Replication

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

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**In recent decades, using SIR models in order to predict some properties of epidemics has been of serious consideration, due to vast majority of outbreaks all around the world. The most recent one is COVID-19 epidemic. Therefore, applying protection policies against these epidemics has been one of the most focused aims of studying them by SIR model. In this article, we shall consider two forms of protection, which are prevention and quarantine. We'll use lattice model, including site percolation for prevention and bond percolation for quarantine and investigate their amount of effectiveness when one or two of them are applied**.

As mentioned before, we've utilized site percolation and bond percolation, simulating prevention and quarantine in Lattice space, respectively. In site percolation, every site can be protected against the disease with a probability, $P_S$, and not protected with a probability, $1-P_S$ (Fig. 1a). Prevention is characterized as the prepared protection before a disease spreads, such as masks, hand-washing, etc. In this model, when prevention is achieved, the site is completely protected, let's say, by vaccination; since, it's the most effective means of protecting one's self.

When dealing with bond percolation, there are two probabilities of existing a barrier between two adjacent sites (or so-called, neighbours), $P_B$, and not existing a barrier between them, $1-P_B$ (Fig. 1b). When intending to show both means of protection on a single lattice, Fig. 1c is obtained.

These lattice models are spatial models and often called local interaction. The Global version of the lattice models' results, which represent the global interaction – that is, all the reactions are between two randomly chosen sites, instead of neighbours- are mathematically calculated by the mean-field theory. Further comparisons of these two models and the measure of effectivity of these two protection policies will be further discussed in the article.

## Discussion

As in SIR model, immediate immunity is achieved when one is recovered, there are unique results that belong only to this model. On the other hand, because recovered individuals become infected over and over again in SIS and SIRS models, the initial level of protection's increment doesn't have a strong effect. Hence, no significant success is to be observed by combining the two measures. The introduced rate of either means of protection in SIR models is very important in the dynamics of the total infected. The results for one of these (prevention) is displayed in Fig. 4.

