Modelling Infectious Disease Spreading Dynamic via Magnetic Spin Distribution: The Stochastic Monte Carlo and Neural Network Analysis

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Abstract. In this work, the disease spreading under SIR framework (susceptible-infected-recovered) agent-based model was investigated via magnetic spin model, stochastic Monte Carlo simulation, and Neural Network analysis. The defined systems were two-dimensional lattice-like, where the spins (representing susceptible, infected, and recovered agents) were allocated on lattice cells. The lattice size, spin density, and infectious period were varied to observe its influence on disease spreading period. In the simulation, each spin was randomly allocated on the lattice and interacted with its first neighbouring spins for disease spreading. The subgroup magnetization profiles were recorded. From the results, numbers of agents in each subgroup as a function of time was found to depend on all considered parameters. Specifically, the disease spreading period slightly increases with increasing system size, decreases with increasing spin density, and exponentially decays with increasing infectious period. Due to many degrees of freedom associated, Neural Network was used to establish complex relationship among parameters. Multi-layer perceptron was considered, where optimized network architecture of 3-19-15-1 was found. Good agreement between predicted and actual outputs was evident. This confirms the validity of using Neural Network as supplements in modelling SIR disease spreading and provides profound database for future deployment.

1. Introduction

Being diseased is an irregular condition that causes disordering or malfunctioning of a living organism. Disease usually causes pain, stress, suffering, or even death. There are many types of the disease, but the one that mostly affects the community in terms of spreading is the infectious disease, which resulted 9.2 million deaths in 2013 (about 17% of all deaths) [1]. This makes the modelling of the infectious disease spreading very important for predicting, controlling, and managing health policies. Modern disease spreading modeling can be categorized as deterministic compartment and stochastic agent-based models. The deterministic is to assign rate of state changing among subgroups to calculate number of agents in each subgroups over time. It is applicable for large populations (e.g. national scale), but usually fail in 'small world' group as fluctuation is ignored. However, the stochastic agent-based investigates each individual to find the course of disease spreading. It is then appropriate for small communities such as schools, hospitals, solider camps, etc.[2]. Although having

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benefits in 'small world', the agent-based is comparatively less investigated due to its complication with degrees of freedom associated. Therefore, this work tried to alleviate the computation difficulties through discrete lattice analysis and disease spreading update via Monte Carlo simulation. In addition, with extensive simulation datasets, the Neural Network was used to ease complex in modelling the 'Big Data' by establishing the relationship among parameters via the knowledge based analysis.

2. Theories and methodologies

2.1. The Spin Hamiltonian and Monte Carlo simulation

Modelling of disease spreading generally categorizes individual agent in subgroups in specifying stage of epidemic. For an influenza type disease, the subgroups are the susceptible (S; not yet infected), the infected (I; sick and infectious), and the recovered (R; immune). With discrete similarities between subgroup and spin model, the spin Hamiltonian $\mathcal{H}_{ij} = -J_{ij}\sigma_i\sigma_j$ was considered and simulated using Monte Carlo technique [3]. The spin σ_i at site i^{th} refers to the agent being in susceptible state (+1;S), infected/infectious state (−1;I), or recovered state (0;R). J_{ij} = 1 was used as unit of disease transmission strength, where the transmission range covered first neighbouring distance. Further, to comply with disease spreading in epidemiology, the spin is only allowed to change from +1(S) to −1(I) upon disease adopting, and from −1(I) to 0(R) when being in the I state up to infectious period.

In Monte Carlo simulation, the system size ($N = L^2$), the spin concentration (c), and infectious period (*Infp*) were varied from 50 to 100, 0.01 to 0.10, and 10 to 200 Monte Carlo step per spin (mcs), respectively. One mcs is the unit of simulation time, being equal to random allocation of N spins. For each condition, all $n = cN$ spins (agents), were randomly allocated on the lattice cells. One spin was assigned in the I state and the other were in the S state. After that, all spins changed state from S $\rightarrow$ I and I $\rightarrow$ R using Hamiltonian minimization and criteria specified above. Time to perform this in one round was assigned as 1 mcs. These procedures repeated until simulation ends. Each simulation was carried out up to 2000 mcs and for each condition, and 1000 independent runs were performed to average out random noises. For the observables, the sub-group magnetization i.e.

$$m^{\mathrm{I}} = \frac{1}{n}\sum_{i=1}^{n}\delta_{\sigma_i,-1}\ ,\ m^{\mathrm{S}} = \frac{1}{n}\sum_{i=1}^{n}\delta_{\sigma_i,1}\ ,\ m^{\mathrm{R}} = \frac{1}{n}\sum_{i=1}^{n}\delta_{\sigma_i,0}\ ,\text{where } m^{\mathrm{S}} + m^{\mathrm{I}} + m^{\mathrm{R}} = 1, \quad (2)$$

were considered. Then, the disease spreading period was defined from the least time that $m^{\mathrm{I}} < 1/n$.

