship as \textit{emergent trade-off}. These results give a guide for understanding the spatial features of epidemics such as wave speed and wavelength, as well as the relationship of the persistence and extinction depending on the parameters. To model the spatio-temporal phenomena, a feasible approach is the combination of grid-based models and movements of individuals. In this paper we take the framework proposed by vBB as a starting point in assessing the phase transitions between the persistence and extinction rather than the emergence of the stable spiral waves.

We use a spatial susceptible-infected-resistant model. The population with \( N \) individuals is categorized according to its infection status: susceptible (\( S \)), infected (\( I \)), or resistant (\( R \)) \[5, 52, 97, 102\]. Within a subpopulation, the dynamics for the local populations obeys a basic reaction scheme conserving the population numbers, which has been studied both in physics and in mathematical epidemiology, namely the stochastic infection dynamics process identified via the following set of reactions \[52, 102\]:

\[
\begin{align*}
S + I & \rightarrow I + I \quad \text{with} \quad \beta, \\
I & \rightarrow R \quad \text{after time} \quad \tau_I, \\
R & \rightarrow S \quad \text{after time} \quad \tau_R.
\end{align*}
\]

The first reaction \((1a)\) reflects the fact that an infected host (\( I \)) can infect susceptible (\( S \)) neighbors with infection rate \( \beta \); the second reaction \((1b)\) indicates the acquisition of resistance such that hosts are infectious for a fixed period \( \tau_I \), after which they become resistant (\( R \)); and the third reaction \((1c)\) indicates the loss of resistance such that after a fixed period \( \tau_R \), resistant hosts once again become susceptible (the symbols \( \tau_I \) and \( \tau_S \) represent fixed times after which the reaction occurs). In the sense of an individual/local level model, infected hosts can infect adjacent susceptible hosts at infection rate \( \beta \). Here, the infection rate is not equal the probability of infection (see \[23, 102, 106\]). In the short range case, the infectious neighborhood consists of two/eight direct nearest neighbors (NN) in 1D/2D square lattices. In addition, some recent investigations have concluded that long range processes in one- and two-dimensional lattice models, reporting quite different results for the occurrence of self-sustained oscillation, extinction and persistence \[6, 7, 21, 56, 83\], \[91\]–\[93\], \[100, 101\], as well as an increasing coupling between subpopulations (subcommunities) in spatially structured environments, can lead to population outbreaks \[81, 105, 106\]. Thus, an understanding of the joint effect of short and long range interaction on the individuals (including the mobility denoted as diffusion, hopping, or stirring; hereafter, we refer to just mobility) is desirable from ecological, statistical and physical points of view \[72\]. Of course, in the present paper, we also study the outbreak of disease, inspired by the stochastic lattice epidemic model with a nearest neighbor interaction (see figure 1). In order to compare with the early work by vBB \[102\], here the cellular automata method is used to simulate the spatial interactions. However, we note that, both with and without spatial influences, the stochastic SIRS model \((1)\) can be simulated using the Gillespie algorithm \[4, 43, 44, 86, 88\].

For ease of comparison, we follow vBB’s model by considering a regular network of sites, each of which contains a single susceptible individual (\( S \)), an infected individual (\( I \)), or a resistant one (\( R \)). The susceptible is infected by contact with an infected neighborhood at a rate \( \beta \) (see figure 1(b)). Every time step \( \Delta t \) (here \( \Delta t = 0.01 \)), all
Figure 1. An illustration of the state transition for the SIRS model. (a) State transition diagram where each arrow shows a possible transition between two states in the model. (b) Diagram of density independent (top two transitions) and density dependent (bottom two transitions) transition rates showing the neighborhood effect. Transition probabilities are shown for transitions between different states (see the text for the symbol description). The third transition shown is from a susceptible site that has one infected neighbor (local infected occupied \( n_I = 1/z \); smaller circle, \( z \) is the number of the cell’s neighborhood) to an infected cell.

Cell states are updated in parallel according to the rules of the previous studies [52, 102], which are illustrated in figure 1.