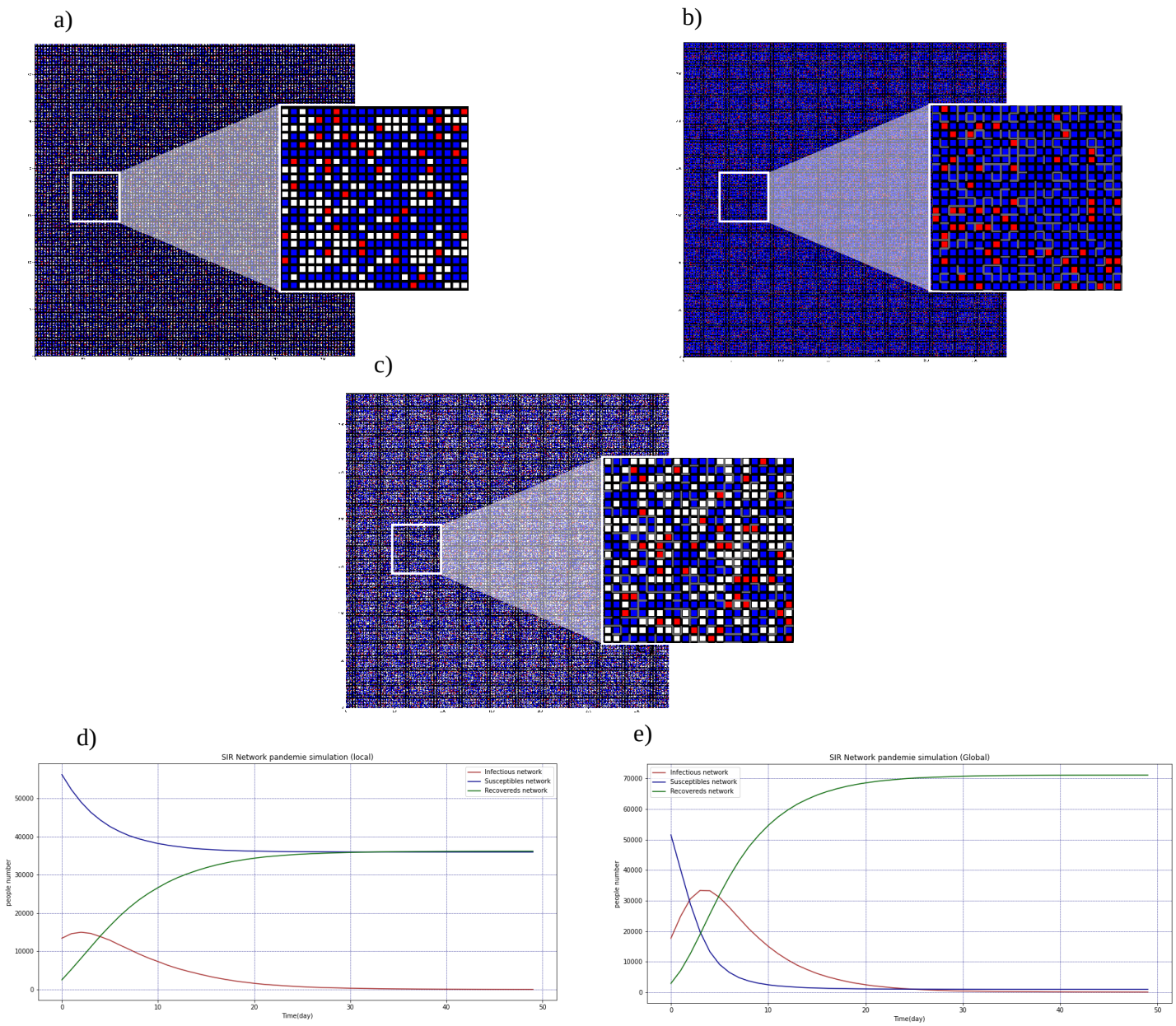



Fig. 1 | **The images and dynamics of SIR model with site and/or bond percolations.** The images are (a) site only. (b) bond only. (c) bond and site. Sits are displayed as the prevented, the susceptible and the infected in colors white, blue and red, respectively. The bonds are shown as a thick grey bar. The time-related dynamics are shown for (d) local and (e) global interactions. Parameter conditions are: $\beta = 1.0$ , $\gamma = 0.2$ , $I0 = 0.1$, $\mathrm{PS} = \mathrm{PB} = 0.35$ , The network size = 333×333. (All these dynamics are network and these are the results of one run only.)

Although, not such effectivity is observed in SIS and SIRS models. In these models, however, the added protection improves the effects slightly as in those under global interactions.

The results imply that in pandemics, when the protection measures are combined simultaneously, they become highly effective and sometimes in the meantime, even more cost effective. i.e., many times, instead of vaccinating the whole population, a more cost-effective way of dealing with the situation is to equally invest in vaccination and quarantine.

In the time of facing an epidemy, many considerations must be taken into account and it is crucial to use as many control-measures as possible; Especially in the case of SIR model's diseases, where any additional policy is vital to be concluded.

