Journal of Pure and Applied Mathematics: Advances and Applications
Volume 5, Number 2, 2011, Pages 125-139

A MODEL BASED ON CELLULAR AUTOMATA TO SIMULATE A SIS EPIDEMIC DISEASE

M. J. FRESNADILLO MARTÍNEZ[1], E. GARCÍA SÁNCHEZ[1], J. E. GARCÍA SÁNCHEZ[1], A. MARTÍN DEL REY[2] and G. RODRÍGUEZ SÁNCHEZ[2]

[1]Department of Preventive Medicine
Public Health and Medic Microbiology
Universidad de Salamanca
Spain

[2]Department of Applied Mathematics
Universidad de Salamanca
Spain
e-mail: delrey@usal.es

Abstract

In this work, a novel model to simulate epidemic spreading is introduced. It is based on the use of two-dimensional cellular automata, where each cell stand for a square portion of the environment. It is suppose that the distribution of the population is homogeneous, that is, all cells have the same population. The laboratory simulations obtained seem to be in agreement with the real behaviour of epidemic spreading.

1. Introduction

The public health issues have a lot of importance in our society, particularly viral spread through populated areas. Epidemics refer to a disease that spreads rapidly and extensively by infection and affecting

2010 Mathematics Subject Classification: 68Q80, 92D30.

Keywords and phrases: cellular automata, epidemic spreading, SIS model.

Received May 9, 2011

© 2011 Scientific Advances Publishers

many individuals in an area at the same time. In this way, the most recent worrying epidemic was the "Severe Acute Respiratory Syndrome" (SARS) outbreak in Asia. Infectious disease accounts for 29 of 96 major causes of human morbidity and mortality listed by the World Health Organization (WHO) and the World Bank, and 25% of global deaths (over 14 million deaths annually). Consequently, since the publication of the first modern mathematical epidemic models in the first years of 20th century (see [5, 8]), several mathematical models to study the dynamics of epidemics have been appeared in the literature.

Traditionally, mathematical models are based on differential equations. Nevertheless, this approach has some drawbacks since they do not take into account spatial factors such as population density, they neglect the local character of the spreading process, they do not include variable susceptibility of individuals, etc.. As a consequence, this can lead to very unrealistic results, such as, for example, endemic patterns relaying on very small densities of individuals, which are called "*atto-foxes*" or "*nano-hawks*" (see [7]). Other mathematical models are based on a particular type of discrete dynamical systems called *cellular automata* (see, for example, [2, 6, 9, 10, 12]). These simple models eliminate the last mentioned shortcomings, and are specially suitable for computer simulations. Roughly speaking, cellular automata (CA for short) are simple models of computation capable to simulate physical, biological or environmental complex phenomena. Consequently, several models based on such mathematical objects have been proposed to simulate growth processes, reaction-diffusion systems, self-reproduction models, epidemic models, forest fire spreading, image processing algorithms, cryptographic protocols, etc. (see, for example, [11, 13]). Specifically, a two-dimensional CA is formed by a two-dimensional array of identical objects called *cells*, which can be disposed in a rectangular, triangular or a hexagonal lattice. These cells are endowed with a state that changes in discrete steps of time according to a specific rule. As the CA evolves, the updated function (whose variables are the states of the neighbour cells) determines how local interactions can influence the global behaviour of the system.

Usually, mathematical models to study epidemic spreading are divided into three types: SIS models, SIR models, and SEIR models, depending on the lasses in which the population can be classified. The model introduced in this paper deals with SIS epidemic diseases (for example, the group of those responsible for the common cold), that is, the population is divided into susceptible individuals (S) and infected individuals (I). The susceptible individuals are those capable to contracting the disease, whereas the infected individuals are those capable of spreading the disease. For an SIS model, infected individuals return to the susceptible class on recovery because the disease confers no immunity against reinfection.

Moreover, some assumptions will be common to all models:

- The disease is transmitted by contact between an infected individual and a susceptible individual.

- There is no latent period for the disease, hence the disease is transmitted instantaneously upon contact.

- All susceptible individuals are equally susceptible and all infected individuals are equally infectious.

- The population under consideration is fixed in size. This means that no births or migration occurs, and all deaths are taken into account.