2.2. Rate equations

By using deterministic rate (or mean field) equations to describe the temporal evolution of the SIRS model, one neglects all spatial correlations. The study of the rate equation is based on the analysis of epidemic spreading and outbreak. In particular, the properties of the rate equations are extremely useful for assessing epidemic persistence using estimated values for the classical expression for \( R_0 \)—the basic reproduction number. \( R_0 \) is defined as the number of secondary infections caused by a single infected individual during its infectious period in an entirely susceptible population [5, 73]. The standard SIRS mean field approximations, also referred to in reaction (1), are as follows:

\[
\frac{dS}{dt} = -\beta SI + \tau_R R, \quad (2a)
\]

\[
\frac{dI}{dt} = \beta SI - \tau_I I, \quad (2b)
\]
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Figure 2. Three different types of the spatial pattern in the absence of mobility with $\tau_I = 0.40$, $\beta_{\text{min}} = 0.60$ and $\beta_{\text{max}} = 2.10$. Here three possible states are shown: susceptible cells (white), infected (red), and resistant (black). (a) Localized disease outbreaks are self-limiting in size for $\beta = 0.62$; (b) turbulent waves for $\beta = 1.20$; (c) stable spiral waves for $\beta = 1.80$. The grid size for all panels is $200 \times 200$. Note that all the patterns arise from the same initial condition. These spatial patterns can be produced by using supporting online material (see the computer code for the cellular automata at http://194.171.24.200/ppages/qliu/). The random number generator itself may be different if you are not using Linux, but it does not make a qualitative difference.

where $S$ and $I$ denote the number of the susceptibles and infecteds, respectively. The number of recovered individuals $R$ is obtained by conservation of the entire population, i.e., $R(t) = N - S(t) - I(t)$. The key quality describing the infection is the basic reproduction number $R_0 = \beta/\tau_I$. If $R_0 > 1$ and the initial relative number of susceptibles is greater than a critical value $S_c = 1/R_0$, an epidemic develops ($dI/dt > 0$). As the number of the infected individuals increases, the number of susceptibles $S$ decreases, and thus the number of contacts of the infected individuals with susceptibles decreases until $S = S_c$; then the epidemic reaches its maximum and subsequently decays. The existence of oscillatory dynamics depends on the particular choice of parameter values.

The ordinary differential equation model (see equations (2)) has implicitly exponential waiting times between compartments. It can be derived as a mean field equation system from a stochastic process with exponential waiting times for all transitions—not only the infection process, but also the recovery etc. To describe the epidemiologically plausible fact of a more fixed resistant period, in modeling approaches often several infected classes are used, hence not $S \rightarrow I \rightarrow R \rightarrow S$ but $S \rightarrow I_1 \rightarrow I_2 \rightarrow \cdots \rightarrow I_n \rightarrow R \rightarrow S$, each transition between infected classes still exponentially distributed, but the transition from $S$ via all $I$ classes to $R$ now gamma distributed, and in the limit of infinitely many $I$ classes (hence $n$ going to infinity) a sharp fixed recovery time. So, the reaction scheme, equations (1), with fixed resistant period $\tau_I$ (as also used by vBB [102]) and the ODE system, equations (2), have different underlying assumptions. This has consequences for the spiral waves. For models with all transitions given by exponential waiting time the walls of eventually appearing spirals are broken off again due to the recovered becoming susceptible just behind the front of the infected wave (see figure 2). For the reaction scheme with fixed
parameter $\tau_R$ and seemingly more important fixed parameter $\tau_I$ transition from recovered to susceptible, the walls of the propagating infected and, behind this front, the recovered wave allow for stable spirals.

3. Simulation and results

To begin with, we revisit some of the findings of vBB [102]. Their results then serve as a reference point for illustrating the difference of our elaborated approach from theirs. This is justified by the fact that vBB only provide the spatial pattern resulting from static dispersal among individuals within a single patch (there is no mobility among the individuals), as well as the emergence of trade-offs between spiral waves and turbulent waves. Our crucial measures are the phase transition between the global extinction and the persistence of the epidemic, and the effect from the movements of individuals. In these simulations we set the migration between patches to zero, i.e. the coupling parameter $LR = 0$ ($LR$ represents the migration ratio between two patches; see the definition in equation (4)), in which our simulations faithfully repeat the general findings of vBB [102]; that spatial patterns of three types emerge (see figure 2), but the differences are found when the mobility of individuals is turned on.

3.1. Phase transitions of persistence and extinction

In this subsection, we analyze the relationship of infection rate and infection period for the phase transition between global persistence and extinction within a single patch. In section 3.3, we will introduce multiple patches into the spatial structure modeled via the strength of coupling between two patches.

When the individuals are static at the local site, then the infection only happens to the nearest neighbors; this form is referred to as local interaction. In particular, we can study the dynamics of such a spatially extended model in one- or two-dimensional space by defining appropriate, microscopic rules. Equations (1) show the explicit transition rates of cell states in space, where the susceptibles infected by the infectious are isotropic. The grid size used in the simulations is $200 \times 200$ cells and the time step equals 0.01. Larger grid sizes do not change the evolutionary dynamics (see the computer code for the CA model at http://194.171.24.200/ppages/qliu/).