## Methods

The main focus of this article is based on SIR

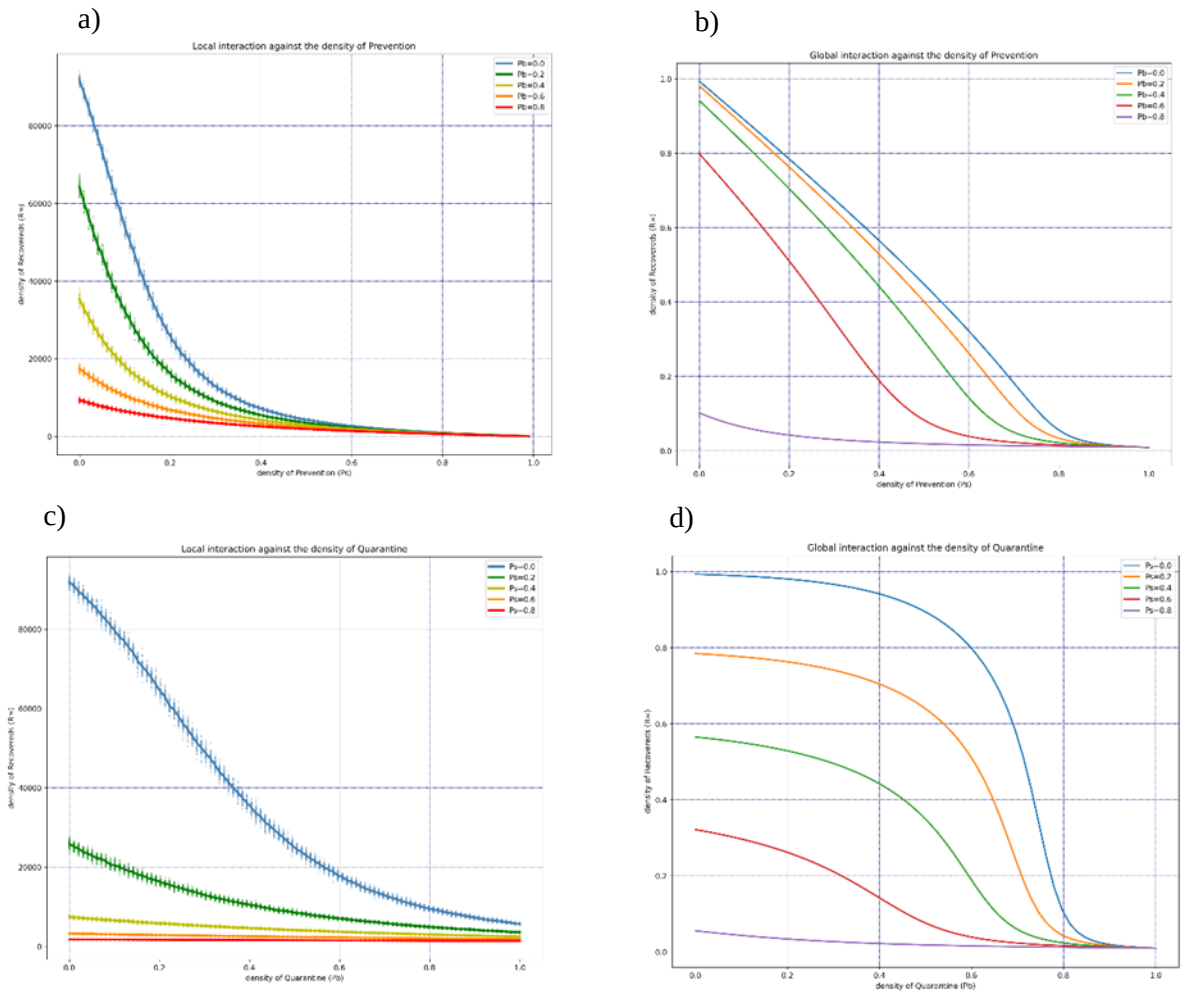
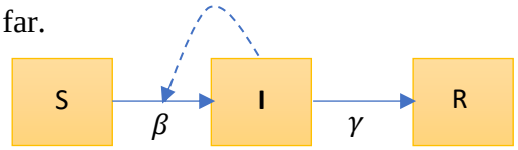



**Fig 2 | The final density of the recovered sites (R) for the combined model of both prevention and quarantine with a constant infection rate $\frac{\beta}{\gamma} = 5$.** (a) local interaction (simulation) and (b) global interaction (mean-field theory analyses) against the density of prevention $P_S$, for different $P_B$ measures. (c) local interaction (simulation) and (d) global interaction (mean-field theory analyses) against the density of quarantine $P_B$, for different $P_S$ measures. $\beta = 1.0\,, \gamma = 0.2\,, I0 = 0.01$, The network size = 333×333.

models, which the results have been displayed so far.




So, we have the following equations of the standard SIR model without demography:

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - \gamma I \qquad (1)$$
$$\frac{dR}{dt} = \gamma I$$

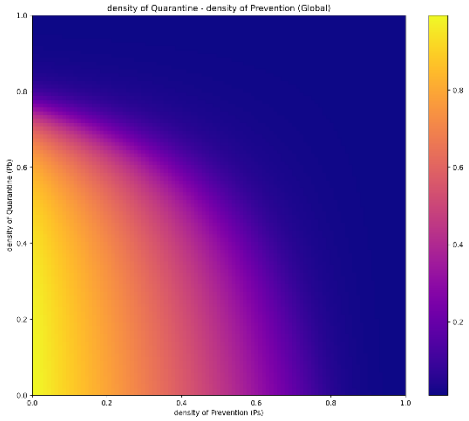
Where, susceptible individuals (S) become infected at rate $\beta$ and infected individuals recover at rate $\gamma$.

