Herd immunity estimation of flu-like disease spreading in SEIR population: The sociophysics modelling via Monte Carlo simulation on discrete-spin model

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Abstract. In this work, the disease spreading under SEIR framework (susceptible-exposed-infected-recovered) and agent-based model was investigated via discrete magnetic spin and Monte Carlo simulation. The defined systems were two-dimensional square-lattice-like, where the spins (representing susceptible, exposed, infected, and recovered agents) were allocated on lattice sites. Taking flu-like disease as a case study, the latent period was fixed at a quarter of infectious period. Then, the system size, the spin population density, and the infectious period were varied to observe its influence on uninfected population. In the simulation, each spin was randomly allocated on the lattice and interacted with its first and second neighbouring spins for disease spreading. The magnetization profiles, representing normalized agents in each state, were recorded. From the results, good agreement between the simulation and real spreading results was qualitatively evident. The uninfected susceptible (survivor) results can be categorized into 2 distinct phases depending on the values of infectious periods. The critical infectious period, which separates low and high survivor phases, was then extracted and power-law scaled with the population density. With this scaling formalism, one can use for specifying the overcrowd situation that conveys epidemic to pandemic, which may benefit epidemiologists and government for future health related policies issuance and deployment.

1. Introduction

Herd immunity is an indirect social-induced immunity, which help protecting susceptible agents in the community from getting disease infection [1]. The herd immunity occurs when a substantial portion of a population (or herd) have immunity (either from vaccination or being recovered from the illness) against the infection. This immune agents then play their roles as protective shield to susceptible agents from infectious agents throughout social interaction. As a result, the herd immunity can effectively stop the disease spreading, which helps people who do not yet develop immunity, e.g. some small children who are too young to be vaccinated, people with immunodeficiency syndrome, and those who are too weak to receive vaccination. However, when immunization rate declines, the herd immunity also falloffs which leads to an increase of new infectious cases. As the herd immunity depends on how immune agents obstruct infectious contacts, to pursue fundamental understanding of how herd immunity varies with the dynamics of the disease spreading is rather complicated, especially in a finite system where fluctuation is important. This is as the number of disease contacts is influenced by many factors, such

as the number of populations, the social interactions, latent period, infectious period, etc. Nonetheless, one way-out to the problem can be arranged by sociophysics.

Sociophysics is a field of science which uses mathematical tools inspired by physics to understand the behavior of human in society. For instance, the disease spreading modeling can be thought of using mathematical tools sharing between the field of epidemiology and physics [2]. Specifically, modern disease spreading techniques can be categorized as deterministic compartment and stochastic agent-based models [3]. The deterministic is to assign rate of state changing among subgroups to calculate number of agents in each state over time, which is comparable with mean-field theory in statistical mechanics. It is applicable for large populations, but usually fails in small group as fluctuation is ignored. In addition, the stochastic agent-based investigates everyone to find the course of disease spreading and is appropriate only for small communities. It is the technique resembling the dynamics of discrete spins in statistical physics, which can be simulated using Monte Carlo simulation. As this work aims to investigate the herd immunity characteristic in finite system associated with the mentioned degrees of freedom, we then used the discrete Potts spin model with stochastic Monte Carlo simulation. The purpose is to model infectious disease spreading in bounded finite system with emphasizing on the herd immunity (interpreted via number of susceptible agent survived from the disease at the end of epidemic, i.e. the survivors). Also, in this work, the common contagious flu-like disease (with latent period of about a quarter of infectious period [4]) was chosen for a case study.

