A discrete approach of the Susceptible-Infectious-Susceptible (SIS) Model of Disease Expansion

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Abstract: - This paper presents a discrete model of the dynamics of infectious disease expansion and builds a link between two conceptually different approaches of the Susceptible-Infectious-Susceptible (SIS) model: the continuous one, depicted by traditional simulation of ordinary differential equations (ODE), and ours, based on both connectivity between individuals and a local binary rule. The connectivity fixes the possible contacts between people and the rule defines whether the contacts are infective or not. The population confines in a grid and contagion extends from the infected centre cell by applying the rule, following the connectivity pattern. Our model provides parameters to tune the rate at which susceptible hosts become infected and the rate at which infected hosts become susceptible. The model has been analyzed and successfully compared to the SIS deterministic compartmental model

Key-Words: - Infectious disease expansion, SIS, deterministic compartmental models, ODE, neighbour binary rules, connectivity.

1 Introduction

The modeling of infectious diseases is a tool used to study the mechanisms by which diseases spread, in order to predict the future course of an outbreak and to evaluate strategies to control an epidemic [1]. In 1662, J. Graunt was the first scientist who tried to quantify causes of death by studying listings of numbers and causes of deaths published weekly. In 1766, D. Bernouilli created a mathematical model to defend the practice of inoculating against smallpox [2]. The calculations from this model showed that universal inoculation against smallpox would increase the life expectancy for more than 3 years [3]. In 1911, Ross [4] demonstrated that malaria is produced by the bite of a mosquito. His mathematical model of expansion based on a set of equations approximated the discrete-time dynamics of malaria and asserted it is possible to control the disease whenever the population of mosquitos is reduced below a threshold. Between 1927 and 1939 Kermack and McKendrick [5, 6] published papers on epidemic models. Their approach was a simple deterministic compartmental model which 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 Bernouilli's model. The model was successful in predicting the behavior of outbreaks very similar to that observed in many recorded epidemics [7]. The SIS model can be easily derived from the SIR model by simply considering the individuals recover with no immunity to the disease that is, individuals are immediately susceptible once they have recovered. Our model aims to establish a link between traditional simulation of the Susceptible-Infectious-Susceptible (SIS) model based on ordinary differential equations (ODE), and a very simple approach based on both connectivity between people defined by means of their neighbourhood type, and a set of elementary local rules that define the result of these contacts. Following the introduction, Section 2 analyses the deterministic SIS model solved by ODE. Section 3 is devoted to present our model, which considers the population is confined in a square lattice. The neighbourhood is defined by means of the connectivity type. In this research we have considered 4-neighbours, 8-neighbours and knight connection. The contacts between neighbours are performed by local binary rules that are tailored to model different situations such as Susceptible or Infected, with or without capability to infect further. The results are compared with those of the simulation of ODE. Section 4 presents a discussion upon the suitability of the model and proposes future research. Section 5 summarizes the work and presents concluding remarks.

2 The Deterministic SIS Model

The deterministic SIS model derives easily from the SIR model [8]. It is depicted by a system of ODE shown in (1).

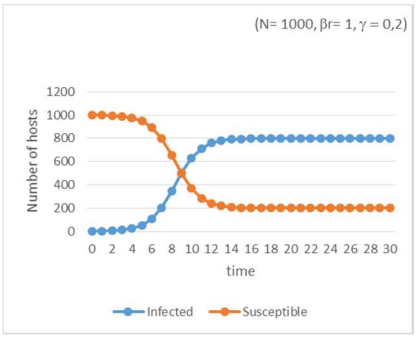
$$\frac{dS}{dt} = -\beta r S\frac{I}{N} + \gamma I \qquad (1)$$
$$\frac{dI}{dt} = \beta r S\frac{I}{N} - \gamma I$$

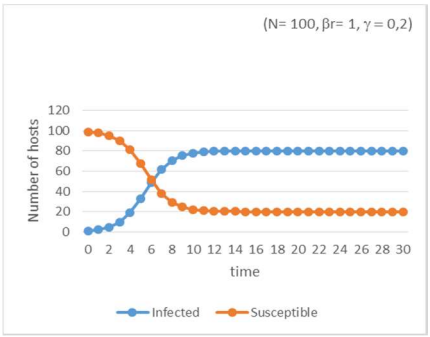
As for the the Kermack-McKendrick (or SIR model), the SIS model assumes that the population size, N, is fixed (i.e., no births, deaths due to disease, nor deaths by natural causes) and incubation period of the infectious agent is instantaneous. The population is divided here into two health states: susceptible to the infection (denoted by S), Infected (denoted by I). There is no recovered state (R) because the SIS model does not provide immunity, that is, individuals are immediately susceptible once they have recovered. The rate at which susceptible hosts become infected is a product of the number of contacts each host has per unit time, r, and the probability of transmission of infection per contact, β. The rate at which infected hosts become susceptible is γ. The total population size is N = S + I.

