

THE SUSCEPTIBLE-INFECTIOUS MODEL OF DISEASE EXPANSION ANALYZED UNDER THE SCOPE OF CONNECTIVITY AND NEIGHBOR RULES

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ABSTRACT

This paper presents a model to approach the dynamics of infectious diseases expansion. Our model aims to establish a link between traditional simulation of the Susceptible-Infectious (SI) model of disease expansion based on ordinary differential equations (ODE), and a very simple approach based on both connectivity between people and elementary binary rules that define the result of these contacts. The SI deterministic compartmental model has been analysed and successfully modelled by our method, in the case of 4-connected neighbourhood.

KEYWORDS

Infectious disease expansion, deterministic compartmental models, ODE, Neighbour binary rules, Connectivity.

1. INTRODUCTION

The outbreaks of infectious disease pandemics have shaped nations and civilizations through the ages. History tells us the terrible impact of biblical plagues in Ancient Egypt, bubonic plague in Europe in the Middle Age, influenza at the beginning of the twentieth century or AIDS the more recently emerging pandemics [1]. Mathematical modeling is playing a very important role to assess and control the potential outbreaks [2]. The first paper presenting a model for an infectious disease appears in 1760. The author, Bernouilli, a swiss mathematician and physicist, dealt with a statistical problem involving censored data in order to analyze smallpox morbidity and mortality that aimed to demonstrate the efficacy of vaccination [3]. At the beginning of the twentieth century two pioneering works can be mentioned. W.H. Hamer [4] published a discrete time epidemic model for the transmission of measles in 1906. The model assumes that the number of cases per unit time (incidence) depends on the product of densities of the susceptibles and infectives. In 1911 R. Ross [5] demonstrated that malaria is produced by the bite of a mosquito.

His mathematical model of expansion is based on a set of equations to approximate the discrete-time dynamics of malaria and asserts it is possible to control the disease whenever the population of mosquitos is reduced below a threshold. This was a new and crucial idea. Between 1927 and 1939 Kermack and McKendrick [6, 7] published papers on epidemic models and obtained the epidemic threshold that the density of susceptibles must exceed for an epidemic outbreak to occur. This model includes three states, the S (susceptible), I (Infectious) and R (Recovered) instead of the two, S and I, of the Bernoulli's model. From the mid-twentieth century a great variety of epidemiological models have been developed after the recognition of the importance of modeling in public health decision making [8]. In the nineties, when the scientists began to pay attention to complex systems new paradigms spread out in order to better understand and model the impact of numerous variables that go beyond the micro host-pathogen level, such as ecological, social, economic, and demographic factors. Many scientists coming from such different fields as medicine, molecular biology, computer science and applied mathematics or economy have teamed up for rapid assessment of urgent situations of contagious diseases by means of a multidisciplinary approach. The case of HIV/AIDS pandemic [9-12] is a good example.

This paper presents a model to approach the dynamics of infectious diseases expansion by means of a set of neighbour rules between elements located in a lattice that represents the whole population. Following the introduction, Section 2 provides a brief summary of the deterministic compartmental models and highlights the Susceptible-Infectious (SI) model which has traditionally been solved by ODE. Section 3 presents our model which considers the population confined in a lattice. The contacts between people are performed by neighbour binary rules, that are tailored to model different situations such as Susceptible, Infected, with or without capability to infect further. The neighborhood is also defined depending of connectivity. We consider 4-connection, 8-connection and horse jumping chess connection. The results are compared with those of the simulation of ODE. Section 4 presents a discussion upon the suitability of the model and proposes futures research. Section 5 summarises the work and presents concluding remarks.

2. MATHEMATICAL MODELLING

Three are the main categories encompassing mathematical modeling [1]. The statistical methods deal with real epidemics. They identify their spatial patterns and allow surveillance of outbreaks. The empirical models are based on machine learning methods such as data mining that allow the forecasting of the evolution of an ongoing epidemic spread. The mathematical or state-space methods provide quantitative predictions that have to be validated to forecast the evolution of a hypothetical or real epidemic spread. These methods also redefine our understanding of underlying mechanisms.