2.2. Neural Network

Neural Network (NN) is a programming technique with ability in modeling relationship between given inputs and related outputs from examples [4]. The NN is made up of neurons or nodes connected. Each node locates in input, hidden, or output layers. The NN then learns relations by tuning weights during the training. In the training, input-output pairs are passed through the model and the weights adjust to minimize the error between the network and the desired outputs. Once the error is minimal, the network is trained and ready to predict outputs for unseen inputs. In this study, the Back Propagation (BP) algorithm was considered. The BP initially performs a 'forward pass', which the output of each node is calculated from weighted sum of inputs (S_j), i.e. $S_j = \sum_i a_j w_{ij}$, where a_i is the activation level, and w_{ij} is the weight from node i to node j. Then, the logistic transfer function, i.e. $g\left(S_j\right) = 1/\left(1+e^{-S_j}\right)$, were applied and $g(S_j)$ becomes the output of node j. The same procedure repeats for all nodes. After that, the BP performs a 'backward pass' where the errors δ_j were calculated to update the weight of each node. These processes repeat for new inputs until the error is minimized.

3. Results and discussions

From the simulation results, typical time dependence of m^{S}, m^{I} and m^{R} were found as shown in Fig. 1(a) [2]. As is seen, m^{S} drops as S changes to I until there is no S left. With continue decreasing S, m^{I}

agents reach maximum, start declining, and result in a peak-like function. Finally, all I agents get recovered (I→R), so only R agents exist. Then, with varying spin/agent densities (c) in Fig. 1(b), the system/lattice sizes N Fig. 1(c), and the infectious period ($Infp$) in Fig. 1(d), the m^I peaks change in theirs characteristic. Specifically, in Fig. 1(b), the denser of the population induces more infectious contact resulting more I agents at the beginning. Hence, the time for infection to completely cease (disease spreading period) does not very much extend over the infectious period of 100 mcs. On the other hand, in Fig. 1(c), the larger N gives more spaces for the agents. Therefore, even with the same density c = 0.01, the smaller N gives more chances for having infectious contact so disease spreading period declines with decreasing N. However, as is seen, this N influence is somewhat insubstantial. Note that there is a sharp drop close to the peak for small N's which is due to the recovery of the first I agent. Therefore, in this normalized fashion, 1 agent missing from small N's (and hence n's) is somewhat prominent. Finally, in Fig. 1(d), the longer infectious period induces the larger disease transmission period as expected [5]. However, the relationship between these two periods is not trivial in formulating, especially when also taking other N and c parameters into account.