The main goal of this work is to introduce a new SIS model to simulate the spread of a general epidemic based on cellular automata. Specifically, in the proposed model, the state of each cell stands for the fraction of the susceptible and infected individuals of the cell at a particular time step. The local transition function is a linear function involving the states of the neighbour cells and other parameters such as the virulence of the epidemic, the rate of recovered infected individuals, etc.. Moreover, two different lattices for cellular space will be used: rectangular and hexagonal lattices.

Unfortunately, there are not many CA-based algorithms to simulate an SIS epidemic model (see, for example, [1, 3, 4]). The standard paradigm of these models states that each cell stands for an only one

individual. In this sense, the main advantage of this model in opposite to the existing SIS models is that each cell stands not for only one individual but for many individuals.

The rest of the paper is organized as follows. In Section 2, a review of two-dimensional cellular automata is given; the proposed model is introduced in Section 3; some graphical simulations are shown in Section 4; and finally, the conclusions and the future work are presented in Section 5.

2. Two-Dimensional Cellular Automata

Two-dimensional cellular automata are discrete dynamical systems formed by a finite number of identical objects called *cells*, which are arranged uniformly in a two-dimensional space. Each cell is endowed with a state, belonging to a finite state set, that changes at every discrete step of time according to a local transition function. More precisely, a CA can be defined as a 4-uplet, $\mathcal{A} = (C, S, V, f)$, where C is the cellular space formed by a two-dimensional array of $r \times c$ cells: $\{(i, j), 1 \le i \le r, 1 \le j \le c\}$. Traditionally, the cells have been represented as identical square areas (see Figure 1(a)), but in this work, the cells will be also represented by means of regular hexagonal areas (see Figure 1(b)), making a tesellation of the plane.

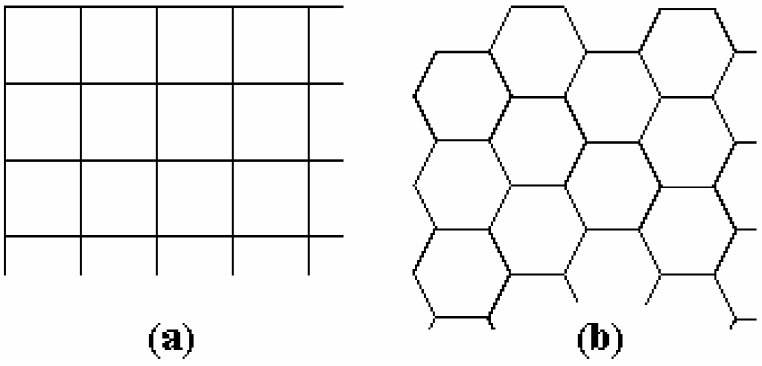

Figure 1. (a) Rectangular cellular space; (b) hexagonal cellular space.

The state of a cell (i, j) at time t is denoted by $s_{ij}^t \in S$, and the matrix $C^t = \left(s_{ij}^t\right)$ is called *configuration of the CA at time t*, where C^0 is the initial configuration. The neighborhood of a cell is the set of all cells, whose states at time t determines the state of the main cell at time $(t+1)$ by means of the local transition function. Depending on the process to be modelled, one can choose an appropriate neighborhood. In this work, we will consider for rectangular cellular spaces the Von Neumann neighborhood (see Figure 2(a)), and the Moore neighborhood (see Figure 2(b)), whereas for hexagonal cellular space, the hexagonal Moore neighborhood (see Figure 2(c)) is considered.

A neighborhood is defined by a finite subset of indices $V \subset \mathbb{Z} \times \mathbb{Z}$, $|V| = m$, such that for every cell (i, j), its neighborhood $V_{(i,j)}$ is the ordered set of m cells given by $V_{(i,j)} = \{(i+\alpha_1, j+\beta_1), \ldots, (i+\alpha_m, j+\beta_m) : (\alpha_k, \beta_k) \in V\}$.