2. Theories and methodologies

In epidemiology, one typically categorizes agents in the system into subcategories corresponding to state of disease spreading dynamics, i.e. the susceptible (S), the exposed (E) the infected/infectious (I), and the recovered (R). With discrete similarities between these subcategories and discrete Potts spin in Physics, the spin model could then be used to investigate the infectious disease system and forms a branch of sociophysics. In this context, the 4-state Potts spin σ becomes appropriate, where its spin states {1,2,3,4} can be used to represent {S,E,I,R} states of the agent. The model for investigating this system can be proposed based on spin Hamiltonian and the change of spin's/agent's states bases on the minimization of a cost function. Specifically, in magnetic system, the cost function can be the Hamiltonian of the subsequent states. Therefore, for disease spreading system, the cost function could be written as functions of spins in SEIR states and the interaction between S and I spins [2],[5]. The change of spin from a current state σ_i to σ_i+1 (S→E, E→I, or I→R) was based on the reduction of

$$\Delta\mathcal{H}_i\left(\sigma_i \to \sigma_i + 1; t \to t + \Delta t\right) = -\delta_{\sigma_i,1} J \sum_{\langle ij \rangle} \delta_{\sigma_j,3} - H\left(t - t_{\sigma_i}^0 - t'_{\sigma_i}\right) \cdot \left(\delta_{\sigma_i,2} + \delta_{\sigma_i,3}\right). \qquad (1)$$

In equation (1), the term $-\delta_{\sigma_i,1} J \sum_{\langle ij \rangle} \delta_{\sigma_j,3}$ refers to interaction between S and I agents. The parameter $J = 1$ (being the interaction strength among spins) was set as unit of the interaction strength. The sum $\langle ij \rangle$ takes only on 1st and 2nd nearest neighbour spins to mimic the real space disease transmission. The spin σ_i denotes the agent positioning on location i, with value being one of the Potts' states. The Kronecker delta function is $\delta_{\sigma_i,k} = 1$ for $\sigma_i = k$ and 0 otherwise. Then, $H\left(t - t_{\sigma_i}^0 - t'_{\sigma_i}\right) \cdot \left(\delta_{\sigma_i,2} + \delta_{\sigma_i,3}\right)$ is the temporal self-interacting local field term responsible for the change from E→I (when being in exposed state up to latent period) or I→R (when being infectious up to infectious period). H is the Heaviside step function which is 1 for $t \geq t_i^0 - t'_i$ and 0 otherwise. The parameter t_i^0 denotes the initial time for the spin being in the state σ_i, while t'_{σ_i} is the latent or infectious periods for σ_i = 2 or 3, respectively. For σ_i = 1 or 4, t'_{σ_i} is discarded as $\left(\delta_{\sigma_i,2} + \delta_{\sigma_i,3}\right) = 0$.

Then, to update the system via Monte Carlo simulation [5], the initial conditions were firstly set. Specifically, the system size ($N = L^2$), the spin concentration (c), and infectious period (D) were varied from L = 100 to 200, c = 0.001 to 0.100, and D = 1 to 600 Monte Carlo steps per spin (mcs), respectively. As considering flu-like disease, latent period was set to 0.25 of the infectious period [4]. Also, one mcs was set as the unit of simulation time, being equal to random allocation of N spins. For each condition,

all $n = cN$ spins (agents), were randomly allocated into the two-dimensional array in computer memory. Then, one spin was assigned in the I state and the other were in the S state. After that, all spins were allowed to have states changed in accordance with the minimization of the Hamiltonian. Specifically, the change of spin state at location i from $\sigma_i \rightarrow \sigma_i + 1$ is allowed only when $\Delta\mathcal{H}_i < 0$. Time to perform this in one round was assigned as 1 mcs. These procedures repeated until there is no I and E agent left in the system (up to 2000 mcs). For each condition, 1000 independent runs were performed to average out random noises, where the sub-category magnetization was extracted, i.e. [5]

$$m^{\mathrm{S}} = \frac{1}{n}\sum_{i=1}^{n}\delta_{\sigma_i,1},\ m^{\mathrm{E}} = \frac{1}{n}\sum_{i=1}^{n}\delta_{\sigma_i,2},\ m^{\mathrm{I}} = \frac{1}{n}\sum_{i=1}^{n}\delta_{\sigma_i,3},\ m^{\mathrm{R}} = \frac{1}{n}\sum_{i=1}^{n}\delta_{\sigma_i,4},\ \text{where } m^{\mathrm{S}} + m^{\mathrm{E}} + m^{\mathrm{I}} + m^{\mathrm{R}} = 1. \quad (2)$$