The analytical solution of the system is as follows. See Equations (2).

$$I(t) = \frac{\frac{N}{r\beta}(r\beta-\gamma)}{1+(\frac{N}{r\beta}\frac{(r\beta-\gamma)}{I_0}-1)\,e^{-(r\beta-\gamma)t}} \qquad (2)$$
$$S(t) = N - I(t)$$

Fig. 1. represents the simulation of the deterministic SIS model. In the equations the values are r=5; β=0,2; N=100; 1000 and S(0)=99; 999, respectively; I(0)=1, γ=0,2; 0,5 and 0,8. Horizontal axis stands for the time (generation number) and vertical axis stands for the number of individuals.





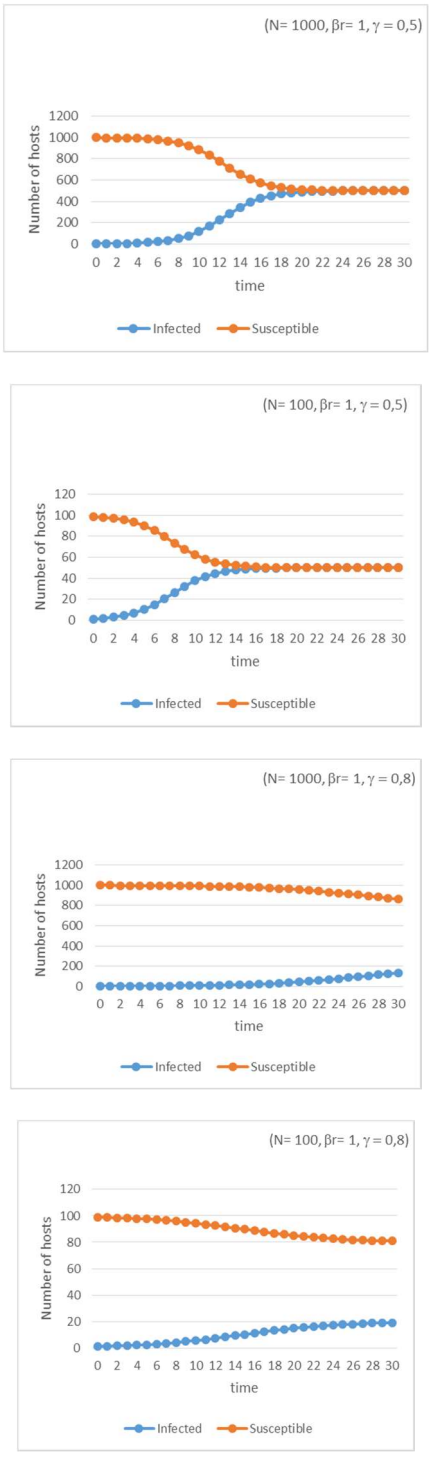


Fig.1. Simulation of the deterministic SIS model.

We draw empirically some conclusions;

- For $\gamma = 0,5$ both the number of Susceptible and Infected converge to the same steady state value N/2, when the equilibrium is reached.
- For $\gamma > 0,5$ although the number of Susceptible decreases it is always greater than the number of Infected that increases.
- For $\gamma < 0,5$ the number of Susceptible decreases and becomes smaller than the number of Infected that increases. That means the plotted graphics are crossed.
- The time to reach the steady state for I(t) and S(t) (horizontal assymptote) increases when γ increases, for the same value of N.
- The time to reach the steady state for I(t) and S(t) (horizontal assymptote) increases when N increases for the same value of γ.

3 Our Approach of the SIS Model

Our approach of the SIS model is based on both a concrete connection between individuals set by a particular neighbourhood pattern which fixes the possible contacts between individuals, and a local binary rule that defines whether the contact is infective or not. This rule is implemented by a binary operation to set the results of the contacts between 0 and 1, as shown by Equation 3.

$$R : \{0,1\} \times \{0,1\} \rightarrow \{0,1\}$$
$$(x,y) \rightarrow R(x,y) = a_i \quad (3)$$

where $a_i \in \{0, 1\}$ and $i \in [0, 3]$; $R(0, 0) = a_3$, $R(0, 1) = a_1$, $R(1, 0) = a_2$, $R(1, 1) = a_0$.