In the case of Von Neumann neighborhood, it is

$$V = \{(0, 0), (-1, 0), (0, 1), (1, 0), (0, -1)\},$$

and if Moore neighborhoods are considered, then

$$V = \{(0, 0), (-1, 0), (-1, 1), (0, 1), (1, 1), (1, 0), (1, -1), (0, -1), (-1, -1)\}.$$

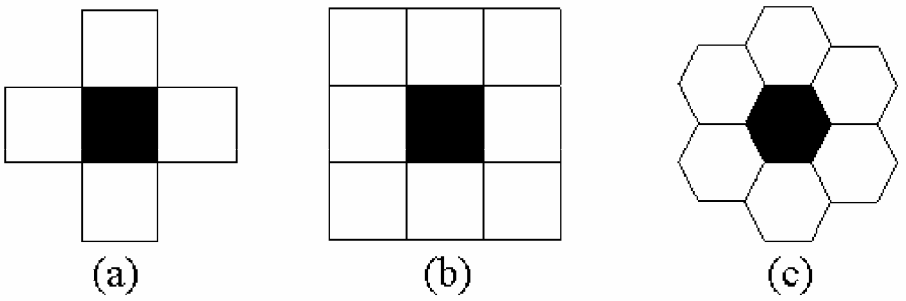

Figure 2. (a) Von Neumann neighborhood; (b) Moore neighborhood; and (c) hexagonal Moore neighborhood.

When hexagonal cellular spaces are taken, the set of indices V depends on the cell, (i, j), considered. Specifically, if j is odd, then the hexagonal Moore neighborhood is given by $V = \{(0, 0), (-1, 0), (0, 1), (1, 1), (1, 0), (1, -1), (0, -1)\}$, whereas if j is even, then the hexagonal Moore neighborhood is given by

$$V = \{(0, 0), (-1, 0), (-1, 1), (0, 1), (1, 0), (0, -1), (-1, -1)\}.$$

Moreover, we set $V^* = V - \{(0, 0)\}$.

As is mentioned above, the CA evolves deterministically in discrete time steps, changing the states of all cells according to a local transition function $f : S^m \to S$. The updated state of the cell (i, j) depends on the m variables of the local transition function, which are the previous states of the cells constituting its neighborhood, that is,

$$s_{ij}^{t+1} = f(s_{i+\alpha_1, j+\beta_1}^t, \ldots, s_{i+\alpha_m, j+\beta_m}^t).$$

As the cellular space is considered to be finite, boundary conditions must be considered in order to assure the well-defined dynamics of the CA. One can state several boundary conditions depending on the simulation, but in the proposed model, null boundary conditions will be considered, that is: $s_{ij}^t = 0$ if $i < 1$ or $i > r$, or if $j < 1$ or $j > c$.

3. Description of the Model

In the proposed model, it is suppose that the ground where the epidemic is spreading stands for the cellular space of the CA considered, which is divided into identical square or hexagonal areas, each one of them representing a cell of the CA. The main features of the epidemic and the environment are the following:

(1) The epidemic is not lethal and no birth, immigration or emigration is considered. As a consequence, the total amount of population is constant.

(2) The population distribution is homogeneous being N the total population of each cell.

(3) The way of infection is the personal contact between an infected individual and a healthy one.

(4) Once the healthy individuals have contracted the disease and have recovered from it, they are susceptible again.

(5) People can move from one cell to another cell.

Let $s_{ij}^t \in [0, 1]$ be the portion of the healthy individuals of the cell (i, j), who are susceptible to infection at time t and set $I_{ij}^t \in [0, 1]$ be the portion of the infected population of the cell, who can transmit the disease to the healthy ones. As the population of each cell is constant, then $1 = S_{ij}^t + I_{ij}^t$, for every time step t and every cell (i, j). The state of each cell is a vector specifying the susceptible and infected individuals of the cell at the particular time step. As S_{ij}^t and I_{ij}^t are real numbers between 0 and 1, and the state set of the CA must be finite, then a suitable discretization of such parameters must be given. In this work, we will consider $0 \le N \le 100$, and consequently, the state set used will be $S = Q \times Q$, where

$$Q = \{0.00, 0.01, 0.02, \ldots, 0.99, 1.00\}.$$

As a consequence, the state of the cell (i, j) is $s_{ij}^t = (DS_{ij}^t, DI_{ij}^t) \in S$, with

$$DS_{ij}^t = \frac{[100 \cdot S_{ij}^t]}{100}, \quad DI_{ij}^t = \frac{[100 \cdot I_{ij}^t]}{100},$$

where $[x]$ is the nearest integer to x.