The persistence phase space is that where the epidemics will persist when the parameters lie in a certain parameter space. Here, we refer to the $(\tau_I, \beta)$ parameter space. The values are determined by simulating $200 \times 200$ cell lattices with ten independent runnings. Each lattice is initially set with random occupation by 100 infected individuals and zero recovery. For each value obtained, we fix the infection period $\tau_I$, and choose an initial infection rate $\beta$. Then, increasing $\beta$ in small steps (we use $\Delta \beta = 0.05$), 10,000 time units are simulated with that infection rate value to determine the critical value of the phase transition. Here, we find that 10,000 time units is a long term run for persistence on this system. However, the typical waiting time $T(N)$ until extinction occurs is generally very long when the system size $N$ is large. This suggests considering the dependence of the waiting time $T(N)$ on $N$. Quantitatively, we discriminate between persistence and extinction by using the concept of extensivity, adapted from statistical physics [85, 86]. If the epidemic is not extinct at that time, the pair parameter values are assumed to
be within the persistence space. In the present model, we find that the epidemic will be persistent when parameter $\beta$ is between a minimal critical infection rate $\beta_{\text{min}}$ and a maximal critical infection rate $\beta_{\text{max}}$. The maximal critical infection rate indicates that for $\beta$ sufficiently large too many susceptibles get infected at the same time and therefore die simultaneously, leading to extinction. There is no susceptible to maintain the survival of the pathogen; this is also called pathogen driven extinction. It is also worth noticing that the definitions of persistence and extinction in the presence of absorbing states are intimately related to the concept of the long term.

We perform extensive computer simulations of the system (1) for the phase transition which describes the persistence and extinction with respect to the $(\tau_I, \beta)$ parameter space shown in figure 3; this also demonstrates that there exists a trade-off between infection period and infection rate. In order to compare $R_0$ in the classic mean field approximation with the phase transition curve for the spatial structure, we use the power laws to fit the simulation data. Moreover, previous studies show that the infection rate ($\beta$) and the virulence ($\alpha$) also have a relation of power laws in the parameters of the infection rate and virulence [21,22]. Yet, this relationship can be expressed generally with an $f(x) = ax^b + c$ formula, as shown in figure 3, in which the red curve and blue curve are fitted by this function with 95% confidence intervals on the parameter estimates. As could

Figure 3. Extinction and persistence phase transition for $(\tau_I, \beta)$ parameter space. The red circles and blue circles are estimates from the simulation for $\beta_{\text{max}}$ and $\beta_{\text{min}}$ respectively. The red line (marked a) is fitted by using the $f(x) = ax^b + c$ function and $a = 0.5264$ (0.4456, 0.6071), $b = -0.7324$ (-0.7647, -0.7001), and $c = 1.008$ (0.7124, 1.304). The blue line (marked b) is fitted by using the $f(x) = ax^b + c$ function and $a = 0.6276$ (0.4384, 0.8168), $b = -0.6658$ (-0.7287, -0.6029), and $c = -0.3256$ (-0.4643, -0.1869). The dashed line (marked c) denotes the prediction obtained from mean field theory for $R_0$. The inset shows the nonzero y-axis intercept $c$ (nonvanishing infection rate) on a double-logarithmic plot, where the symbol bullets (●) and circles (○) correspond to curves a and b respectively.
be expected, there exist minimal and maximal critical values of the infection rate $\beta$ for the phase transition. The open circles ($\bigcirc$) and filled circles ($\bullet$) indicate the simulated results for the minimal critical infection rate $\beta_{\text{min}}$ and maximal critical infection rate $\beta_{\text{max}}$, respectively. By comparison between fitted curves and simulations, one can see that there is a nonlinear trade-off; $\beta(\tau_I)$ is a monotonically decreasing function of $\tau_I$ and bounded from below when $\tau_I$ is within a certain interval. According to the numerical fits reported in figure 3, we have $\beta = 0$ approximately at $\tau_I = 2.6$. Therefore, one could conclude that $\beta$ is bounded from below by a positive constant when $\tau_I < 2.0$. The dashed line in figure 3 shows the prediction on the phase space from mean field theory for $R_0$. From the classical expression for $R_0$, we know that the diseases will die out if $R_0 < 1$. This has important implications, as it implies that for a wide range of $(\beta, \tau_I)$ space there are two parameter domains in the $(\beta, \tau_I)$ space. One is the domain of extinction and the other is the domain of persistence. This contrasts with the result obtained in a spatially explicit model where the parameter domain is divided into three parts: domains I and III are disease free; domain II is endemic (see figure 3). The numerical results clearly indicate (see figure 3, inset) the validity of the spatial thresholds for evaluating the invasion of parasites. In the log–log plot (inset of figure 3), the dashed line and power laws ($c = 0$) are straight lines of slope $-1$ shown on the graph. Then, the same is found where $\log(f(x) - c)$ is plotted against $\log(x)$. One can see that a nonzero $c$ (nonvanishing infection rate) gives a better straight line. In this case, the nonzero $c$ corresponds to the phase boundaries for large $\tau_I$.