3. Results and discussions

From the results, the time dependence characteristic of normalized number of agents in {S,E,I,R} states, or $\{m^{\mathrm{S}}, m^{\mathrm{E}}, m^{\mathrm{I}}, m^{\mathrm{R}}\}$, were found in good agreement with typical disease spreading behavior [3]**.** Except only N, the parameters c, D and time are significant parameters. Specifically, as seen in figure 1(a), m^{S} drops due to disease transmission which triggers out the rise of E agents (and m^{E}). Furthermore, when time passes, E agents turn to I agents (see m^{I}), which become new sources of disease transmission. With further decreasing in S agents, m^{I} reaches maximum and declines, resulting in a peak-like function. This is as for I agents being in infectious state equal to D, they recover to R agents with immunity to the disease obtained. Note that S no longer changes when all E and I agents vanish. In addition, since E agents are the result of S-I contacts, it signifies D as a key parameter. By defining the S agents survived from infection as survivor S', the normalized survivor $m^{\mathrm{S}'}$ (= m^{S} at $t \rightarrow \infty$) yields several characteristics with varying D, e.g. see figure 1(b). Specifically, at small D, each I agent has less time to distribute the disease, giving less chance to infect S agents. Hence, S agents have more chances to survive from adopting the disease. However, with longer D, I agents have longer time to spread the disease, and when the D is large enough, all S agents get infected [6], i.e. pandemic situation.

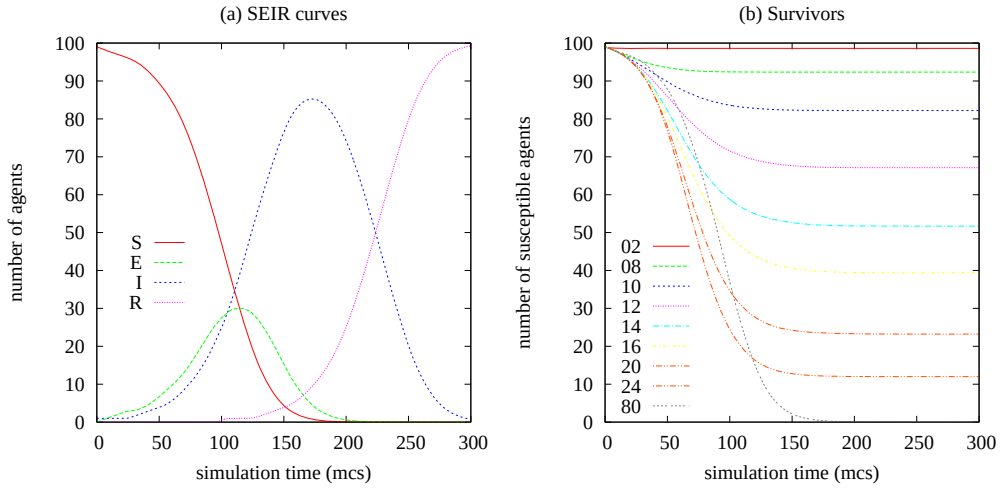

Figure 1. (a) The number of SEIR agents simulated at D of 100 mcs, and (b) the number of S agents with varying D from 2 to 80 mcs. In the figures, $N = 100^2$ and $c = 0.01$ were fixed.