The local transition function of the CA-based model is the following:

$$I_{ij}^t = (1 - \varepsilon) I_{ij}^{t-1} + \upsilon S_{ij}^{t-1} I_{ij}^{t-1} + \sum_{(\alpha, \beta) \in V^*} \mu_{\alpha\beta}^{(i,j)} S_{ij}^{t-1} I_{i+\alpha, j+\beta}^{t-1}, \tag{1}$$

$$S_{ij}^{t} = S_{ij}^{t-1} + \varepsilon I_{ij}^{t-1} - \upsilon S_{ij}^{t-1} I_{ij}^{t-1} - \sum_{(\alpha,\beta)\in V^{*}} \mu_{\alpha\beta}^{(i,j)} S_{ij}^{t-1} I_{i+\alpha,\, j+\beta}^{t-1}, \qquad (2)$$

with $\mu_{\alpha\beta}^{(i,j)} = c_{\alpha\beta}^{(i,j)} m_{\alpha\beta}^{(i,j)} \upsilon$, where $c_{\alpha\beta}^{(i,j)}$ stands for the connection factor, $m_{\alpha\beta}^{(i,j)}$ is the movement factor between the main cell (i, j) and the neighbour cell $(i + \alpha, j + \beta)$, $\upsilon \in [0, 1]$ is the virulence of the epidemic, and $\varepsilon \in [0, 1]$ stands for the portion of infected individuals, which are recovered and becomes to be susceptible at each time step.

The Equation (1) can be interpreted as saying that the portion of infected individuals of the cell (i, j) at a particular time step t is given by:

(1) The portion of infected individuals of this cell, which have not been recovered from the disease (first sum of the summation).

(2) The portion of susceptible individuals of the same cell at time $(t - 1)$, which have been infected by the infected individuals at time $(t - 1)$ of the cell (second sum of the summation).

(3) The portion of the susceptible individuals of the cell, which have been infected by infected individuals of the neighbour cells, which have travelled to the cell (i, j) (third sum of the summation).

Moreover, Equation (2) gives the portion of susceptible individuals of the cell (i, j) at time t as the difference between the portion of susceptible individuals at the previous time step plus the infected individuals, which becomes to be susceptible, and the portion of susceptible individuals, which have been infected. Note that, as a simple calculus shows, $S_{ij}^{t} + I_{ij}^{t} = 1$.

As is mentioned above, the way of infection of the epidemic considered is the contact between a sick individual and a healthy one. As a consequence, the healthy individuals of a particular cell can be infected by the infected individuals of this cell or by the infected individuals of the neighbour cells that have travelled to the main cell. The first case, that is,

when an individual is infected by another individual of his/her cell, is reflected in the first sum of the summation given in (1). On the other hand, i.e., when the infection is carried out by individuals belonging to neighbour cells, some type of connection between the cells must be exist, $c_{\alpha\beta}^{(i,j)} \in [0, 1]$, in order to allow the epidemic spreading. It is given by the second sum of the summation of (1). The parameter $m_{\alpha\beta}^{(i,j)} \in [0, 1]$ stands for the probability of an infected individual belonging to the neighbour cell $(i + \alpha, j + \beta)$ to be moved to the main cell (i, j).

It is very important to decide whether or not the outbreak disease occurs. In this sense, the epidemic spread from one cell, (i, j) to its neighbour cells if

$$I_{ij}^{0} \geq \frac{\min \{s \in Q - \{0\}\}}{v c_{ij} m_{ij}},$$

where $c_{ij} = \max \{c_{\alpha,\beta}^{(i,j)}, (\alpha, \beta) \in V^*\}$ and $m_{ij} = \max \{m_{\alpha,\beta}^{(i,j)}, (\alpha, \beta) \in V^*\}$. Moreover, the number of infected individuals of a particular cell (i, j) grows if $S_{ij}^{t} > \varepsilon / v$, if there is not infected neighbour cells, or if

$$S_{ij}^{t} > \frac{\varepsilon}{v} \frac{I_{i,j}^{t}}{I_{i,j}^{t} + \sum_{(\alpha,\beta)\in V^*} c_{\alpha,\beta}^{(i,j)} m_{\alpha,\beta}^{(i,j)} I_{i+\alpha,j+\beta}^{t}},$$

if there are infected neighbour cells.

