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Combined effects of prevention and quarantine on a breakout in SIR model

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Recent breakouts of several epidemics, such as flu pandemics, are serious threats to human health. The measures of protection against these epidemics are urgent issues in epidemiological studies. Prevention and quarantine are two major approaches against disease spreads. We here investigate the combined effects of these two measures of protection using the SIR model. We use site percolation for prevention and bond percolation for quarantine applying on a lattice model. We find a strong synergistic effect of prevention and quarantine under local interactions. A slight increase in protection measures is extremely effective in the initial disease spreads. Combination of the two measures is more effective than a single protection measure. Our results suggest that the protection policy against epidemics should account for both prevention and quarantine measures simultaneously.

Recently human society has been threatened by repeated pandemics and heavy epidemics of several new infectious diseases, e.g., AIDS, SARS, swine flu. The measure of protection against these new diseases is an urgent global issue in clinical and epidemiological studies1–5. We consider prevention and quarantine, the two major protection approaches. Prevention is characterized as the prepared protection before a disease spreads, such as masks, hand-washing, and gargling. Vaccination is the most effective method of prevention for a prospective infected person. Here prevention is defined as the complete protection of a person against infection. On the other hand, quarantine is the strict isolation imposed on a person to prevent the spread of disease. By isolating a contagious infected person, quarantine reduces further transmission to susceptible people.

We evaluate the effects of either prevention, or quarantine, or the both. We study the infection dynamics of an SIR model⁶. In epidemiology SIR is the simplest model for many contagious (mostly respiratory) diseases with recovery process. The spatial versions of SIR models are studied extensively in biomathematics⁷–⁹ and in physics¹¹–¹⁵.

We introduce site percolation for prevention and bond percolation for quarantine on a lattice space¹⁶ (Fig. 1). In site percolation (Fig. 1a), every site on a square lattice is independently either “prevented,” with probability $p_S$, or not with probability $1 - p_S$. The prevented site (person) is completely protected from disease, as in vaccination. On the other hand, quarantine is introduced as bond percolation (Fig. 1b), where the “barrier” is placed (or not placed) with probability $p_B$ (or $1 - p_B$) between adjacent sites. The barrier protects the infection (interaction) between neighbors (adjacent sites). Unlike previous studies which introduce either site or bond percolation¹⁶, we introduce both site and bond percolations on a single lattice space (Fig. 1c). We evaluate the effects of both protection measures on the spread and outbreak of diseases. These lattice models are spatial models and often called local interaction¹³. We also calculate the global versions of the lattice models, where all the reactions are performed between two randomly chosen sites¹⁵. They are often called global interaction or lattice gas models and their mathematical solution can be obtained (called mean-field theory). We also calculate the mean-field theory (exact solutions for global interaction) for all the corresponding cases of local interactions.

Results

In the SIR model, the infection phenomena are best understood by the number of the recovered (R) at the final equilibrium, since all the infected (I) are eventually recovered. We find a strong synergistic effect of prevention and quarantine under local interaction. Both measures of protection (prevention $p_S$ and/or quarantine $p_B$) are highly effective (Figs. 2). The total number of the recovered decreases rapidly with an increase in the protection levels (convex down in Figs. 2a and 2b). This means that a slight increase in the level of protection (by either prevention and/or quarantine) will greatly reduce the possibility of initial disease spreading. When both
prevention and quarantine are combined, the number of recovered is always effectively reduced compared with sole protection alone (Figs. 2c). The effects of prevention (site) are always slightly stronger than those of quarantine (bond), when the density of sites and bonds are compared (Fig. S1 in SI).

Under global interaction, the combined measures of both protections are more effective than that of a sole protection (Figs. 2c, 2d, and 2f). However, the overall effects are much weaker compared with those of local interactions (Figs. 2a vs. 2c, 2b vs. 2d and 2e vs. 2f). The recovered decreases almost linearly for prevention (Fig. 2c) and quite slowly (concave down) for quarantine (Fig. 2d). The combined effects under global interaction are always stronger than the sole effects. However, it is much weaker compared with those under local interactions (Figs. 2f vs. 2e). Under global interaction, the effects of prevention (site) are always much stronger than those of quarantine (bond), when the density of sites and bonds are compared (Figs. 2f, see also Fig. S1 in SI).

We also evaluate the effects of prevention when prevention sites are introduced at a given rate (Fig. 3). When the delayed time (τ) to reach at $p_S=0.5$ is changed from $\tau=0$ (no delay: the case of Fig. 2a) to $\tau=1000$ (Fig. 3a), the final density of recovered (total infected) is decreased to a nearly half (Fig. 3b).

If the level of protection is increased slightly, the total infected persons (recovered, R) decrease radically. We compare the protection level where the density of infected is reduced 50% (a half reduction) between the current SIR and SIS models (Fig. 2g). Note that the density of the infected is measured as the final density in the SIR model, while the steady state density in the SIS model. The level of both bond and site percolations are considerably small compared with the global interaction (mean-field theory), with the local interaction of the SIS model. Thus the combined effect is much stronger under local interaction than global interaction (Fig. 2g). Since many common infectious diseases spread with personal contacts, the infection mode should be more like the local interactions. Therefore, the combined approach of both prevention and quarantine is extremely efficient for the measures of disease protection policy.

**Discussion**

The current result is unique to the SIR model, where the infected persons are expected to recover with immediate immunity. In contrast, if a recovered person can be infected repeatedly as in the SIS or SIRS models, the increase in the initial level of protection does not have a strong effect (Fig. 2g, see also Figs. S2 in SI). Therefore, we cannot expect a significant success by combining the two measures. The introduced rate of protection (either prevention or quarantine) is also an important factor affecting the total infected in the SIR model (the result of prevention is shown in Fig. 3). Thus the measure of protection is quite significant in the SIR model, but not in the SIS and SIRS models. In these models, however, the added protection improves the effects slightly as in those under global interactions (see Fig. S2 in SI).