As suggested in figure 1(b), $m^{\mathrm{S}'}$ is a of function D. Interestingly, $m^{\mathrm{S}'}$ presents a somewhat similar behaviour to temperature dependent ferromagnetic magnetization. There are 2 distinct phases associated to high and low survivors, like low and elevated temperature phases in ferromagnetic. Therefore, it is of interest to extract the so called critical infectious period D_C, which separates high survivor phase from low survivor phase. This could be done via the extract of D from where $m^{\mathrm{S}'}$ has maximum gradient. However, due to fluctuations, even the average from 1,000 independent runs, there are still some small rises and falls among data points on D domain. Therefore, the average data was smoothed and

interpolated with natural smoothing spline, and the D_C was then extracted from the smoothed D that yields maximum slope magnitude. Furthermore, in the vicinity of D_C, the fluctuation in $m^{S'}$ was found extreme like critical fluctuation in phase transition phenomena topic [7]. Therefore, we also managed to retrieve D_C via the standard variation of the $m^{S'}$, as shown in figure 2(b), where the peak was used to locate D_C. In addition, figure 2 shows that D_C shifts to lower D with increasing c. This is as the larger c the more chances the I agents can distribute their disease. Consequently, D does not need to be very high to cause pandemic (no survivor left). Note that in figure 2(a), $m^{S'}$ at $D = 1$ mcs for various c are different as the results were normalized with N. Then, for having one S agent out, the low c system was affected the most. This one S agent missing effect perishes at higher c. Further, at very high c, $D = 1$ mcs may lie in the low survivor phase, causing $m^{S'}$ to decrease with increasing c. As figure 2 tells how survivor (the indirect 'herd immunity') and its standard deviation depend on c and D parameters, it is of interest to perform quantitative analysis on the D_C. The non-linear power law regression was performed for fitting D_C and c, which gives $D_C = 0.1196c^{-1.049}$ ($R^2 = 0.9994$) and $D_C = 0.0964c^{-1.056}$ ($R^2 = 0.9982$) for $m^{S'}$ and its standard deviation results, respectively. As seen, the fitted parameters are close, suggesting the same D_C characteristic.

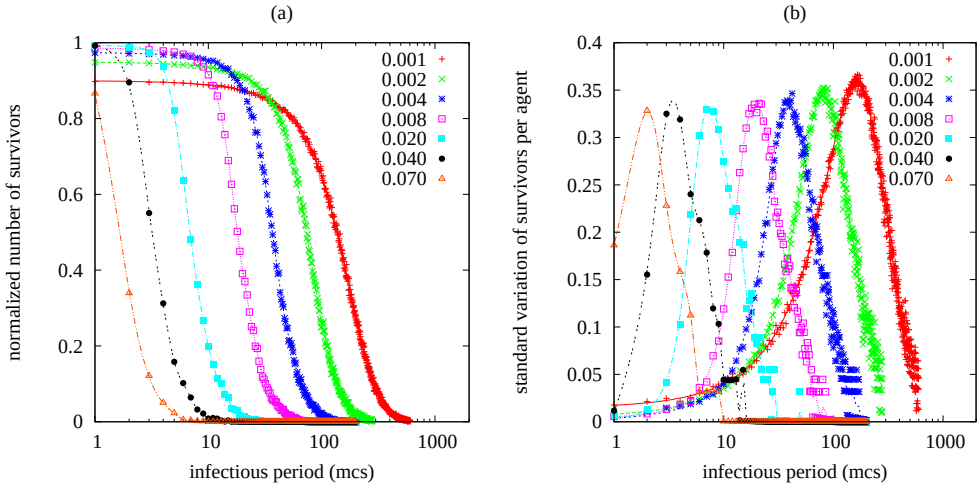

Figure 2. (a) $m^{S'}$ and (b) its standard variation as a function of infectious period D. The results were simulated at $N = 100^2$, $D = 100$ mcs, and $c = [0.001, 0.070]$. The curves were used for visual aids.

4. Conclusions

In this work, the disease spreading was investigated using SEIR model, the discrete spin Hamiltonian and the Monte Carlo simulation. The time dependent behaviour of the subgroup magnetization was investigated and used to define low and high survivor phases. The critical infectious period used to separate these phases was found to depend the population density, where the power law relationship was suggested. From the power law function, it reveals the inherited 'herd immunity' of the system, which one can use to issue policies in overcoming the pandemic for a disease with particular infectious period, such as how to control the population density to discontinue the disease spreading.

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