4. Simulations

The cellular space in the next simulations will be formed by a two-dimensional array of 40×40 cells and also, it is suppose that $m_{\alpha\beta}^{(i,j)} = 0.5$ for all (i, j), $v = \epsilon = 0.5$.

Furthermore, the initial configuration is formed by all cells without infected individuals with except of the central cell, whose state is given by $s^0_{20,20} = (0.85, 0.15)$. Note that for the sake of simplicity, these parameters are artificially chosen. Finally, let us consider the case in which each cell is connected with all of its neighbour cells, that is, $c^{(i,j)}_{\alpha\beta} = 1$ for all (i, j) and $(\alpha, \beta) \in V^*$. Moreover, the size of the time step and the size of cells must be considered according to the main characteristic of the epidemic and the environment. In the graphic simulations, only the portion of infected individuals is considered in each cell according to a gray-level code: The colour runs from white for the cells (i, j) with $I^t_{ij} = 0$ at time step t, to black for cells (i, j) with $I^t_{ij} = 1$. Moreover, only the configurations at times $t = 0, 5, 10, 15, 20, 25$ are shown. In Figures 3 and 4, the simulations obtained by using a CA with rectangular array and Von Neumann and Moore neighborhoods, respectively, are shown.

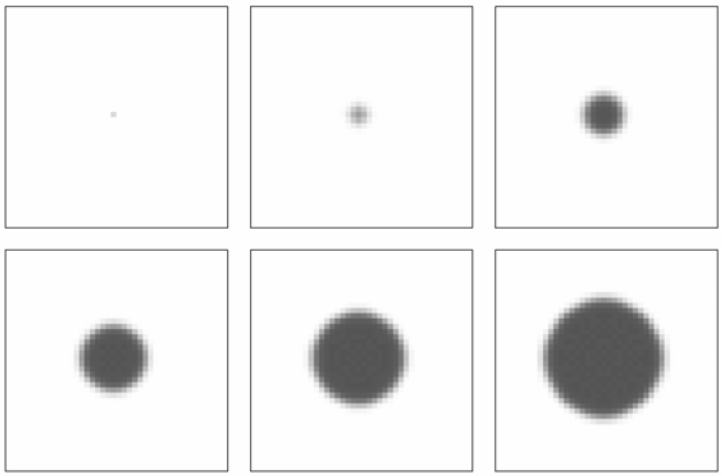

Figure 3. Simulations obtained with Von Neumann neighborhoods.

The simulation presented in Figure 5 is obtained by using a hexagonal CA endowed with hexagonal Moore neighborhoods.

Note that the number of infected individuals increases as the number of neighbour cells also increases. Also, the speed of propagation of the front of the epidemic is higher in the cases with more neighbour cells: 8 cells in Moore neighborhood and 6 cells in hexagonal Moore neighborhood. Finally, remark that when hexagonal cellular space is considered, the shape of the epidemic fronts is more similar to circular shape than in the case of rectangular cellular space.

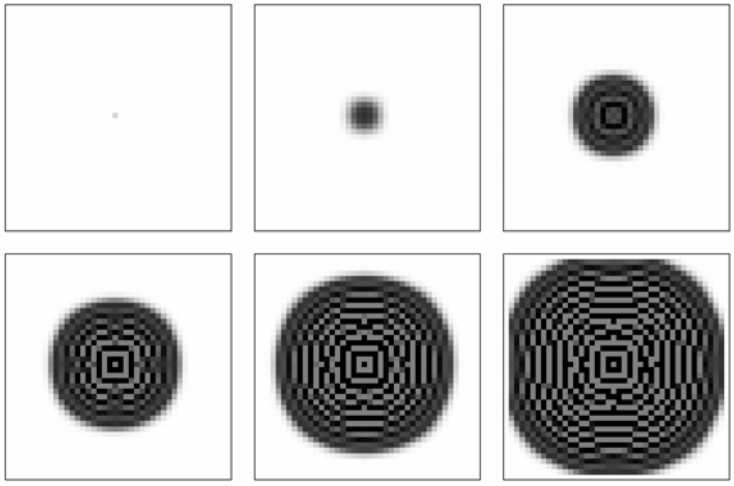

Figure 4. Simulations obtained with Moore neighborhoods.

