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Effects of prevention and quarantine for SIS model on a square lattice

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Abstract: The spatial and temporal dynamics for epidemic diseases have growing interest. A variety of theoretical models have been presented by many authors. Examples are SIR, SIS, SIRS models. By the use of these models, both effects of prevention and quarantine have been explored for the suppression of disease. Here, the term "prevention" denotes that the susceptible person behaves not to be infected; examples are vaccination and preventable behaviors. In contrast, we use the term "quarantine" as the decrease of infection opportunity; if people avoid the interactions, the infection will be reduced.

In the present paper, we study the SIS model on a square lattice: It is called "contact process" or "lattice logistic model." The contact process has been extensively investigated by many fields, such as mathematics, physics and ecology. Each lattice site takes one of three states: susceptible (S), infected (I) and prevention (P) sites. Infection is assumed to occur between S and I at adjacent sites: no infection occurs for P. To explore both effects of prevention and quarantine, we apply the site and bond percolations respectively. Computer simulations reveal that the system evolves into an equilibrium state. When the infection rate β increases, or when the recovering rate γ decreases, then the equilibrium density of I increases. The final equilibrium state becomes either infectious or disease-free phase. The boundary between both phases can be represented by a scaling law. The mean-field theory well predicts such infection dynamics and the scaling law. However, the theory never predicts the following "percolation thresholds": When both levels of prevention and quarantine exceed a threshold (percolation threshold), the disease is effectively suppressed irrespective of the values of β and γ. The percolation means the spatial connection of protected people which cooperatively prohibits the infection.

Keywords: *SIS model, prevention, quarantine, percolation threshold, scaling law*

1. INTRODUCTION

The threat of infectious diseases remains violent. Prevention of infectious (contagious) disease is an important problem. Typical defensive methods are prevention and quarantine. Here the "prevention" denotes the susceptible person who behaves not to be infected. Examples are vaccination and preventable behaviors. In contrast, the quarantine means the decrease of infection opportunity. Infection may be effectively suppressed, if the levels of prevention and quarantine exceed thresholds. So far such a threshold phenomenon has been explained by many theories (Kermack and McKendric, 1927). In the present paper, we report a threshold phenomenon originated in the "percolation transition". The percolation means the spatial connection of prevention people. They cooperatively (collectively) prohibits the infection of disease.

There are two major theoretical approaches to study the dynamics of epidemic diseases: spatial and non-spatial theories. In most cases, they have applied (partial) differential equations (Stone et al, 2007). In the present paper, however, we apply a stochastic cellular automaton for SIS model (Harris, 1974). In the next section, we describe simulation methods. Both effects of prevention and quarantine are estimated by site and bond percolations, respectively. In the section 3, the mean-field theory is presented. The section 4 is devoted for the report of results. We mainly report two results: a scaling law and the percolation threshold.

2. MODELS AND METHODS

2. 1. Prevention Model

The term "prevention" denotes that the susceptible person behaves not to be infected. A typical example is vaccination. Another example is preventable behaviors. Consider the SIS model on a lattice. It is often called "contact process" or "lattice logistic model." Each lattice site is labeled by susceptible (S), infected (I) and prevention (P) sites. The interactions are defined by

$$S + I \rightarrow 2I \qquad (\text{rate } \beta) \qquad (1a)$$

$$I \rightarrow S \qquad (\text{rate } \gamma) \qquad (1b)$$

The reactions (1a) and (1b) respectively mean the infection and recovery processes. The parameters β and γ thus represent the infection and recovery rates, respectively.

Simulation is performed by the contact process (lattice logistic model) (Harris, 1974; Konno, 1994):

1) Initially, we randomly distribute S, I and P on a square-lattice. The location and the density p of prevention site are unchanged throughout the simulation.
2) Each reaction process is performed in the following two steps
   (i) We perform the infection process (1a). Choose one square-lattice point randomly, and then specify one of nearest-neighbor points. When the pair is (I, S), then the site S will become I by the probability β. Here we employ periodic boundary conditions.
   (ii) Next we perform the recovery process (1b). Choose one lattice point randomly. If the point is occupied by I, then it becomes S by the probability γ.