2.1. The Deterministic Compartmental Models

The description used in epidemiologic compartmental models is composed of standard categories represented by the variables that model the main characteristics of the system. These compartments, in the simplest case, divide the population into two health states: susceptible to the infection (denoted by S) and infected (denoted by I) [13]. The way that these compartments interact is often based upon phenomenological assumptions, and the model is built up from there. Usually these models are depicted by ODE, which are deterministic, but can also be viewed in more realistic stochastic framework [14]. To achieve more realism, other compartments are often included, namely the recovered (or removed or immune) compartment labelled by R, or the

exposed compartment, labelled by E. The stratification of these compartments lead to well-known models such as SIS [15], SIR [16], SEIR [17, 18],...or more complex ones [19]. The number of variables to be incorporated to the model depends on the particular disease being studied as well as on the desired complexity of the model. Other variables incorporated into the equation represent fundamental quantities such as birth rate, rate of transmission of infectious agent, death rate, and so forth, and are constants that can be changed.

2.2. The Traditional Susceptible-Infectious (SI) Model

In the SI model the two groups are the susceptible hosts, S, that are not infected by the pathogen but can get infected, and the infected hosts, I, who are infected by the pathogen. Assuming the mass-action model, the rate at which susceptible hosts become infected is a product of the number of contacts each host has per unit time, r , the probability of transmission of infection per contact, β , and the proportion of the host population that is infectious, I/N , where $N = S + I$ is the total population size. This model is suitable to represent the case of the human immune deficiency virus (HIV) where there is no recovery. A schematic of the model is shown in Figure 1.



Figure 1. SI Model

Equations (1) for the SI model are as follows:

$$\begin{aligned} \frac{dS}{dt} &= -\beta r S \frac{I}{N} \\ \frac{dI}{dt} &= \beta r S \frac{I}{N} \end{aligned} \quad (1)$$

Since the population size is fixed, we can reduce the system to one dimension with the substitution $S = N - I$ to provide the logistic Equation (2).

$$\frac{dI}{dt} = \beta r (N - I) \frac{I}{N} \quad (2)$$

We can analytically solve Equation (2) with the initial condition $I(0) = I_0$, so

$$I(t) = \frac{I_0 N}{(N - I_0)e^{-\beta r t} + I_0} \quad (3)$$

The simulation of the SI model is shown in Figure 2 with the initial value $I(0) = I_0 = 1$

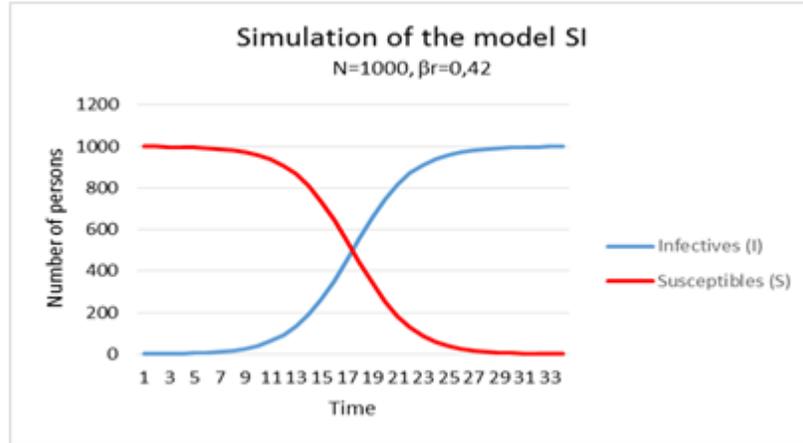


Figure 2. Simulation of the traditional SI Model

3. OUR PROPOSAL

Our proposal is based on a set of elementary binary rules that have the capability to model interactions between two individuals [20-23]. Without loss of generality we consider a two-dimensional square lattice, every cell represents a susceptible person except the one at the center which locates an infected one. When the infected person contacts with his/her neighbors he/she spreads the disease. The new infected people have then the capability to infect other people.

3.1. Binary neighbor rules

Equation (4) defines a generic binary neighbor rule denoted \otimes .

$$\begin{aligned} \otimes : (0,1) &\rightarrow (0,1) \\ (x, y) &\rightarrow z = x \otimes y \end{aligned} \quad (4)$$

The \otimes rule can be represented by a two input table that defines concretely the operation, as shown in Figure 3.

\otimes	0	1
0	a_3	a_1
1	a_2	a_0

Figure 3. Generic neighbor rule represented by a table

Let m stand for the number of the rule. The number is represented by the four bits stored in the cells; $m = a_3 a_2 a_1 a_0, \in [0, 2^4-1]$; $a_i \in (0, 1)$; $i \in [0, 3]$. As an example, we consider $m = 7$, that is to say $a_3 = 0$; $a_2 = 1$; $a_1 = 1$ and $a_0 = 1$.

