An Ising-based Model for the Spread of Infection

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Abstract—A zero-field ferromagnetic Ising model is utilized to simulate the propagation of infection in a population that assumes a square lattice structure. The rate of infection increases with temperature. The disease spreads faster among individuals with low J values. Such effect, however, diminishes at higher temperatures.

Keywords—Epidemiology, Ising model, lattice models

I. INTRODUCTION

ONE of the simplest models in epidemiology separates the population into two discrete states: S for susceptible and I for infected or infective [1]. In the SI model, the time rate of change in number of susceptible and infected individuals varies is given by the following equations:

$$\frac{dS}{dt} = -\beta SI \tag{1}$$

and

$$\frac{dI}{dt} = \beta SI . (2)$$

where β is the infection rate. To consider a closed system, we consider a constant population size. Thus at any given time, the sum of susceptibles and infectives is equal to some constant, say N. That is, S(t) + I(t) = N. In which case, the above equations yield the following solutions:

$$S(t) = \frac{N}{1 + e^{\beta(t - t_c)}} \tag{3}$$

and

$$I(t) = \frac{N}{1 + e^{-\beta(t - t_c)}}. (4)$$

Eq. 3 and 4 are logistic or S-curves. The point of inflection occurs at the critical time t_c .

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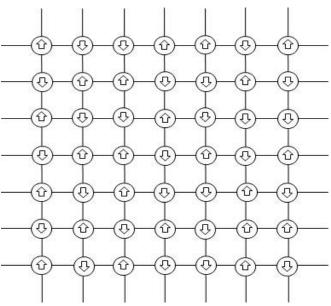


Fig. 1 A square lattice containing suceptible *spin down* and infected *spin down* individuals

In this work, we adapt the Ising model [2] framework to study dynamics of disease spread. Similar to [3], we confine our investigation to a square lattice and impose periodic boundary conditions. We observe the infection rate, β , varies with parameters such as temperature T and neighbor interactions.

II. THE MODEL

Consider a closed community described by the square lattice in Fig. 1. Each site is occupied by one individual which can either be susceptible (spin down, $\sigma = -1$) or infective (spin up, $\sigma = +1$). The edges of the lattice are connected, thus forming a torus or a donut. Associate with each lattice configuration is a Hamiltonian which takes the form

$$H = -J \sum_{i,j} \sum_{x,y} \sigma_{ij} \sigma_{xy} , \qquad (5)$$

Where $(x,y) \in \{(i+1,j), (i-1,j), (i,j+1), (i,j-1)\}$ describes the Von Neumann neighborhood and J is the interaction parameter whiich described the (coupling) strength between spins.

Initially, the lattice is filled with susceptibles (all spin down). A random site is then selected and the corresponding spin flipped (from down to up). This is the first infective. For succeeding iterations, a randomly chosen site changes state if the change in energy, $\Delta H = H_{new}$ - $H_{previous}$, is less than or equal to zero. If $\Delta H \geq 0$, the flip is accepted according to the probability

$$p = e^{-\Delta H/T}, (6)$$

where T is the scaled temperature. Flipping is allowed only in one direction (up \rightarrow down, $S \rightarrow I$). Whenever a site is infected, it remains that way until the end of the simulation. Data presented in the next section correspond to the mean of 10 independent trials for a population size N=100 (10 \times 10 lattice).

III. RESULTS AND DISCUSSION

Simulation results reveal a logistic type of behavior, which is characteristic of the standard SI model. Fig. 2 shows the propagation of infection when T=2.20 and J=1.00. At the onset, the spread of the disease approximates an exponential growth. But as time progresses, the growth slows down and saturates at the value of N. Since there is no recovery (flipping is one way), the entire community eventually becomes infected. The point of inflection, t_c , corresponds to the time when 50% of the population is already affected by the disease.

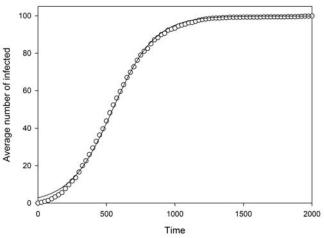


Fig. 2 Infection curve associated with T=2.2 and J=1.0

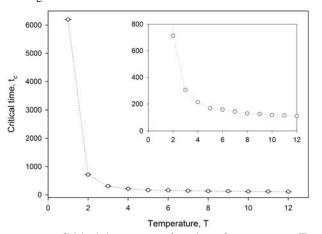


Fig. 3 Critical time, t_c , as a function of temperature, T

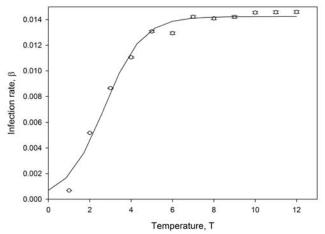


Fig. 4 Temperature dependence of the infection rate

Estimates of the critical time, t_c , and infection rate, β , are obtained by using Eqn. 4 as a fitting function. Fig. 3 reveal a sudden drop in t_c associated with a slight rise in temperature (from 6000 to <1000 when T was changed from 1.0 to 2.0). But as T is increased further, the variation in critical times becomes minimal. The point of inflection occurs at an earlier time at larger T values. Thus, the disease spreads more rapidly at higher temperatures. Calculated values of the rates, β , are plotted in Fig. 4. As T increases, β approaches a constant value. In our Ising-based SI model, the concept of temperature may be related to the level of aggression of a particular virus or associated to external parameters like ambient temperature and humidity. The concept of temperature may also be perceived with a broader scope to include the effects of cultural and socio-economic risk factors.

Next, we present the effect of varying interaction parameter, J, on the infection curves. In Fig. 5(a), saturation (100% infective) is achieved fastest when J=0.25. The rate of spread of infection decreases with increasing J. This parameter may be interpreted as the inverse of the contact time. Lower J values can mean prolonged exposure to the agent or contagion. The effect of the parameter J on the spread of the disease, however, vanishes at higher temperatures. Fig. 5(b) shows overlapping plots, independent of the J value.

IV. SUMMARY AND CONCLUSION

The dynamics of the spread of infection on a two-dimensional square lattice with periodic boundary conditions was analyzed using an Ising-based SI model. Beginning from a single infective, the disease was able to propagate faster at higher temperatures. Increasing the value of the interaction parameter, J, slowed down the infection spread. These effects, however, became less evident as the temperature is increased further.

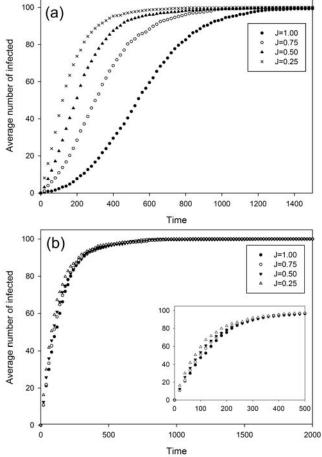


Fig. 5 Effect of varying J: (a) T=2.2 and (b) T=10.0

REFERENCES

- [1] HW Hethcote, "Three basic epidemiological models", In: Levin, Hallan, Gross (Eds.), *Applied mathematical ecology. Biomathematics*, vol. 18, New York: Springer-Verlag, 1989, pp. 119—144.
- [2] BA Cipra, "An introduction to the Ising model", The American Mathematical Monthly, vol. 94, no. 10, pp. 937--959, 1987.
- [3] LF Lopez and E Massad, "Time-dependent discrete, Ising-like model for SIS epidemic systems", In Proceedings of European Conference on Mathematical and Theoretical Biology, 2011.