3) Repeat step 2) by L^2 times, where L^2 is the total number of square-lattice sites. This step is called the Monte Carlo step (Tainaka 1988).

4) Repeat the step 3) for 2000-3000 Monte Carlo steps.

2. 2. Quarantine Model

The term "quarantine" is used as the decrease of infection opportunity. If people reduce the interaction, the infection will be reduced. We put "barrier" on a bond. Namely, we put barrier between a pair of neighboring lattice sites. The probability of barrier on each bond is defined by p. In the case of quarantine model, each lattice site is labeled by susceptible (S) and infected (I) sites; there is no prevention site. The interactions are the same as defined by (1a) and (1b). The simulation method is similar as the prevention model, but the steps 1) and 2) (i) are different as follows:

1) Initially, we randomly distribute S and I on a square-lattice. We randomly put barriers between a pair of adjacent sites. The location and the barrier density p are unchanged throughout the simulation.

2) (i) We perform the infection process (1a). Choose one square-lattice point randomly, and then specify one of nearest-neighbor points. When the pair is (I, S) and when there is no barrier between them, then the site S will become I by the probability β.

Typical examples of spatial pattern are illustrated in Fig. 1, where (a) and (b) are prevention and quarantine models, respectively. In Fig. 1 (a) the white sites denote the prevention, while in Fig. 1 (b) the white bonds represent the quarantine.

In the field of physics (Stauffer, 1985), it is well known that the distribution of prevention sites or barriers shows "percolation transition". When the density p takes an extremely small value, no prevention sites (barriers) may connect with each other. On the contrary, when p takes a large value near unity, almost all barriers are connected. Below, we call cluster for a clump of each connection, and percolation in the case that the largest cluster reaches the whole size of system. The probability of percolation takes a nonzero value, when p exceeds a critical point p_C. It is known that $p_C \approx 0.6$ for site percolation (prevention model), and $p_C = 1/2$ for bond percolation (quarantine model) in the square lattice. Percolation means that the region of susceptible site (S) may be fragmented into small segments for $p > p_C$.

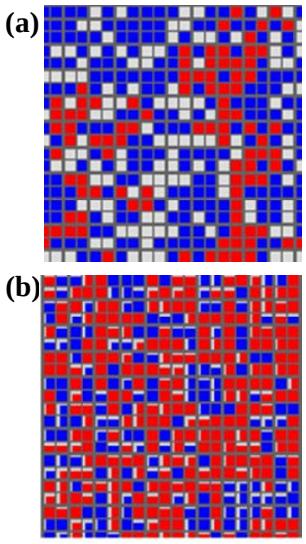

Figure 1. Prevention and quarantine models on a lattice. The spatial patterns for (a) prevention and (b) quarantine models are illustrated, respectively. In (a) the white denotes the prevention site, while in (b) the white bonds represent the quarantine. Both red and blue mean susceptible (S) and infected (I) sites, respectively.

3. MEAN FIELD THEORY

3. 1. Prevention Model

It is well known that the mean field theory (MFT) is a first and crude approximation in systems with short-range interactions (local interaction). In MFT, the infection is assumed to occur between any pair of lattice sites (global interaction). Time evolution for the prevention model is represented.