\otimes	0	1
0	0	1
1	1	1

Figure 4. The rule n° 7 represented by a table ($a_3 = 0$; $a_2 = 1$; $a_1 = 1$; $a_0 = 1$)

The table defines concretely the operation as follows:

$0 \otimes 0 = 0$; $0 \otimes 1 = 1$; $1 \otimes 0 = 1$ and $1 \otimes 1 = 1$

This operation is suitable to model the interaction between infected people and susceptible people by identifying “0” as susceptible and “1” as infected. The previous operation means that infected people can transmit the disease to susceptible people ($1 \otimes 0 = 1$), when infected people contact other infected people, all them remain infected ($1 \otimes 1 = 1$), susceptible people have no effects upon people ($0 \otimes 0 = 0$ and $0 \otimes 1 = 1$).

3.2. Neighborhood rules modelling the spreading of a disease

In the following two-dimensional square lattices we present the spreading of a disease by a unique Infected (“1”) located at the center of the lattice. All the empty cells are considered to be Susceptible (“0”). The red numbers stand for the generation number (time unit) the spreading occurs. Figure 5 shows the case of a 5x5 lattice with a contagion rate $\rho = 4$ per generation (4-connected cells are neighbors to every cell that touches one of their edges, following the Von Neumann neighbourhood).

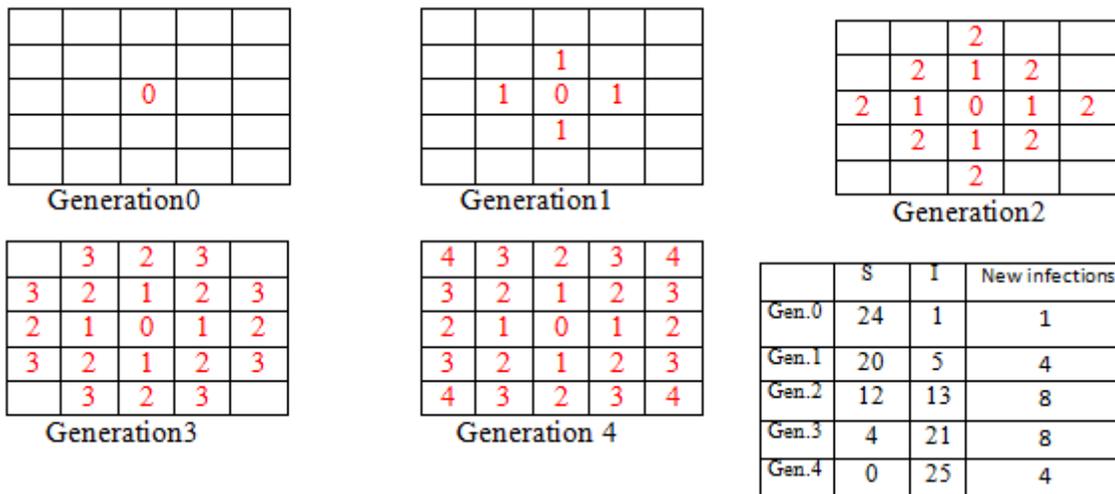


Figure 5. The spreading of a disease in a 5x5 lattice with $\rho = 4$ (4-connected cells)

The spreading of the disease results in a diamond-shaped region shown for rate = 4 in Figure 5. The evolution of the infected people can be carried out by means of the equation $1+2\rho(\rho+1)$, where ρ stands for the rate.

The same example is presented for 8-connected cells (with horizontal, vertical, and diagonal connection) following the Moore neighborhood, and for the jumping chess neighbourhood. See Figures 6 and 7 respectively.

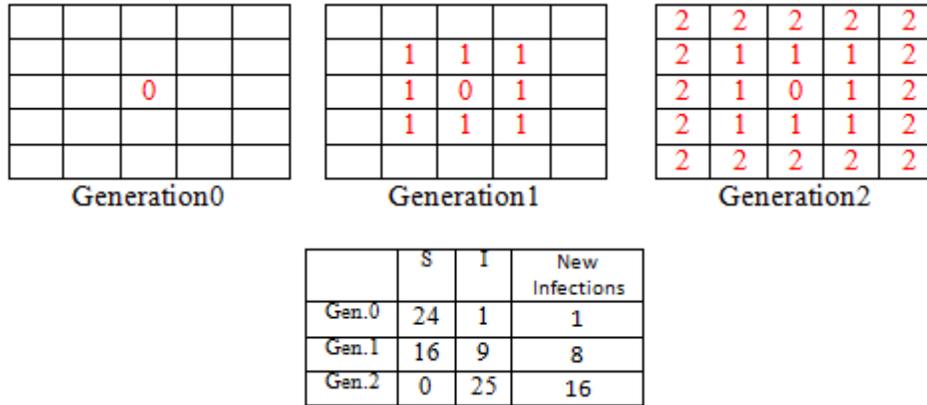


Figure 6. The spreading of a disease in a 5x5 lattice with $\rho = 8$ (8-connected cells)

For the 8-connected neighborhood, the evolution of the infected people can be carried out by means of the equation $(2\rho+1)^2$.