While discussing the procedures, we realized that for the global part, the mean-field theory can be applicated as a substitute for Network model, as their results are the same.

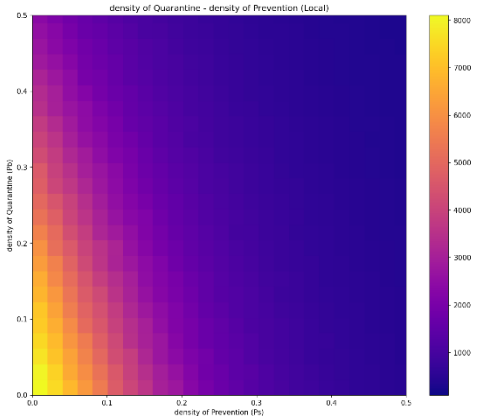
Accordingly, there are two things we can do:

1. Using $\beta(1\text{-}P_B)$ instead of $\beta$. In which, $\beta$ is the transition rate and $P_B$ is the probability of existing a barrier between two nodes.

a)




b)




c)

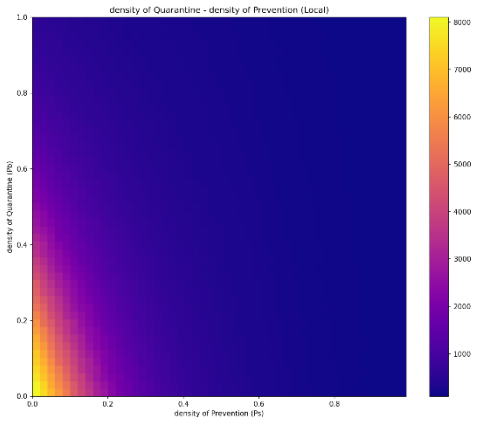



**Fig. 3 | Phase diagrams** along with the densities of both prevention ($P_S$) and quarantine ($P_B$) for (a) Global interaction by mean-field theory, (b) Local interaction by simulation and (c) the scaled version of (b) (densities of the recovered (R) are shown in the color-bar). The network size for (b) and (c) is 100×100 and for (a) is 333×333, Constant infection rate and $\frac{\beta}{\gamma} = 5$.

2. Since in order to influence on the dynamic through $P_S$, we should study the S sites, the following equations must be changed with the standard SIR equations we've introduced before.

$$\dot{S} = -\beta(1 - P_B)(1 - I - R - P_S)I$$

$$\dot{I} = \beta(1 - P_B)(1 - I - R - P_S)I - \gamma I \quad (2)$$

$$\dot{R} = \gamma I$$

In which, S, I and R are defined as the density of the proportion of the people in each class; and the term $\beta(1 - P_B)$ adds the barriers and $(1 - I - R - P_S)$ adds the prevention.

## Simulation Procedure

### The Initial Conditions:

As a means to create the initial conditions, random nodes with the density of $P_S$ are chosen. All these nodes will be out of the dynamic and the operation of converting nodes into a prevented mode is done only once, at the beginning. One point of doing this is that the calculation reduces noticeably and the run-time at great scales will decrease as well.