$$\dot{x} = \beta(1-x-p)x - \gamma x \quad (2)$$

where x is density of I, and the dot denotes the derivative with respect to the time t. The term $(1-x-p)$ thus means the density of S. The first and second terms in the right-hand side of equation (2) come from the reaction (1a) and (1b), respectively. Equation (2) is equivalent to the logistic equation, and the dynamics is well known. The infection density x eventually evolves into a stationary state (equilibrium). The equilibrium density can be obtained by setting the time derivative in (2) to be zero. It follows that

$$x = (1-p) - \gamma/\beta \quad \text{for } (1-p) > \gamma/\beta \qquad (3a)$$

and

$$x = 0 \quad \text{for } (1-p) \le \gamma/\beta \,. \quad (3b)$$

Hence, the final equilibrium state becomes either infectious or disease-free phase. The boundary between both phases can be represented by $\beta(1-p)/\gamma = 1$. When we put

$$\lambda \equiv \beta(1-p)/\gamma \,, \qquad (4)$$

Thus, λ represent the basic reproduction number (R_0). The disease prevails for $\lambda > 1$, whereas it never exists for $\lambda \le 1$.

3. 2. Quarantine Model

In the case of quarantine model, MFT neglects the detail information for barrier locations. Barriers are placed uniformly (probabilistically), according to its density (p). The mean-filed theory (MFT) can be represented by

$$\dot{x} = \beta(1-p)(1-x)x - \gamma x \,, \qquad (5)$$

where the factor $(1-p)$ denotes the probability that the barrier is absent between a pair of adjacent sites, and $(1-x)$ is the density of S. The equilibrium density can be expressed as follows:

$$x = 1 - 1/\lambda \quad \text{for} \quad \lambda > 1 \quad (6a)$$

and

$$x = 0 \quad \text{for} \quad \lambda \le 1 \,. \qquad (6b)$$

Therefore MFT predicts that the phase boundary takes the same value ($\lambda = 1$) for both prevention and quarantine models.

4. SIMULATION RESULTS

4. 1. Results for Prevention Model

The population dynamics for the prevention model is very similar to the mean-field theory; the lattice system evolves into a stationary state (equilibrium). However, the steady-state density obtained by lattice simulation takes a different value from the prediction of theory. In Fig. 2(a), the steady-state density is plotted against β/γ for various values of parameter p r, where each plot is obtained by averaging not only over the period $2000 \le t \le 4000$ but also over 10 different distributions ("ensembles") of prevention sites. The ensemble average is necessary, because in the case of a large value of p , the equilibrium density takes slightly different values for different ensembles. This figure exhibits a phase transition between a phase where disease survives and a phase where it is free. Figure 2(b) is the same as Fig. 2(a), but the horizontal axis in (b) rescaled by λ [see equation (4)] . For the sake of comparison, the results of mean-filed theory (MFT) are also depicted. We find from Fig. 2(b) that the threshold λ_C between both phases is unchanged irrespective of the value of p . Such a scaling law is qualitatively predicted by MFT, but there is a quantitative difference between simulation results ($\lambda_C \approx 1.7$) and MFT theory ($\lambda_C = 1$).