The classical mean field theory on the disease spreading has shown that the number of secondary cases due to a single infected individual (referred to $R_0$) is maximized with respect to susceptibles in the well-mixed model (2) and therefore maximum transmission $\beta$ is selected. Here, our results show that once the spatial structure is included, $R_0$ is no longer maximized and the transmission rate is limited (see the line labeled a in figure 3). We note that these results are consistent with parasite evolution and extinction in the SIR model [22]. In addition, in the spatially explicit model, the diseases can survive for a lower infection rate than for the mean field case, because a low infection rate will tend to increase the local density of susceptible individuals around infectious. The spatial structure of clusters of individuals favors a mild infection rate. Our recent investigations show that this is also true in the spatial multi-strain models [67]. Conversely, with higher infection rate there will be a tendency for all the individuals in a cluster to be infected quickly. This in turn leads to relatively rapid local cluster extinction. Now a question is whether the disease spreading can be sustained for a larger $\tau_I$ but with a finite value and an arbitrarily small $\beta$ or an opposite combination. Our simulation shows that the spatially local interaction model with the combination of large infection period and small infection rate will lead to the disease persisting, and the opposite combination will lead to the disease becoming extinct.

Moreover, we also find that, for fixed infection period $\tau_I$, different moving spatial patterns emerge with infection rate $\beta$ increasing. Figure 2 shows typical snapshots of the stable spatial patterns when the parameters are within domain of persistence II in figure 3, in which the results indicate that the threshold value $\beta_{\text{min}}$ is equal to 0.60 and $\beta_{\text{max}}$ equal to 2.10 for $\tau_I = 0.40$. When the infection rate is low (but just above the critical value $\beta_{\text{min}}$), we find that the susceptible and infectious individuals coexist, and self-organized spatial patterns have limited size of the moving clusters. This means that localized disease outbreaks are self-limiting in size for this case (see figure 2(a)). With increasing infection

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rate $\beta$, these structures grow in size and form patterns of moving turbulence/spirals (see figures 2(b) and (c)), and disappear for large enough $\beta$ (exceeding the critical value $\beta_{\text{max}}$).

Bartlett observed in a series of papers [9]–[11] that measles in large cities had recurring outbreaks, while it went extinct in small communities until reintroduced from external sources. This means that the time to extinction is an increasing function of the community size. However, the analysis by Nasell showed that Bartlett’s approximation for the extinction time was unsatisfactory in an important part of parameter space [74,75]. Our present system (1) is a simple individual-based stochastic SIRS model with diffusion processes [3,17,88,96]. The goal of the analysis is to derive information about the quasi-stationary distribution and the time to extinction. Pursuing this goal leads to difficult mathematical problems. Exact solutions cannot be found. A possible way to proceed is therefore to work with approximations by simulation. Early efforts to derive approximations of the expected time to extinction were shown by Nasell [74] to lead to large errors. The resulting approximation of the expected time to extinction was rather coarse with a linear relation, but turned out to be an accepted approximation for the present analysis of extinction and persistence.

The dependence of the average time ($T_{\text{ex}}$) to extinction on the system size of changes, $N$, is shown in figure 4, which also shows the dependence of $T_{\text{ex}}$ on the size of the populations when the parameters are within the domain of extinction. As seen, average extinction time increases linearly with system size $N$ (we test the system size from 10,000 to 90,000). More quantitatively, $T_{\text{ex}}$ is approximately proportional to $N$; we fit the simulation results by using a linear function, and then obtain the relationship as $T_{\text{ex}} = 0.055N + 758.75$ as shown in figure 4. To ensure that the averaging over just 100 independent runs used yields good statistics, we present in figure 4 the data for $T_{\text{ex}}$ obtained for 100 runnings for $\tau_I = 0.40$ and $\beta = 0.50$ (these parameters corresponding to extinction domain I in figure 3). It should be noted that previous phase transition analysis

Figure 4. Average time to extinction $T_{\text{ex}}$ versus system size $N$ for $\tau_I = 0.40$ and $\beta = 0.50$. The least-squares linear fit of the data has a slope of 0.055 and an intercept of 758.75 (see the text for details).
is convincing because three extinction times were used to estimate the phase transitions of persistence and extinction. One can see that $10^4$ time units are a long term run for persistence in this system. Here, it is instructive to note that our results show that the persistence and extinction are dependent on the population size. The opposite view has been obtained from well-mixed systems recently [24], where a critical population size above which coexistence is likely was demonstrated.