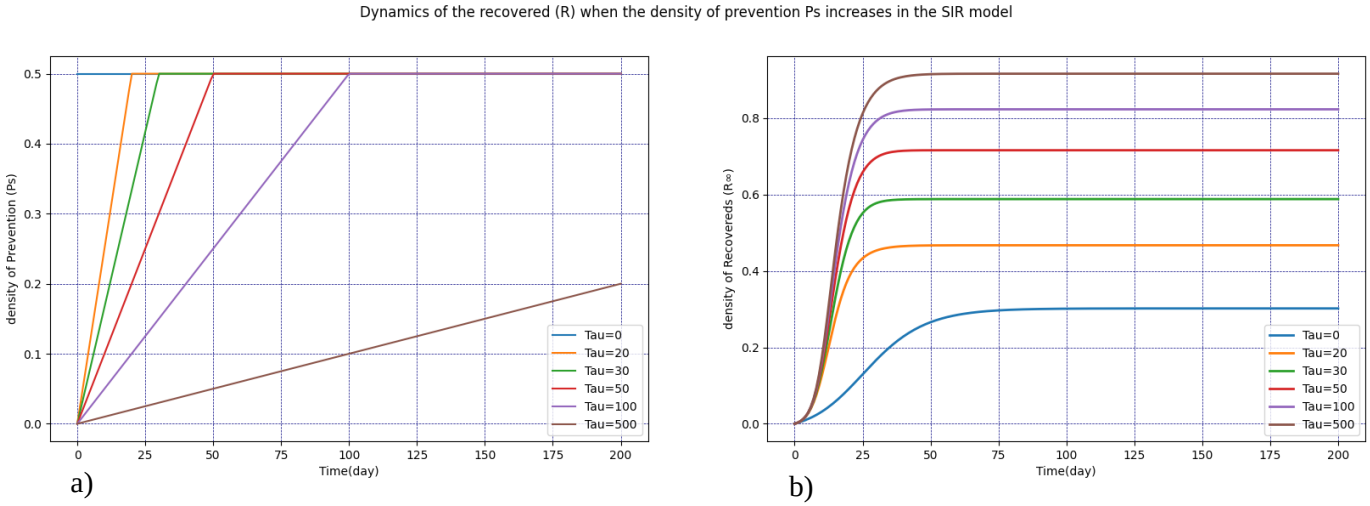



**Figure 3 | Dynamics of the recovered (R) when the density of prevention $P_S$ increases in the SIR model.** (a) The time-related dynamics of prevention and (b)the recovered density (R). The final proportion of prevention (site blocked) is set at $P_S = 0.5$ and the rate $\tau$ is defined as the time to reach at $P_S = 0.5$. Four different delayed times $\tau$ of protection are used for comparisons. $P_B$=0.4.

Barriers are randomly placed on edges with the density of $P_B$; so that, throughout the dynamic these connections are blocked and no transition can happen.

At last, the initial infected are randomly with the density of I0 are placed in the network.

**Infection Transition:**

One node is randomly chosen. If the node is infected, then every susceptible neighbour can become infected with the probability of transition, $\beta$.

**Recovery:**

If the nodes are infected, then they will recover at the rate of $\gamma$, and enter the R class.

All these stages will be iterated for $L^2$ times (until equilibrium is reached), which is the total number of sites (so-called the Monte Carlo step).

For the global version, one of the methods is instead of choosing one node and studying its neighbours, 2 random nodes are selected. If there exists an effective connection (a kind of connection where there are no barriers in between) and one of the two are infected (I-S or S-I), the site which is susceptible will become infected with the probability of $\beta$. But there is another method that a reduction in runtime is followed. Ant that is, two nodes are randomly chosen and if one of them is infected and the other susceptible, then with the probability of $\beta(1 - P_B)$, the S will become I.

There are some parameters that remain unchanged along the dynamic. $\beta = 1 \; and \; \gamma = \frac{1}{5}$.

## Results

In SIR model, epidemics are best understood by the number of recovered(R) at the final equilibrium since all the infected (I) are eventually recovered.

From the plots, it's clear that, as the protection level increases slightly, the total recovered is always greatly decreased (Fig. 2a and 2b). furthermore, when both quarantine and prevention is combined, most effective results

are shown compared with sole protection alone (Fig. 3c).

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. 3a vs. 3c). Under global interaction, the combination of protection measures is more effective than only one means of protection (Fig. 2c, 2d and 3a); but the overall effects are even stronger under local interaction (Fig. 2a vs. 2c, 2b vs. 2d and 2d vs. 3a). The recovered decreases almost linearly for prevention (Fig. 2c), but it decreases slowly for quarantine, as it's displayed by the concave plots of Fig. 2d.