So we can define $2^4 = 16$ different local rules depending of the values of the sequence $a_3\ a_2\ a_1\ a_0$. Let m stand for the number of a particular rule, R_m. This number has binary representation, that is to say $m = a_3\ a_2\ a_1\ a_0$, $m \in [0, 2^4-1]$. As an example, if we consider m=6 we will have $a_3=0$; $a_2=1$; $a_1=1$ and $a_0=0$. In this research we have considered three types of connectivity defined by a neighbourhood relationship on a square nxn-sized grid. The Von Neuman neighbourhood is composed of a central cell and its four adjacent cells (4-neighbours, horizontal and vertical connection). The Moore neighbourhood is composed of a central cell and the eight cells surrounding it (8-neighbours, horizontal, vertical and diagonal connection). Finally, the knight neighbourhood follows the "L" pattern, i.e. moving two squares horizontally then one square vertically, or moving one square horizontally then two squares vertically. For the implementation of the SIS model we are interested in rule R_6 because we have $R_6(1, 0) = 1$; $R_6(1, 1) = 0$, which models both the contagion mode triggered by the value 1 (changes 0 to 1, from the centre of the grid filled with 0 values) and the healing mode triggered also by the value 1 (changes 1 to 0). Then we have $R_6(0, 1) = 1$ and $R_6(0, 0) = 0$ which means the value 0 has no effect on 0 nor 1. Fig. 2. shows the

contagion/healing process for the SIS model in a 5x5 grid for a Von Neumann neighbourhood. We assume that the healing process always begins at t+2, that is, 2 generations after the contagion.

0	0	0	0	0
0	0	0	0	0
0	0	1	0	0
0	0	0	0	0
0	0	0	0	0

t=0 (one infected in the centre of the grid).

0	0	0	0	0
0	0	1	0	0
0	1	1	1	0
0	0	1	0	0
0	0	0	0	0

t=1 (4 new infected hosts).

0	0	1	0	0
0	1	1	1	0
1	1	0	1	1
0	1	1	1	0
0	0	1	0	0

t=2 (8 new infected hosts. The host infected at t=0 is now susceptible)

0	1	1	1	0
1	1	0	1	1
1	0	1	0	1
1	1	0	1	1
0	1	1	1	0

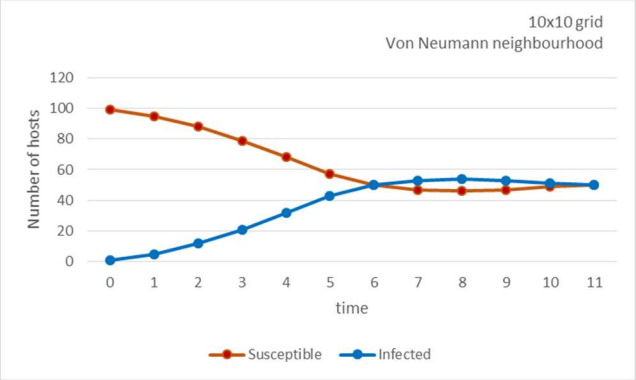
t=3 (8+1 new infected hosts. The host infected at t=0 and susceptible at t=2 is infected again. The hosts infected at t=1 are now susceptible.)

1	1	0	1	1
1	0	1	0	1
0	1	0	1	0
1	0	1	0	1
1	1	0	1	1

t=4 (4+4 new infected hosts. The hosts infected at t=2 are now susceptible as well as the host infected at t=0)

Fig.2. Contagion/healing process for the SIS model in a 5x5 grid for a Von Neuman neighborhood

Fig.3. plots the evolution of the number of Susceptible/Infected hosts vs. time. In this case we have a 10x10 grid and the three types of connectivity. We assume here that the healing begins at t+2, that is to say 2 generations after the contagion.