Our results suggest that when the protection measures against infectious diseases are combined simultaneously, the disease control becomes highly effective easily. For example, when quarantine is not perfect as in the case of SIRS, the ordinary prevention of infection spread could be still extremely effective, such as gargling, hand-washing, and wearing a mask. In the recent spread of the swine flu, these ordinary measures might have put down the infection level.
Figure 2 | The final density of the recovered sites (R) for the combined model of both prevention (site percolation) and quarantine (bond percolation) with a constant infection rate ($\beta/c = 5.0$). (a) local interaction (simulation) and (b) global interaction (mean-field theory analyses) against the density of prevention $P_s$, where $P_b = 0$ (blue), 0.2 (red), 0.4 (green), 0.6 (purple) and 0.8 (yellow). (c) local interaction (simulation) and (d) global interaction (mean-field theory analyses) against the density of quarantine $P_b$, where $P_s = 0$ (blue), 0.2 (red), 0.4 (green), 0.6 (purple) and 0.8 (yellow). Phase diagrams along with the densities of both prevention ($P_s$) and quarantine ($P_b$) for (e) local interaction by simulation and (f) global interaction by mean-field theory (densities of the recovered (R) are shown in colors: blue = 0–0.2; red = 0.2–0.4; green = 0.4–0.6; purple = 0.6–0.8; pail blue = 0.8–1.0). (g) comparisons with the steady state density of SIS models for a half (50%) reduction in infection level for both global and local interactions (Black solid line: SIS for local; black dashed line: SIS for global; red solid line: SIR for local; and red dashed line: SIR for global). The combined effect is the largest for the local SIR model; a half (50%) reduction is achieved merely by $P_s = P_b = 0.1$ or less.
Furthermore, we have an advantage in the economic aspect of disease control. A combined method of control might not only work better, but may also be more cost effective. For example, the costs for vaccination per person increase rapidly with the number of the vaccinated persons. But by investing equally in prevention and quarantine may be more cost effective than vaccinating twice (prevention alone). Certainly, there are many complicated issues regarding the introduction of protection measures. Many factors need to be taken into consideration, e.g., costs, uptake levels by the populace, time-scale of introduction, feasibility. However, the current result is still valid: introducing and combining any measures of protection is worth trying if available without difficulty.

Against epidemics, it is very important to introduce any additional measure of protection as long as we expect even a very slight effect. In the SIR type diseases, an added measure may be a key to the successful protection approaches.

**Methods**

**Basic structures and mathematical models.** We consider the SIR model on a square lattice:

\[
S + I \rightarrow 2I \quad \text{(rate } \beta) \\
I \rightarrow R, \quad \text{(rate } \gamma)
\]

where the susceptible (S) becomes the infected (I) with the infection rate \( \beta \) in the infection process \( (1a) \), while the infected (I) becomes the recovered (R) with the recovery rate \( \gamma \) in the recovery process \( (1b) \).

With prevention, the prevented person (white site in Fig. 1a and 1b) becomes completely inactive. For quarantine under local interaction, the infection (equation \( 1a \)) is inhibited between persons (grey bond between site in Fig. 1b and 1c). For quarantine under global interaction, the average infection rate \( \langle \beta \rangle \) is reduced between two randomly chosen persons depending on the total number of barriers (inactive bonds).

The mean-field theory (MFT) is the analytical version of global interaction\(^{12} \), which is the first and crude approximation for local interaction. Time evolution can be expressed as:

\[
x = \beta(1 - P_S)(1 - x - y - P_R)x - \gamma x
\]

where \( x \) and \( y \) are the density of I and R, respectively and the dot denotes the derivative with respect to time \( t \). The term \( (1 - x - y - P_R) \) 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 that can be solved easily. Since the results of global simulations agree well with the mean-field theory, we use the latter results for discussion.

**Simulation procedures.** The simulation procedure for local interaction is listed below:

1) Initially, we randomly distribute the prevention site \( P_S \) on a square-lattice with a given density \( P_S \). Next, we randomly put the “barrier” \( P_B \) on a bond (link between adjacent sites). The locations of \( P_S \) and \( P_B \) are unchanged throughout a simulation. We then distribute S and I randomly on the remaining sites.

2) Reaction processes are performed in the following two steps.

   (i) the infection process \( (1a) \). First, we choose one square-lattice point randomly, and then specify one of four adjacent points. When the pair is \( (I, S) \) without the barrier \( P_B \), then the site \( S \) will become I with the probability \( \beta \).

   (ii) the recovery process \( (1b) \). We choose one lattice point randomly. If the point is occupied by I, then it becomes R with the probability \( \gamma \).

3) Repeat step 2) by \( L^2 \) times, where \( L^2 \) is the total number of cells. This step is called the Monte Carlo step which is used for time unit\(^{16} \).

4) Repeat the step 3) until the system reaches a stationary state.

The procedure for global interaction is almost the same, except the following two points in the procedure 2(i). One is the choice of two points that are randomly chosen from the entire lattice space. The other is the infection rate \( (1 - P_B) \beta \). The examples of temporal dynamics are shown for local interaction in Fig. 1d and for global interaction in Fig. 1e. The initial density of I is set at 0.01 in all simulation runs.

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Author contributions
F. K., K. T. and J.Y. develop the models. F.K. runs simulation. S. S., S. M., H. I. and K. T. survey the previous models and compare them with the current models. All authors contribute manuscript preparation. F. K. and K. T. both contributed equally.

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