Reinfection induced disease in a spatial SIRI model

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Received: 27 August 2010 / Accepted: 27 September 2010 /
Published online: 13 October 2010
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Abstract Spatial models are widely used in epidemiology to investigate persistence and extinction of disease as well as their spatial patterns. One of the most important issues in studying epidemic models is the role of infection in the persistence and extinction of the disease. In this paper, we investigate a spatial susceptible–infected–recovered–infected model using cellular automata. We show that, in the regime where disease disappears in the susceptible–infected–recovered–susceptible model, spiral and target waves will emerge in the two-dimensional space due to the reinfection. The obtained results may point out that reinfection has great influence on the epidemic spreading, which enriches the findings of spatiotemporal dynamics in epidemic models.

Keywords Cellular automata · Reinfection · Epidemiology · Persistence

1 Introduction

There has been an increased awareness of the threats posed by newly emerging infectious diseases, such as SARS, the H5N1 strain of avian influenza, H1N1, and so on [1–8]. Many different approaches have been used to model the dynamics of spatially structured populations by theoretical epidemiologists. The advent of modern computing facilities has

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encouraged epidemiologists to turn to individual-based spatial simulations, such as cellular automata [9–13].

The cellular automaton model is a discrete approach to the time, space, and state. Moreover, such a model shows the features of the epidemic model clearly with both mathematical analysis and numerical simulations. It is assumed that the cellular automata model has habitable sites, and a site can be occupied by only one individual. Spatially structured pathogen transmission means that the probability that a susceptible host suffers from some disease depends more on the infection density of its neighbors than on the global infection density [14, 15].

In the previous work, an epidemic model using probability-based cellular automata has been investigated [14]. The essential mathematical features were analyzed with the help of stability theory. A discrete and spatiotemporal approach is shown by using cellular automata theory. Finally, a comparison was made with experimental data for SARS disease in China in 2003 and a brief discussion were given. In [11], Liu et al. made a study on epidemic models with spatial structure based on the cellular automata method. The construction of the cellular automata is based on a reaction–diffusion approach. The results showed that the spatial epidemic models exhibit spontaneous formation of irregular spiral waves at large scales and exhibit chaotic behavior. Moreover, the irregular spiral waves grow stably, which confirms that populations embed and disperse more stably in space than they do in nonspatial counterparts. Sun et al. investigated the influence of infection rate and migration on extinction of disease in spatial epidemics using cellular automata [15]. They found that when the infection rate is small or large enough, the disease will disappear for a long time, which provides a mechanism for explaining the extinction of disease.

Recently, Liu et al. extended the earlier work on static dispersal between neighboring individuals to the mobility of individuals as well as multi-patch environments [16]. The emergence of trade-offs of extinction and persistence for the parameters of the infection period and infection rate was observed. It is also found that higher mobility can pronouncedly promote the persistence of epidemics. For the multi-patch case, lower coupling strength leads to anti-phase oscillations of the infected fraction, while higher coupling strength corresponds to in-phase oscillations.

In the present paper, we use a susceptible–infected–recovered–infected (SIRI) model—in which the recovered will be infected by the infected. This model corresponds to the case of cross immune or multi-strain epidemics [17–21]. The paper is organized as follows: In Section 2, we present the spatial SIRI model using cellular automata. We show the effect of reinfection by performing a series of numerical simulations in Section 3. Finally, we give some discussion and conclusion.

2 Spatial SIRI model

Recently, van Ballegooijen and Boerlijst showed that there were three types of spatial patterns in a spatial susceptible–infected–recovered–susceptible (SIRS) model for disease dynamics by using a cellular automata model [22]. Within a subpopulation, the dynamics for the local populations obeys a basic reaction scheme conserving the population size, according to the following set of reactions:

In the cellular automation, S, I, and R represent the state in one discrete lattice. The first reaction (Eq. 1a) means that the susceptible individuals (S) can be infected at a probability of β through the contact between the susceptible and infected individuals. As a result, a susceptible individual can become infected at a probability $1 - (1 - \beta)^{N_I}$ [23], where N_I is the sum of infected individuals in its neighborhood. The second reaction (Eq. 1b) indicates the acquisition of resistance where hosts recover from infection after a fixed infectious period τ_I , and the third reaction (Eq. 1c) indicates the loss of resistance where after another fixed period τ_R , recovered hosts become susceptible once again.

Stollenwerk et al. presented a SIRI model, which obeys the following rules [24]:

Equation 2d means that a recovered individual can be infected at a probability $1 - (1 - \bar{\beta})^{M_I}$, where M_I is the sum of infected individuals in the recovered individual's neighborhood.

We assume that the individuals are static at the local site and the infection only happens to the nearest neighbors, which is referred to as a local interaction. In particular, we can study the dynamics of such a spatial model in two-dimensional space by defining appropriate, microscopic rules. Equation 1 shows the explicit transition rates of cell states in space, where the susceptibles infected by the infected individuals are isotropic. The grid size used in the simulations is 300×300 cells and the time step equals 0.001. We have confirmed that larger grid size and smaller time step do not change the evolutionary dynamics.