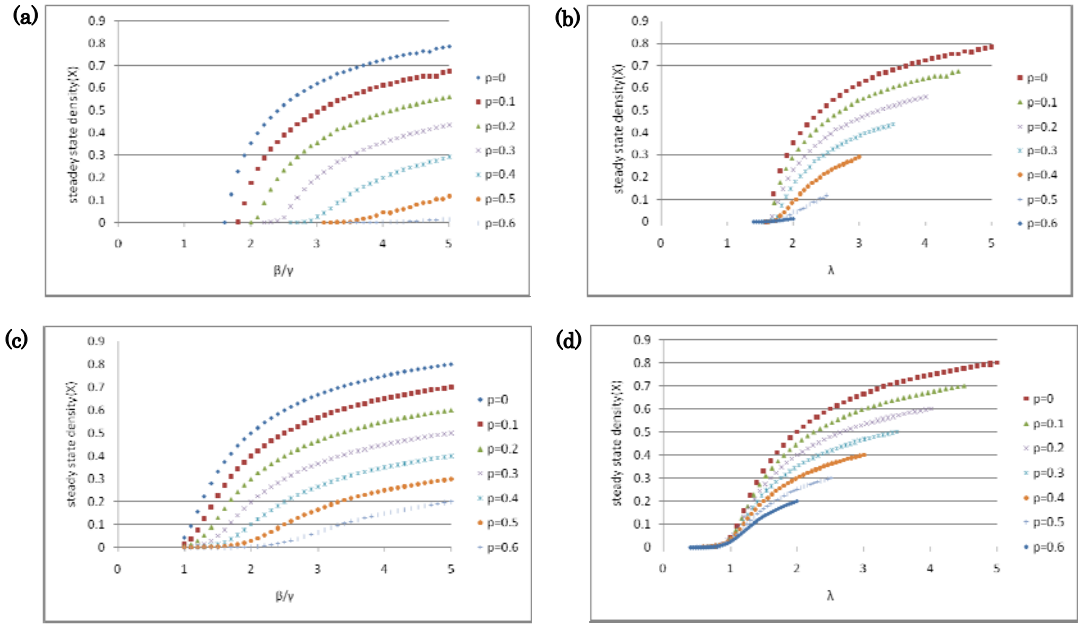

Figure 2. Results for prevention model. The steady-state (equilibrium) density of infected site is shown. Here (a) and (b) are simulation results of lattice model ($L = 100$), and (c) and (d) are the predictions of mean-field theory. In (a) and (b) the horizontal axis denotes β/γ, while in (a) and (b) it denotes λ defined by equation (4).

Next, we report a qualitative difference between theory and simulation: MFT cannot predict the percolation transition. In the case of MFT, the disease survives so long as $\lambda > 1$. Irrespective of the value of p, the equilibrium density of I sites can be expressed as $x = 1 - 1/\lambda$. However, in the case of simulation (lattice model), the disease disappears, when the density p is significantly larger than transition point of percolation. Even though $\lambda > \lambda_C$ ($\lambda_C \approx 1.7$), the disease disappears. In Fig. 3, the disappearance dynamics is illustrated for $\lambda > \lambda_C$. If the density p is sufficiently large ($p = 0.7$), the disease density x eventually reaches zero, in spite of a high value of infection rate ($\beta / \gamma = 10$).

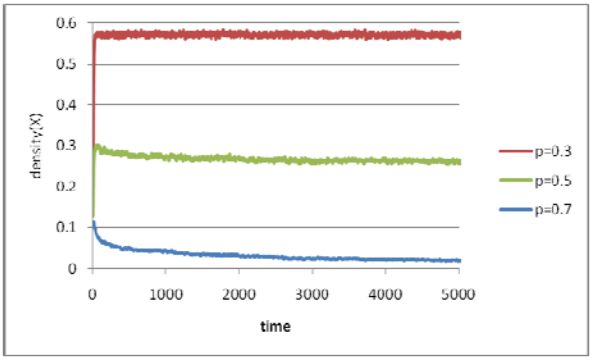

Figure 3. The dynamics in prevention model at high infection rate ($\beta / \gamma = 10$). The time dependences of disease density x are plotted for various levels of prevention. The disease disappears in spite of $\lambda > \lambda_C$. If the density p is sufficiently large ($p = 0.7$), the disease density x eventually reaches zero.

4. 2. Results for Quarantine Model

Steady-state densities for quarantine model differ from those for prevention version. In Fig. 4, we plot the steady-state density obtained from simulation and mean-field theory. We find from Fig. 4(b) that the threshold λ_C between surviving and disappearing phases is unchanged irrespective of the value of p. Moreover, we have $\lambda_C \approx 1.7$ which is the same threshold as for prevention model.