3.2. Mobility promotes persistence in a spatial epidemic model

In fact, the individual always exhibits motion in the space. How does an individual’s mobility, in addition to nonlinearity, affect the system’s behaviors (i.e., persistence and extinction, spatial pattern, invasion speed and so on)? Insight into this important issue can be gained from the cellular automata model. In particular, one can compare the results of the present paper with the previous works [102]. Two models are commonly used to predict the spatial spread of a disease. The first is the distributed-contact model, often described using a contact distribution among stationary individuals. Distributed-contact models are particularly appropriate for the study of plant disease [103, 104], but researchers are also using the closely related framework of contact networks to study disease transmission in human populations [76, 84] and animal species. Notice that recently developed spatial moment closure methods are also based on this framework [20, 32]. The second model is the distributed-infective model, often described using the mobility of infected individuals. This approach relies on the assumptions that disease is transmitted through interactions between dispersing individuals and that infected individuals move in uncorrelated random walks. Medlock and Kot developed a distributed-infective framework that uses a flexible kernel-based approach similar to that employed in distributed-contact models [71]. They found that inappropriate application of either the distributed-contact or distributed-infective approaches can generate inaccurate projections of epidemic spread. Hence, here we consider a scenario wherein the transmission process involves components of both distributed-contact and distributed-infective diffusion of infected individuals. For instance in the spread of rabies, foxes tend to be restricted to discrete home ranges. In Central Europe, the home range size is about 4 km$^2$, but this may differ considerably between areas (range 2.5–16 km$^2$) [50, 55]. On the basis of section 3.1, now we investigate the individuals with a certain form of mobility. Namely, at rate $\varepsilon$ all individuals can exchange their position with a nearest neighbor. Each individual randomly exchanges with its eight neighbors (north, west, south, east, northwest, southwest, southeast, and northeast) at each time step. This is reasonable for a realistic system. These exchange processes lead to an effective mobility of the individuals.

In this subsection we analyze the dynamics of the system by considering individual spatial diffusion processes. In fact, susceptibles’ mobility will also lead to the spread of the disease since it changes the spatial distribution of the infected individuals. In the present paper, we consider the mobile individuals including susceptible, infected, and resistant ones. Hereafter, we use the terms exchange, mobility, and diffusion as having the same meaning—the mobility of individuals within nearest neighbors. Hence, we simply refer to them as ‘mobility’. We refer to the mobility as follows:

$$XY \xrightarrow{\varepsilon} YX,$$

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where $X \in \{S, I, R\}$. It has to be noted that this mobility is not taken into account at the mean field rate equation level, or in vBB’s study [102]. Denote as $L$ the linear size of the $d$-dimensional hypercubic lattice (i.e. the number of sites along one edge), such that the total number of sites reads $N = L^d$. Choosing the linear dimension of the lattice as the basic length unit, the macroscopic diffusion constant $D$ of individuals stemming from mobility processes reads $D = \varepsilon d^{-1} N^{-2/d}$ for a continuum limit [87,88]. In this limit, a description of the stochastic lattice system through stochastic partial differential equations (SPDE) becomes feasible. But here we study the system’s behavior using the approach of the cellular automata model.

Note that we performed cellular automata simulations in figure 1 with the mobility process equation (3). The space and time steps were chosen as the same as in section 3.1. However, one of the reviewers pointed out that the Gillespie algorithm is also very appropriate in spatially explicit cases as well—for example, see the recent work of Reichenbach et al [88]. The simulation of the reaction (1) with the Gillespie [43,44] algorithm is beyond the scope of the present paper. Here, we use the cellular automata method to examine how the movements of individuals affect the persistence and extinction of an epidemic in the spatially structured population. In particular, we will examine the role of the mobility’s strength $\varepsilon$ when the parameters are less than the critical values $\beta_{\text{min}}$. First, we investigate the time series of the infected fraction. We have kept the infection period $\tau_I$ fixed at a value $\tau_I = 0.40$, and systematically varied the mobility rate $\varepsilon$. In figures 5 and 6, we show typical long time series of the infections for various values of the mobility rate with the parameters $\beta = 0.42$ (below the critical value $\beta_{\text{min}} \approx 0.6$) and $\beta = 2.50$ (above the critical value $\beta_{\text{max}} \approx 2.10$), respectively. Figures 5(a) and (b) show a family time series for an infected fraction before and after the mobility rate is turned on; different $\varepsilon$ values marked a, b, and c appear in figure 5(b). We observe in figure 5 that the mobility raises the possibility of shift from extinction to persistence state.
in the spatial epidemic model. Furthermore, this persistence with irregular fluctuation is around a positive equilibrium. It is worth noting that the large mobility rate $\varepsilon$ can promote the persistence of spatial epidemics despite the parameter $\beta$ being below the minimal critical value $\beta_{\text{min}}$. Our simulations indicate that similar results hold when the parameter is above the maximal critical value $\beta_{\text{max}}$, but in which the persistence corresponds to low mobility rate $\varepsilon$ (see figure 6).