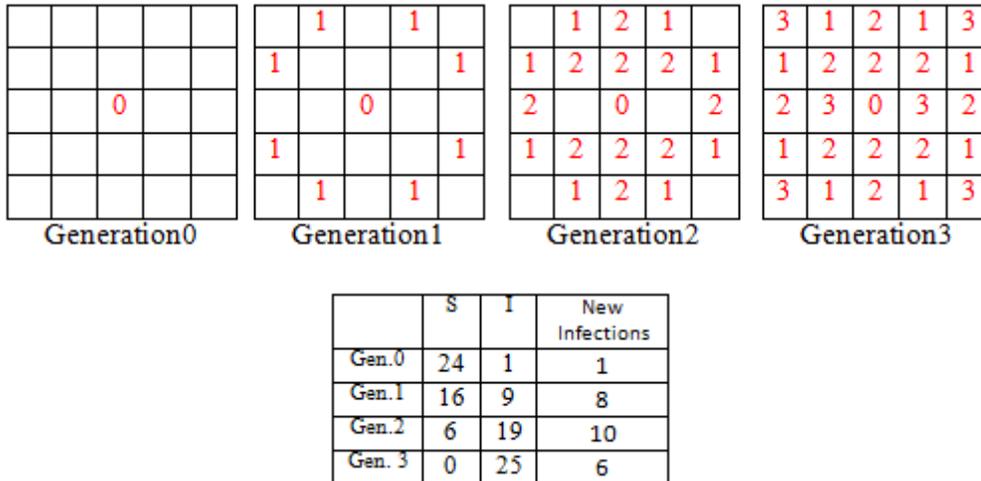


Figure 7. The spreading of a disease in a 5x5 lattice with $\rho = 8$ (horse jumping chess connected cells)

3.3. Comparison Between the Traditional SI Model and the 4-Connected Neighborhood Model

The following graph represents the 4-connected case in a 32x32 lattice, equivalent to $N=1024$ (in order to approximate the graphic shown in Figure 2 where $N=1000$).

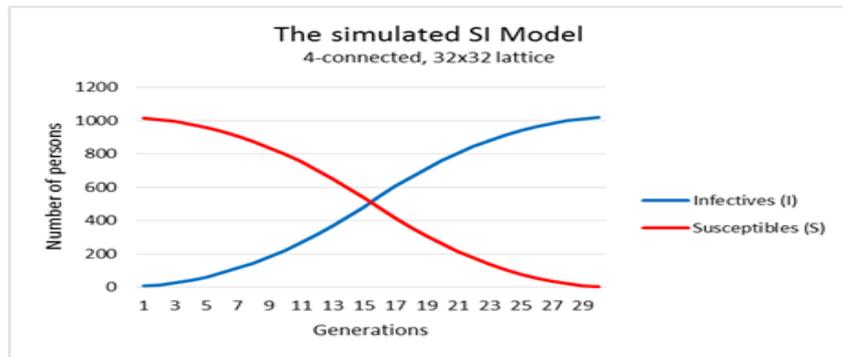


Figure 8. The spreading of a disease in a $32 \times 32 = 1024$ lattice with $\rho = 4$ (4 -connected cells).

The comparison between Figures 2 and 8 suggests that the 4-connected cells (Von Neumann neighbourhood) could be a suitable approximation to approach the traditional SI model. In order to better compare the models, we now compare the previous simulation based on Equation 2 with our 4-connected neighborhood model for similar populations, lattices 10×10 , equivalent to $N=100$ and 100×100 , equivalent to $N=10000$, See Figures 9 and 10.