We also evaluate the effects of prevention when prevention sites are introduced at a given rate (Fig. 4). When the delayed time ($\tau$) to reach at $P_S$=0.5 is changed from $\tau = 0$ (no delay: the case of Fig. 2a) to $\tau$ =1000 (Fig. 4a), the final density of recovered (total infected) is decreased to a nearly half (Fig. 4b).

We have also compared the protection level between the SIR and SIS models, where the density of infected is half reduced. Consider the fact that the density of infected is measured as the final density in the SIR model, and measured as the steady state density is reached in the SIS model. The level of both means of protection in the local interaction are extremely small compared with the global interaction. Accordingly, the combined effect is much stronger under local interaction than global interaction. Also, since many common infectious diseases spread with personal contacts, and this includes more of dealing the connections between neighbours instead of two randomly chosen sites, introducing local interaction is more suited for this model.


1. Anderson, R.M. & May, R.M. Infectious Diseases of Humans: Dynamics and Control (Oxford Univ. Press, 1991).

2. Nowak, M.A. & May, R.M. Virus Dynamics (Oxford Univ. Press, 2000).

3. Hethcote, H.W. The mathematics of infectious diseases. SIAM Review 42, 599– 653 (2000).

4. Galvani, A.P. & May, R.M. 2005. Dimensions of superspreading.Nature 438, 293– 295 (2005).

5. Choisy, M. & Gue´gan, J.F. & Rohani, P. 2006. Resonance effects and the dynamics of infectious diseases. Physica D 223, 26–35 (2006).

6. Kermack, W.O. & McKendrick, A.G. Contributions to the mathematical theory of epidemics 1. Proc.R Soc Edin A 115, 700–721 (1927).

7. Grassberger, P. On the critical behavior of the general epidemic process and dynamical percolation. Math Biosc 63, 157–172 (1983).

8. Kamp, C. & Bornholdt, S. From HIV infection to AIDS: a dynamically induced percolation transition? Proc R Soc Lond B 269, 2035–2040 (2002).

9. Grassly, N.C. & Fraser, C. Mathematical models of infectious disease transmission. Nature Reviews Microbiology 6, 477–487 (2008).

10. Keeling, M.J. The Effects of Local Spatial Structure on Epidemiological Invasions. Proc R Soc Lond B 266, 859–867 (1999).

11. Cardy, J.L. & Grassberger, P. Epidemic models and percolation. J Phys A 18, L267– L271 (1985).

12. Boccara, N. & Cheong, K. Automata network SIR models for the spread of infectious diseases in populations of moving individuals. J Phys A 25, 2447–2461 (1992).

13. Fuks, H. & Lawniczak, A.T. Individual-based lattice model for spatial spread of epidemics. Discrete Dynamics in Nature and Society 6, 191–200 (2001).

14. Arashiro, E. & Tome, T. The threshold of coexistence and critical behaviour of a predato-prey cellular automaton. J Phys A 40, 887–900 (2007).

15. de Souza, D.R. & Tome, T. Stochastic lattice gas model describing the dynamics of the SIRS epidemic process. Physica A 389, 1142–1150 (2010).

16. Stauffer, D. & Aharony, A. Introduction to Percolation Theory, (Taylor and Francis, London, 1994).

17. Sakisaka, Y. & Yoshimura, J. & Takeuchi, Y. & Sugiura, K. & Tainaka, K. Infection threshold for an epidemic model in site and bond percolation worlds. J Phys Soc Japan 79, 023002 (2010).

18. Coker, R. Swine flu. BMJ 338, b1791 (2009).

19. Satomura, K. & Kitamura, T. & Kawamura, T. & Shimbo, T. & Watanabe, M. & Kamei, M. & Takano, Y. & Tamakoshi, A. Prevention of upper respiratory tract infections by gargling: A randomized trial. American Journal of Preventive Medicine 29 (4), 302–307 (2005).

20. Yamada, H. & Takuma, N. & Daimon, T. & Hara, Y. Gargling with tea catechin extracts for the prevention of influenza infection in elderly nursing home residents: a prospective clinical study. Journal of Alternative and Complementary Medicine 12 (7), 669–672 (2006).