Next, we examine the effect of mobility on the regions where the epidemic will be extinct when the mobility rate is varied. By using the method developed in the literature [86], we know that this scenario can be distinguished by computing the probability $P_{\text{ext}}$ when the epidemic has gone extinct after a waiting time $t \propto N$. For illustration, we have considered the fixed parameters $\beta = 0.42$ and $\tau_I = 0.40$. In figure 7, we obtain the dependence of $P_{\text{ext}}$ on the mobility rate $\varepsilon$. With increasing mobility rate, the extinction probability with a sharpened transition emerges at a critical value $\varepsilon_c = 8.0 \pm 0.05$ for the entire $200 \times 200$ lattice area explored in $10000$ time units. One can see that, above $\varepsilon_c$, the extinction probability $P_{\text{ext}}$ tends to zero as the mobility rate increases, and the epidemic has stable persistence. On the other hand, below the critical mobility rate $\varepsilon_c$, the extinction probability approaches $1$ for small mobility rate, and the epidemic occurrence is unstable. One of our central results is that we have identified a mobility threshold for persistence of epidemics. Without loss of generality, we have also tested $P_{\text{ext}}$ data for the other parameter values $\tau_I$ and $\beta$; the same sharpened transitions have been observed from the lattice simulations.

There exists a critical value $\varepsilon_c$ such that a high mobility $\varepsilon > \varepsilon_c$ changes the extinction phase to a persistence phase, while $\varepsilon < \varepsilon_c$ extinction remains, leaving a uniform state with only susceptibles in the entire space. It is worth noting that the above results arise from domain I of the extinction phase in figure 3 where $\beta < \beta_{\text{min}}$ and we have the absence
The critical mobility $\varepsilon_c$ with different system sizes. Mobility above the value $\varepsilon_c$ induces persistence, while there is extinction below that threshold. The data are obtained from lattice simulations of the system after long temporal development (that is, at time $t \propto N$) and for different values of $\varepsilon$ with $\beta = 0.42$ and $\tau_I = 0.40$. Here, we have considered the extinction probability $P_{\text{ext}}$ starting with randomly distributed individuals on a $200 \times 200$ square lattice and a $100 \times 100$ square lattice.

Figure 7. The critical mobility $\varepsilon_c$ with different system sizes. Mobility above the value $\varepsilon_c$ induces persistence, while there is extinction below that threshold. The data are obtained from lattice simulations of the system after long temporal development (that is, at time $t \propto N$) and for different values of $\varepsilon$ with $\beta = 0.42$ and $\tau_I = 0.40$. Here, we have considered the extinction probability $P_{\text{ext}}$ starting with randomly distributed individuals on a $200 \times 200$ square lattice and a $100 \times 100$ square lattice.

Therefore, our results also indicate that stronger mobility can increase the parameter region where the epidemic persistence occurs. This may be completely explained from the local and global infection [21]. Higher mobility leads to an increase of the global contact among individuals in the lattices.

Reichenbach et al assessed the effects of mobility on the spatial pattern of rock–paper–scissors games [86]. Results demonstrated a critical influence of mobility on species diversity: when mobility exceeds a certain value, biodiversity is jeopardized and lost. In contrast, below this critical threshold all subpopulations coexist and an entanglement of traveling spiral waves forms over the course of time. In our study, we incorporate the scenarios outlined [30, 86] and also investigate a spatial pattern induced by mobility when the parameters are within domain II of figure 3. We perform extensive computer simulations of the stochastic system (see equations (1) and (3)) and typical snapshots of the steady states are shown in figure 8. With increasing mobility $\varepsilon$, these moving spiral structures grow in size and saturate at a certain value, but they do not disappear over time. This is different from the rock–paper–scissors games case where the spiral wave will disappear for large mobility [86]. As shown in figure 8, the wavelength of the spirals $\lambda$ rises with increase of the mobility of individuals. When the parameters are within domain I, we have not observed spatial pattern emergence for varied mobility rate $\varepsilon$. However, we find that high mobility leads to global disease outbreaks instead of localized disease outbreaks (see figure 9).
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Figure 8. The effect of mobility on spiraling patterns. We show snapshots obtained from lattice simulation of typical states of the system after long temporal development and for different values of $\varepsilon$ starting with randomly distributed individuals on a square lattice. (a) $\varepsilon = 0.01$; (b) $\varepsilon = 8.05$. The parameters are $\tau_I = 0.40$ and $\beta = 1.80$.