In Figures 6 and 7, the evolutions of the number of susceptible and infected individuals are shown for rectangular cellular spaces with Von Neumann and Moore neighborhoods, respectively.

On the other hand, the evolutions of these classes of population for hexagonal cellular spaces are also shown in Figure 8 for hexagonal Moore neighborhoods.

These graphics shows again that the number of infected individuals with Moore and hexagonal Moore neighborhoods increases rapidly than with Von Neumann neighborhoods.

In Figure 9, the comparison between the evolution of infected population considering rectangular and hexagonal cellular spaces and Von Neumann, Moore, and hexagonal Moore neighborhoods are shown. Note that for each type of cellular space considered, the number of infected individuals grows as the number of neighbour cells, and although the number of infected individuals with Moore neighborhoods in the rectangular case grows more rapidly than with Hexagonal Moore neighborhoods in the hexagonal case, both curves converge.

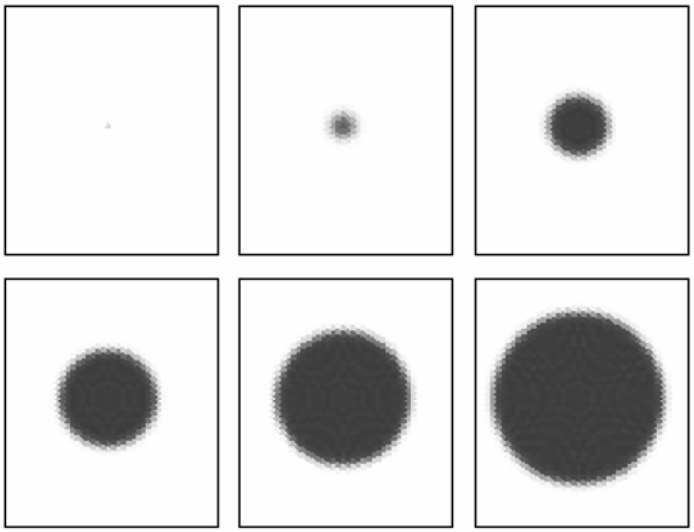

Figure 5. Simulations obtained with hexagonal Moore neighborhoods.

5. Conclusions and Future Work

In this work, a new mathematical model to simulate the spreading of an epidemic is introduced. It is based on the use of two-dimensional cellular automata endowed with a suitable local transition function. The state of each cell is considered to be the portion of its population, which is infected at each time step. Rectangular and hexagonal cellular spaces are considered with different types of neighborhoods: Von Neumann neighborhood, Moore neighborhood, hexagonal Moore neighborhood, and extended Moore neighborhood. It is shown that the simulations obtained

are more acute when hexagonal cellular space is considered. Moreover, the laboratory simulations obtained seem to be in agreement with the expected behaviour of a real epidemic. Future work aimed at designing a more complete CA-based epidemic model involving additional effects such as the population movement, virus mutation, etc.. Furthermore, it is also interesting to consider non-constant connections factors.

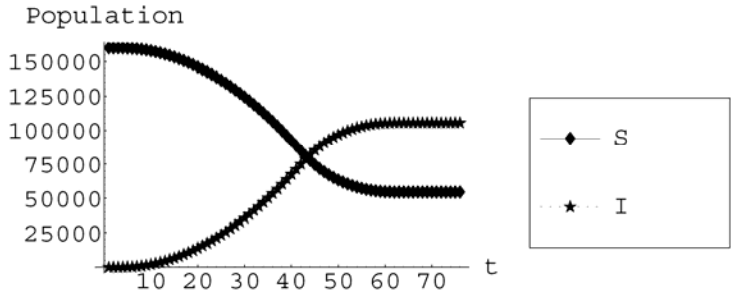

Figure 6. Evolution of susceptible and infected individuals with Von Neumann neighborhoods.