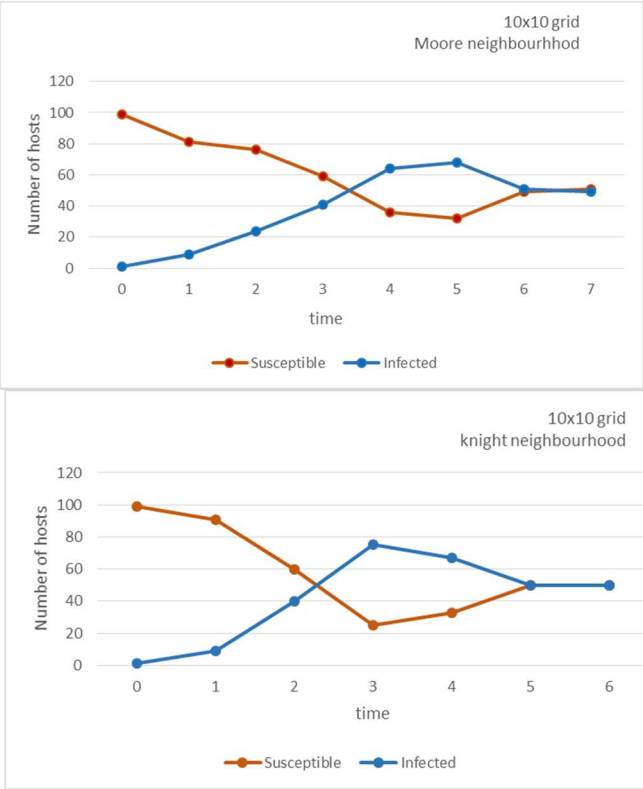


Fig.3. Number of Susceptible/Infected hosts vs. time in the case of a 10x10 grid for the three types of connectivity (healing begins 2 generations after contagion)

We observe in the graphics Fig.3. that the three types of connectivity cause more or less crossing of the Susceptible and Infected curves that always reach the steady state with the value N/2. The Von Neumann neighbourhood for a grid of 10x10 shows similarity with the deterministic model for N=100 and γ= 0,5. In order to study the influence of the time it takes for an infected host to become susceptible again, we analyze the process assuming that the healing begins at t+3 and at t+1, that is, 3 or 1 generation after the contagion, respectively. See Fig.4. and Fig.5.

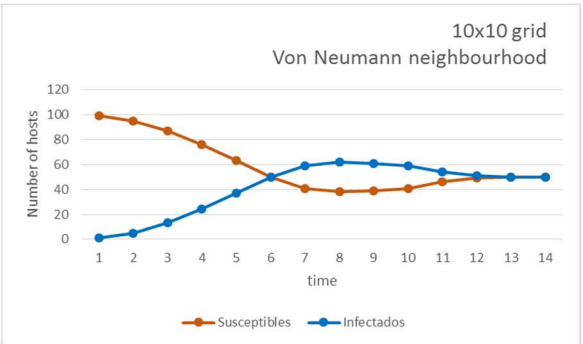


Fig.4. Number of Susceptible/Infected hosts vs. time, 10x10 grid, Von Neumann neighbourhood (healing begins 3 generations after contagion)

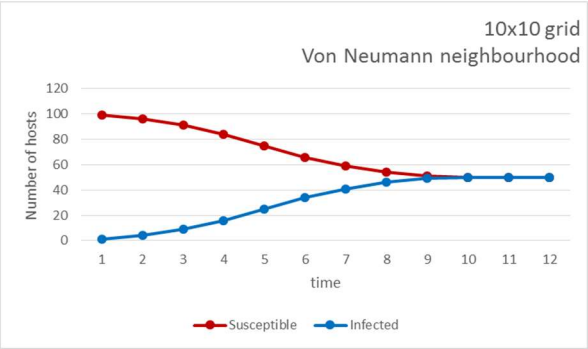


Fig.5. Number of Susceptible/Infected hosts vs. time, 10x10 grid, Von Neumann neighbourhood (healing begins 1 generation after contagion)

In Fig.4. and Fig.5. we observe that the longer the time for healing, the longer it takes to reach the value N/2, and the shorter the time, the less important is the crossing of the curves. Fig. 5. exhibits the strongest similarity with the deterministic model for N=100 and γ= 0,5. Although a deeper analysis is needed in order to better assess our model, it appears that our approach can meet most of the results provided by the deterministic SIS model.

4 Discussion

This empirical approach builds a link between two conceptually different models: the continuous and discrete one. Table 1 links the corresponding parameters. The two main contributions of our model are the rate at which susceptible hosts become infected (rβ) which is implemented in our model by the combination of a neighbourhood type and a local rule, and the rate at which infected hosts become susceptible (γ) which is carried out by the delay between contagion and healing.

Parameters	Deterministic SIS model	Our SIS model
Population size	N	nxn grid
The rate at which susceptible hosts become infected	rβ	Neighbourhood type + R_m
S(0)	N-1	nxn-1
I(0)	1	1
The rate at which infected hosts become susceptible	γ	Delay between contagion and healing

Table 1. Comparison between the parameters of the models

We have also pointed out the influence of this delay on the time to reach the steady state value and the more or less crossing curves. Some questions that have not been considered in this paper will be considered in future research, such as the stabilization at different steady state values for Susceptible and Infected, or the combination of different local rules, or the definition of new neighbourhood relationships.

5 Conclusion

This paper is based on previous own research [8, 9], which aims to establish a link between traditional deterministic models of infectious disease expansion and a very simple approach based on both connectivity between people, defined by means of a neighbourhood type, and a set of elementary local rules that define the result of these contacts. The initial satisfactory results encourage to pursue the improvement of the model to apply to real scenarios.

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