Figure 9. Spatial pattern in the lattice simulation for mobility above the value $\varepsilon_c$ induces persistence with $\beta = 0.42$ and $\tau_I = 0.40$. Here, we have considered the spatial pattern starting with randomly distributed individuals on a square lattice. (a) $\varepsilon = 8.0$; (b) $\varepsilon = 12.0$.

From the above observations we know that the mobility of individuals can dramatically affect epidemic extinction/persistence and spatial patterns. We should point out here that, although our results in this study come from cellular automata simulations, they are also useful for assessing the dynamics of deterministic systems, for example, which is better for predicting disease persistence. By modeling the interaction among individuals, we are able to understand the role of spatial mixing (cause by mobility) in invasion dynamics without the need for complex mathematical methods. Whether an organism can successfully invade and persist in the long term is dependent on many factors. Here, we have identified a mobility factor for epidemic persistence and invasion, which is distinct from the nonlinear dynamics of the epidemic models.

3.3. Spatio-temporal synchronous dynamics of spatial epidemics

As individuals travel around the world (human) or migrate (animals), disease may spread from one place to another. The spatial spread of epidemics has been much studied,
Figure 10. Typical spatial structured model of the two-patch spread of epidemics (schematic). The two patches $i$ and $j$ are connected by migration processes. Each patch contains a population of individuals who are characterized with respect to their stage of the disease (e.g. susceptible, infected, resistant), and identified with a different color in the picture.

particularly as regards pandemic invasion waves \[36, 45, 46, 54, 105, 106\]. Simulation models incorporating transportation have generated important insights into the spread of epidemics. However, the key underlying relationship between human movement and disease spread has not been verified across wide spatial scales. To quantify the traveling behavior of individuals, we consider the linked dynamics of two host communities each of size $N$, where individuals possess mobility in each patch. Spatial coupling is the critical parameter determining phase coherence and spatial synchrony \[48, 60, 68, 69\] and the persistence of host–pathogen systems \[47, 99\]. For simplicity, we assume that $N$ is constant through time for each patch. A typical schematic for two patches is shown in figure 10.

Consider now two patches with sizes $200 \times 200$ cells, each representing a community or suburb, and suppose a small number of infective individuals in each community at the initial time. An individual can pass from one community to the other. This migration or the individual traveling is randomly chosen for some given individuals moving from a community to another community in large metapopulation systems. The strength of coupling between the two communities is measured by

$$LR = \text{number migrating/size of community}$$

(4)

with respect to unit time. The rules for implementing the migration from one patch to another are: every step, we let $LR$ per cent of the total number of individuals exchange with another patch in which all the individuals are chosen in random positions from the lattices. Although the approach is only a rough approximation of the dynamic nature of transportation between two communities, it nevertheless captures some of the essential elements \[26, 52\]. Figure 11 shows simulation results with different strengths of coupling between the two communities, in which the mobility within a patch is ignored. In this case, the infectives in each community are seen to oscillate irregularly and in local anti-phase for weak coupling strength (see figures 11(a) and (b)). Upon increasing the strength
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Figure 11. The time series of infected fractions in two coupled communities with $\varepsilon = 0$, $\beta = 1.80$, and $\tau_I = 0.40$. (a) With weak coupling, $LR = 0.001$, rapidly anti-phase synchronization comes to predominate; (b) with intermediate coupling, $LR = 0.01$, the remaining anti-phase synchronization predominates; (c) with strong coupling, $LR = 0.1$, the epidemic peaks and the troughs between them show rapid in-phase synchronization.