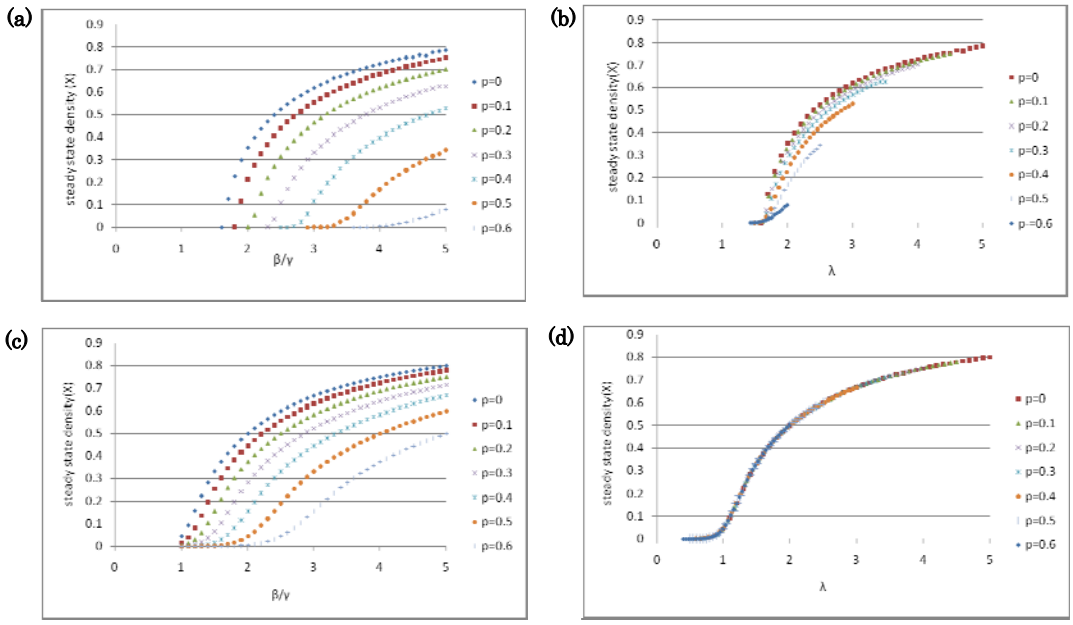

Figure 4. Same as Fig. 2, but for quarantine model.

5. CONCLUSIONS

We have presented the SIS model on a square lattice: It is called “contact process” or “lattice logistic model.” The contact process has been extensively investigated by many fields, such as mathematics (Harris, 1974; Liggett, 1985), physics (Konno, 1994). To explore both effects of prevention and quarantine, we apply the site and bond percolations respectively. Computer simulations reveal that the system evolves into an equilibrium state. When the infection rate β increases, or when the recovering rate γ or prevention (quarantine) density p decreases, then the equilibrium density X of infection sites increases. The final equilibrium state becomes either infectious or disease-free phase. The boundary between both phases can be represented by the scaling law: $\lambda = \lambda_C$ ($\lambda_C \approx 1.7$). Here $\lambda = \lambda_C$ is defined by equation (4). The mean-field theory (MFT) well predicts such infection dynamics and the scaling law ($\lambda_C = 1$).

However, the theory never predicts the percolation thresholds: The percolation means the spatial connection of prevention sites or quarantine bonds. The MFT fails, when the level p of prevention or quarantine is sufficiently high. If p exceed a threshold, the disease is effectively suppressed irrespective of the values of β and γ. The prevention sites or quarantine bonds cooperatively (collectively) prohibit the infection of disease. Our model contains some oversimplifications. For example, the infection occurs between adjacent sites. More refined models should be necessary.

Finally we discuss the percolation (connection) of susceptible (S) sites. We compare the results in Fig. 2(a) with those in Fig. 4(a). From this comparison, we notice that the prevention is more effective than quarantine. Such a result is paradoxical, since p_C for prevention model is higher than that for quarantine model. The paradox can be solved, if we take into account the percolation of susceptible sites. The percolation transition point of susceptible sites occurs at $p_C \approx 0.4$ for site percolation (prevention model), and $p_C = 1/2$ for bond percolation (quarantine model). Hence, the prevention is more effective than quarantine.

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