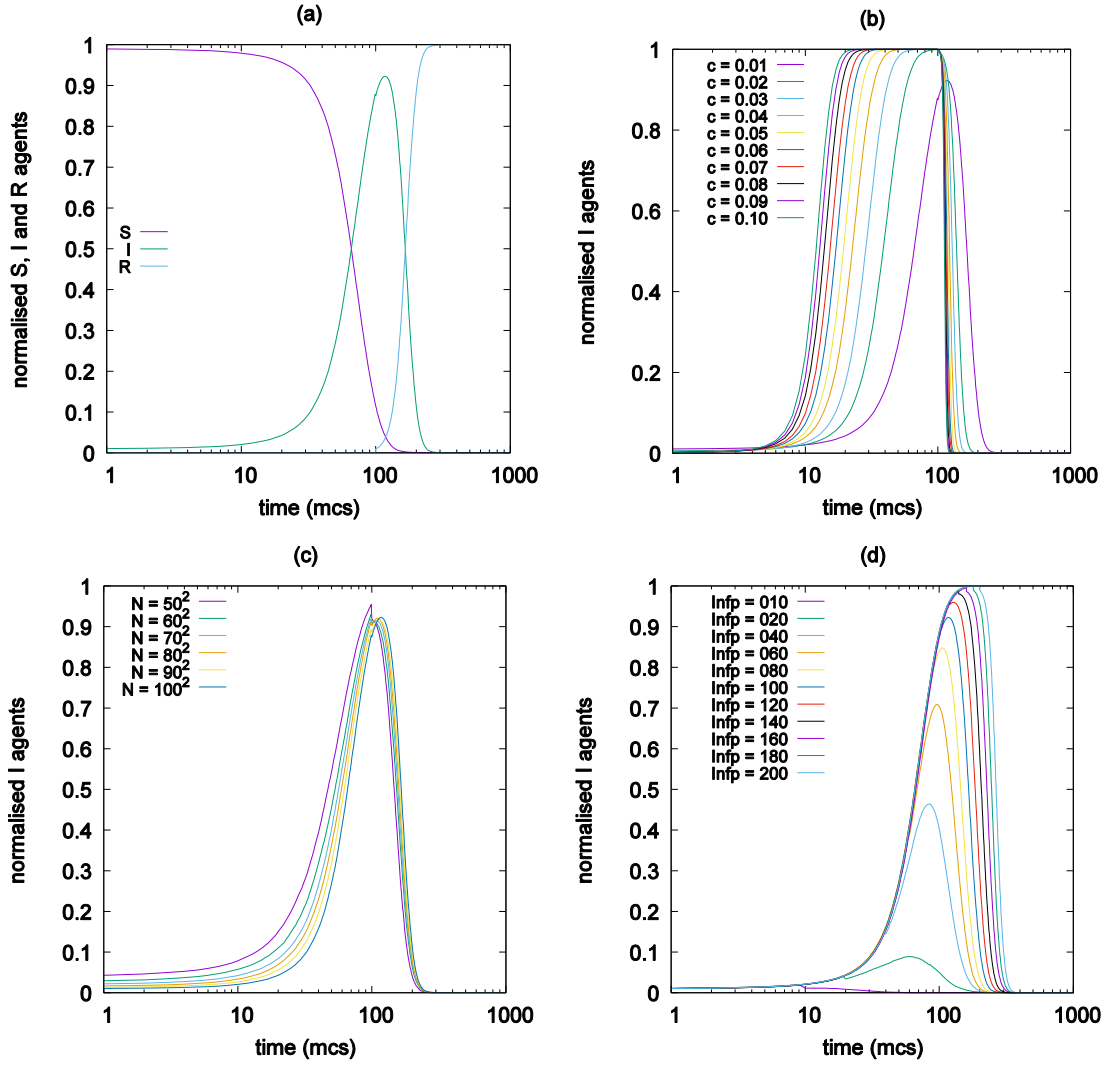

Figure 1. (a) The typical normalized SIR curves for $N = 10^2$, c = 0.01, and infectious period of 100 mcs. On the other hand, (b) to (c) show the normalized I with varying c, N and infectious period (Infp). If not varied, the parameters used were c = 0.01, $N = 100^2$, and $Infp$ = 100 mcs.

Therefore, all disease spreading period data were supplied to the Neural Network (NN) modelling. The spin density, the lattice size, and the infectious period were used as input parameters, whereas the

disease transmission period was used as the output parameter. Two hidden layers with up to 40 nodes in each layer were used in extensive search for extracting the optimized network architecture. The optimized one was found at 3-19-15-1, where these numbers are for number of nodes in input, first hidden, second hidden and output layer respectively. The good agreement from the predicted and actual real date was found when performing scattering plot (not shown). The linear trend was suggested with very good R-square ≈ 0.9937. Then, the NN weights were used to predict the disease spreading data. Examples can be shown in Fig. 2 which tells how the spreading period depends on *N*, *n* and *Infp* parameters. Specifically, the disease spreading period was found to slightly increases with increasing *N*, decrease with increasing *c*, and exponentially decay with increasing *Infp* (except at low *Infp* and low *c* due to very few infectious contacts). These results, where applicable, qualitatively correspond with previous using different technique, i.e. the deterministic [6].

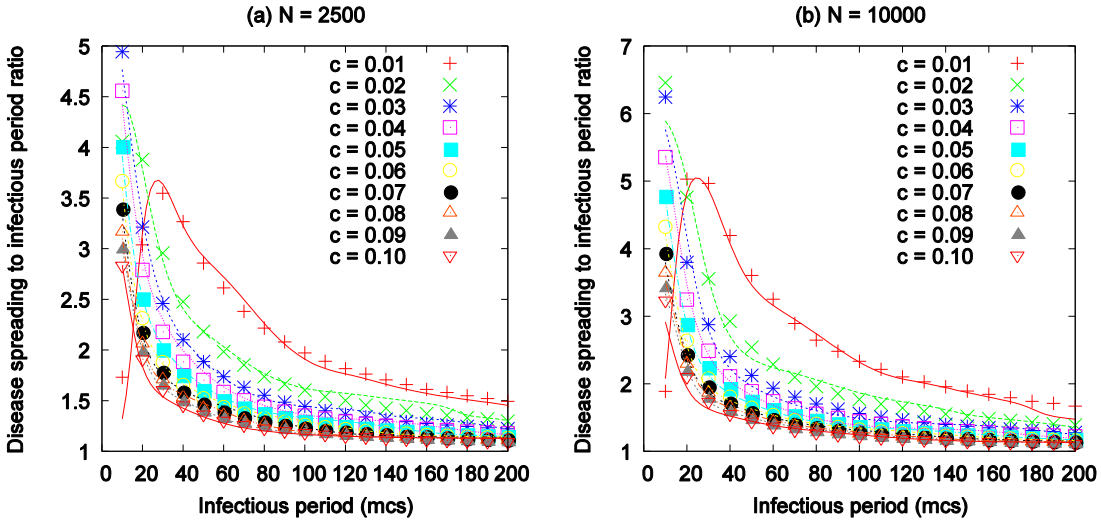

Figure 2. The disease spreading to infectious period ratio for (a) $N = 50^2$ and (b) $N = 100^2$. The discrete data points were from simulation and the lines were from NN interpolation.

4. Conclusions

In this work, the disease spreading was investigated using SIR model, the modified spin Hamiltonian, the Monte Carlo simulation, and the Neural Network (NN) analysis. The time dependent behaviour of the subgroup magnetization was revealed and used to extract the disease spreading period. The period varies with changing system size, spin density and infectious period as expected. The NN was then used to establish complex relationship among parameters with good accuracy. This then provides a sophisticated tool for multi-dimensional modeling of SIR problem, and provides another fruitful step in modeling infectious disease spreading in the nowadays Big Data era.

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