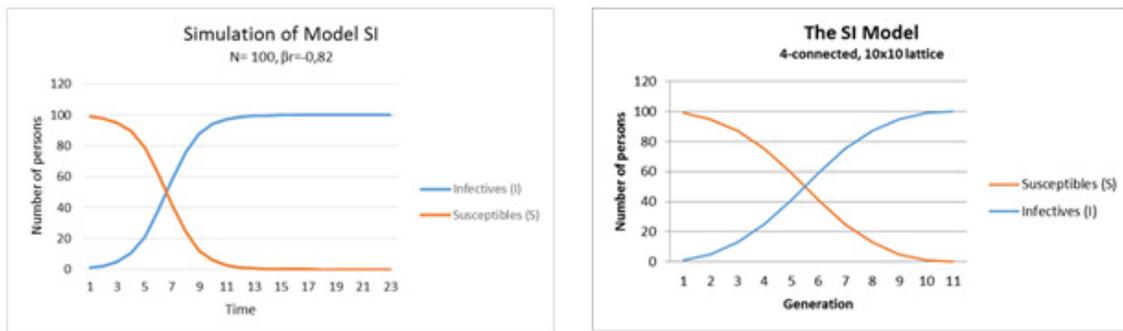


Figure 9. The spreading of a disease in a 10×10 lattice with a $\rho = 4$ (4 -connected cells), compared to the simulation of the traditional model ($N=100$, $\beta r = 0,82$).

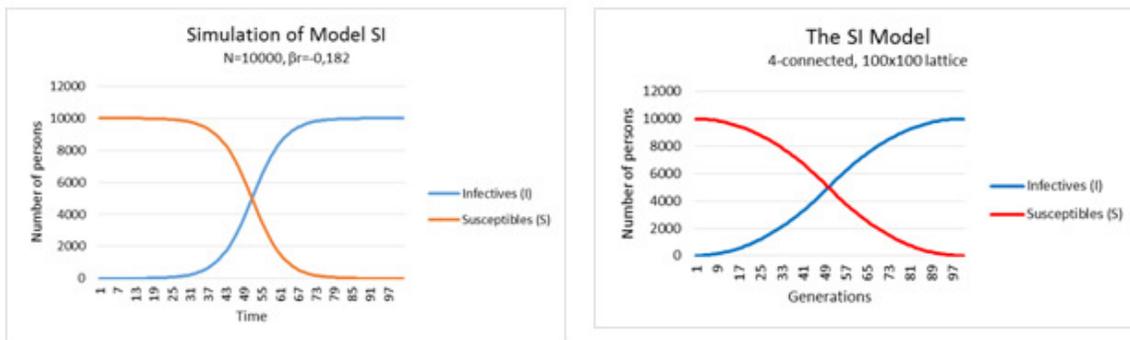


Figure 10. The spreading of a disease in a 100×100 lattice with $\rho = 4$ (4 -connected cells), compared to the simulation of the traditional model ($N=10000$, $\beta r = 0,182$).

Empirically, the rate βr of the traditional simulation of Model SI has been modified when N varies in order to impose the crossing point between (I) and (S) occurs at the same “time” (obviously we have assumed that “Time” in the traditional model is equivalent to “Generation” in ours, as explained in Section 4.).

4. DISCUSSION

Our model establishes a link between the traditional ODE simulation of the SI deterministic compartmental model of disease expansion and a very simple model based on both the connectivity between people and different rules that define the results of the contacts. The parameters of each model must harmonize. Our lattice size stands for N , the number of persons ($N = S + I$). In the ODE model, the number of contacts each host has per unit time, r , and the probability of transmission of infection per contact, β , have been englobed in a unique variable, βr , which stands for the number of actual infections that occur in a unit time, which is equivalent to the connectivity, ρ , in our model. Obviously, the time scale is different in each model, so, unit time may be days, hours, etc... In our model, “Generation” is a dimensionless unit, which only means how the sequence of infections occur. In order to allow meaningful comparisons between the different approaches we have harmonized the parameters of the models as follows, $N \equiv$ size of the lattice and $\beta r \equiv \rho$. In this initial paper we have equalized the values of the studied population ($N=100$ or 10000 vs lattice size 10×10 or 100×100 , respectively) as well as the crossing point of the plotted values of S and I , which is 49, by tuning the value of βr (see Figures 9 and 10). This empirical approach reveals the capability of our model to meet the desired values. For a more refined modelling some attention should be paid to the slopes, by means of a balanced choice of (lattice size, rate, generation).

5. CONCLUSION

We have presented a new approach to the SI deterministic compartmental model. Our proposal is based on both connectivity between people and elementary binary rules that quantify the contacts between people. Our model fits the results carried out by traditional simulation of ODE. This encouraging empirical result must be improved in the future by means of a deeper analysis of the connectivity including a probabilistic approach of it. The SIR, SIS, SIRS models will be also studied. Finally, experimental data coming from statistics on real cases must be directly confronted in order to validate our model. Further we envisage to adapt this model to the field of virus expansion in computers.

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