3 Results

For the model given by Eqs. 1a–1c, it was shown that there were three different types of spatial patterns. They were localized disease outbreaks with self-limited size, turbulent waves, and stable spiral waves, respectively [22]. Furthermore, in Fig. 1, we show that the epidemic will be persistent when parameter β is between a minimal critical infection rate $\beta_{\min}$ and a maximal critical infection rate $\beta_{\max}$.

In the present paper, we analyze the effect of reinfection rate in the model given by Eqs. 2a–2d, namely $\bar{\beta}$, on the persistence of the disease when $\beta < \beta_{\min}$ and $\beta > \beta_{\max}$, with the aim of reporting reinfection-induced disease. To address this issue, we set $\tau_I = 0.4$ and $\tau_R = 1$ and vary β and $\bar{\beta}$.

In Fig. 2, spatial patterns of infected population at $t = 0, 100, 200, 300, 400$, and 500 with random initial conditions and $\beta = 0.1$ are presented. It can be seen from this figure that the random initial distribution leads to the formation of a strongly irregular transient pattern in the domain. After the irregular pattern forms, a stable spiral wave emerges in the two-dimensional space and the dynamics of the system does not undergo any further changes. For the overall analysis, we show the density of the infected individuals in Fig. 3. In a word, reinfection can induce the persistence of the disease with small β .

An interesting question is whether reinfection will induce the persistence of the disease when $\beta > \beta_{\max}$. In order to understand the mechanism, we performed extensive simulations

Fig. 1 An illustration of the density of infected individuals for fixed τ_I and τ_R with $\beta_{\min} = 0.2$ and $\beta_{\max} = 3$. Here, $\tau_I = 0.4$ and $\tau_R = 1$

Fig. 2 Spatial pattern of susceptible (white), infected (red), and recovered (black) individuals for different times. All panels are depicted on a 300×300 spatial grid. Parameter values are: $\beta = 0.1$, $\bar{\beta} = 5$, $\tau_I = 0.4$, and $\tau_R = 1$. **a** $t = 0$; **b** $t = 100$; **c** $t = 200$; **d** $t = 300$; **e** $t = 400$; **f** $t = 500$

Fig. 3 Time series of the density of infected individuals.

Parameter values used are:
 $\tau_I = 0.4$, $\tau_R = 1$, $\beta = 0.1$, and
 $\beta = 5$

Fig. 4 Spatial pattern of susceptible (*white*), infected (*red*), and recovered (*black*) individuals for different times.

All panels are depicted on a 300×300 spatial grid.
 Parameter values are: $\beta = 5$,
 $\beta = 10$, $\tau_I = 0.4$, and $\tau_R = 1$. **a** $t = 0$; **b** $t = 50$;
c $t = 100$; **d** $t = 150$; **e** $t = 200$;
f $t = 300$

Fig. 5 Time series of the density of infected individuals.

Parameter values used are:

$\tau_I = 0.4$, $\tau_R = 1$, $\beta = 5$, and $\bar{\beta} = 10$

with different parameter values (in total, more than 200 different values of β and $\bar{\beta}$ are examined). We find that when $\bar{\beta}$ is sufficiently large, the density of infected individuals will be larger than zero in the limit. In Fig. 4, we show the evolution of the spatial pattern of the infected population at $t = 0, 50, 100, 150, 200$, and 300 , with $\beta = 5$ and $\bar{\beta} = 10$. It can be seen from this figure that a stable target wave prevails in the two-dimensional space, which means that the infection density is high and the disease will persist. Time series of the infected population is shown in Fig. 5.

In order to demonstrate the effect of reinfection on the persistence of the disease, we show the invasion regime of the disease with respect to the two parameters β and $\bar{\beta}$ in Fig. 6 with $\beta < \beta_{\min}$. One can see that, for small $\bar{\beta}$, the density of disease is still zero. However, for larger $\bar{\beta}$, the disease will persist.

Fig. 6 Extinction and persistence regimes of disease in parameter space. Domains I and II correspond to the extinction and persistence regimes, respectively. Other parameter values used are: $\tau_I = 0.4$ and $\tau_R = 1$

4 Discussion and conclusions

In summary, a spatial epidemic model with reinfection is investigated. The numerical results show that the reinfection can induce the persistence of the disease. That is to say, reinfection plays a notable role in epidemic spreading, which may well explain observations in the real world [25–27].

From Fig. 6, we can see that when the reinfection rate is small, the disease will become extinct. To control the disease, we should try to reduce the reinfection rate by increasing the amount of vaccination, giving more publicity to disease or enlarging the quarantine rate.

Spatial epidemics via local dispersal abilities can have waves and regular patterns as well as self-organized patterns [6, 16, 28–30]. The analytical expressions for the wavelengths of the spirals can be determined by a complex Ginzburg–Landau equation [31–34], which will be addressed in future studies.

Acknowledgements We are grateful for the constructive suggestions of the anonymous referee on our original manuscript. This work is supported by the National Natural Science Foundation of China under Grant No. 60771026 and by the International and Technical Cooperation Project of Shanxi Province (2010081005).

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