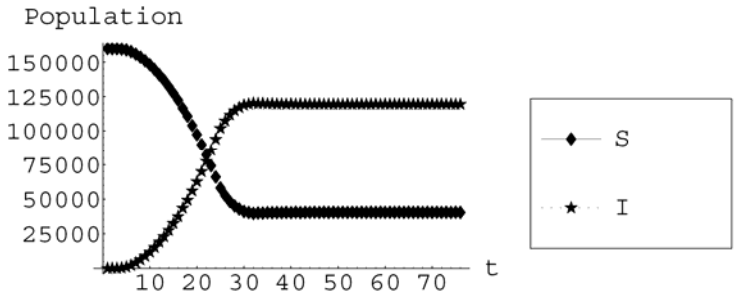

Figure 7. Evolution of susceptible and infected individuals with Moore neighborhoods.

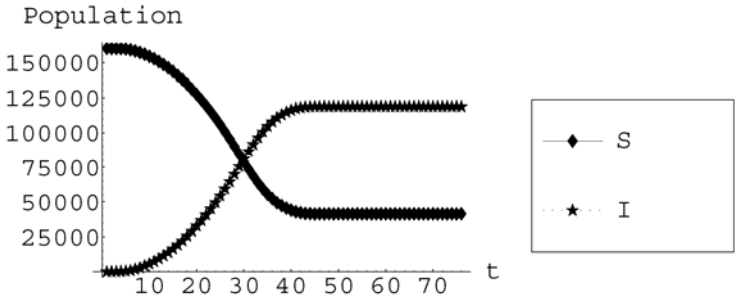

Figure 8. Evolution of susceptible and infected individuals with hexagonal Moore neighborhoods.

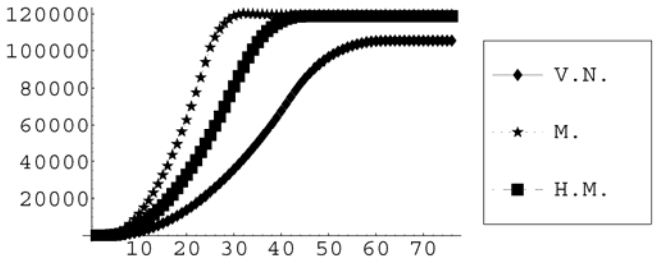

Figure 9. Evolution of infected individuals with Von Neumann, Moore, and hexagonal Moore neighborhoods.

References

[1] E. Ahmed and A. S. Elgazzar, On some applications of cellular automata, Physica A 296 (2001), 529.

[2] C. Beauchemin, J. Samuel and J. Tuszynski, A simple cellular automaton models for influenza: A viral infections, J. Theor. Biol. 232 (2005), 223.

[3] N. Boccara and K. Cheong, Critical behaviour of a probablistic automata network SIS model for the spread of an infectious disease in a population of moving individuals, J. Phys. A-Math. Gen. 26 (1993), 3707.

[4] M. A. Fuentes and M. N. Kuperman, Cellular automata and epidemiological models with spatial dependence, Physica A 267 (1999), 471.

[5] W. O. Kermack and A. G. McKendrick, Contributions to the mathematical theory of epidemics, part I, Proc. Roy. Soc. Edin. A 115 (1927), 700.

[6] M. L. Martins et al., A cellular automata model for citrus variegated chlorosis, Physica A 295 (2001), 42.

[7] D. Molisson, The dependence of epidemic and population velocities on basic parameters, Math. Biosci. 107 (1991), 255.

[8] R. Ross, The Prevention of Malaria, 2nd Edition, Murray, London, 1911.

[9] J. Satsuma et al., Extending the SIR epidemic model, Physica A 336 (2004), 369.

[10] G. Ch. Sirakoulis, I. Karafyllidis and A. Thanailakis, A cellular automaton model for the effects of population movement and vaccination on epidemic propagation, Ecol. Model. 133 (2000), 209.

[11] T. Toffoli and N. Margolus, Cellular Automata Machines: A New Environment for Modelling, The MIT Press, 1987.

[12] S. Hoya White, A. Martín del Rey and G. Rodríguez Sánchez, A model based on cellular automata to simulate epidemic diseases, Proc. ACRI 2006, Lecture Notes in Computer Science 4173 (2006), 304.

[13] W. Wolfram, A New Kind of Science, Wolfram Media Inc., 2002.

■