of coupling between the communities, the suburbs show in-phase synchronization (see figure 11(c)). Notice that similar results have been obtained by using SIRS network models [52, 61]. As the results from section 3.2 show, at the single-patch level, the epidemic behavior on the global scale is also determined by the mobility of individuals. In particular, the effects due to the finite size of communities and the stochastic nature of the mobility might have a crucial role in the problem of resurgent epidemics, extinction and eradication (also see [8, 26, 28, 29]). Therefore it is important to consider the effect of mobility rate on synchronization for the metapopulation system. In figure 12, we report the effect of differing mobility rate on the time series of infected fractions from lattice simulations. Figure 12 provides clear evidence of spatio-temporal synchrony in the metapopulation system independent of the self-mobility, but it depends on the strength of coupling among patches. One can draw a conclusion that mobility within each patch does

3 Note that here synchronization or asynchronization refers to oscillation with uniform phase evolution, yet having chaotic amplitudes (UPCA). In addition, the synchronization can be quantified by calculating the phase of each time series of infectives and plotting the phase difference between the two suburbs [13, 48, 90].

4 Here, ‘global’ refers to the single patch, rather than the metapopulation system.

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Figure 12. The time series of infected fractions in two coupled communities with different mobility rates inside each patch. (a) With low mobility rate, $\varepsilon = 0.5$, anti-phase synchronization predominates; (b) with high mobility rate, $\varepsilon = 4.5$, the remaining anti-phase synchronization predominates. The other parameters are the same as in figure 11(b).

Figure 13. The time series of weekly measles case reports for Birmingham and Newcastle between 1948 and 1969. Data made available by Professor Benjamin Bolker (www.zoo.ufl.edu/bolker/measdata.html).

not play a signification role, and spatial structure within patches can be ignored when we investigate the coupling of patches.

One of our main goals is to study the manner in which two or more communities or suburbs synchronize in time, as recurring waves of infection sweep through their respective populations by means of cellular automata simulation. This is a fascinating outcome of
Spatial dynamics whereby a few migrating infective individuals have the potential to spread the epidemic from one community to the other, giving rise to synchrony in the long term. For instance, the time series of measles infections in various cities of England (1944–1958) are reported in the literature [12, 48, 52]. Birmingham and Newcastle appear to show in-phase synchronization while Cambridge and Norwich are clearly out of phase by 180° (see figure 13 and figure 2 in [12, 48] respectively). Here, we simulate a simple coupled spatial community model to gain insights into the coupling strength. These results provide useful insights for the basic theoretical understanding of mechanistic epidemic models in complex multi-patch environments, which can then be used to build more realistic data driven large scale computational approaches for real case scenarios and spatially targeted control measures. For instance, the spread of influenza shows spatial synchrony in 49 states of the United States [105].

4. Conclusions and discussion

To summarize, from the simulations of processes of the spreading of individuals among lattices randomly distributed in a space, we can identify different effects of parameters describing the spatial structure and the temporal developments of disease for individuals, such as $\tau_I$, $\beta$, $\varepsilon$, $LR$ and $N$, on the dynamical behavior—persistence, extinction and synchrony. We change every parameter which we consider can affect the spreading process. Although the short term behavior (persistence and extinction) may depend on the system size $N$ and details of the spatial distribution, the long term behavior mainly depends on three parameters: the infection period $\tau_I$, infection rate $\beta$, and the mobility $\varepsilon$ for a single community. The two former parameters determine what types of spatial pattern will emerge, and the latter dramatically increases the parameter region where the epidemic pandemic occurs in the space. Moreover, we investigate the coupling between multiple communities, in which the two former parameters $\tau_I$ and $\beta$ reflect the nature of the developing and pandemic periods of a disease for individuals, and the coupling strength $LR$ is a parameter related to the population density, the frequencies of people’s contacts, and the extent of traveling of people between different places or cities [47]. These results may be helpful for the analysis of processes of spreading of disease in a space.

The possibility of spiral waves, self-organized spatial patterns, spotted patterns, traveling waves as well as trade-offs in spatial epidemics and host–parasitoid relationships via local dispersal abilities has long been recognized [27, 48, 51, 65, 66, 98, 102, 105]. What is new here is that the trade-off between persistence and extinction has been understood in the spatial structure epidemic model. The discrete character of the individuals involved in the reactions (1) and the mobility processes (3) are responsible for the intrinsic stochasticity arising in the system. The mobility of individuals and intrinsic noise have crucial influences on the self-formation of spatial patterns. The analytical expressions for the wavelengths of the spirals as functions of mobility can be determined by means of a complex Ginzburg–Landau equation (CGLE) obtained by recasting the PDE derived from the interacting particle approach [86, 88]. Hence, to qualitatively explain these findings, a profound understanding is still desirable and could motivate further investigations. The other open question is whether power laws exist for the infected cluster size in the present spatial system when the parameters are at the critical points [79, 89].

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