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ScienceDirect
Applied Mathematics and Computation 186 (2007) 193–202
APPLIED MATHEMATICS AND COMPUTATION
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Modeling epidemics using cellular automata

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Abstract

The main goal of this work is to introduce a theoretical model, based on cellular automata, to simulate epidemic spreading. Specifically, it divides the population into three classes: susceptible, infected and recovered, and the state of each cell stands for the portion of these classes of individuals in the cell at every step of time. The effect of population vaccination is also considered. The proposed model can serve as a basis for the development of other algorithms to simulate real epidemics based on real data.

Keywords: Cellular automata; Epidemic spreading; Mathematical modeling; Rectangular lattices; SIR model

1. Introduction

Nowadays, public health issues have a lot of importance in our society, particularly viral spread through populated areas. Epidemics refer to a disease that spreads extensively and rapidly by infection and affecting many individuals in an area or a population at the same time. Some examples of epidemics are the Black Death during the mid-14th century, the so-called Spanish Flu pandemic in 1918, the Severe Acute Respiratory Syndrome, better known by its acronym SARS, in 2002, or more recently, the Avian Influenza.

Whilst a single infected host might not be significant, a disease that spreads through a large population yields serious health and economic threats. Consequently, since the first years of the last century, an interdisciplinary effort to study the spreading of a disease in a social system has been made. In this sense, mathematical epidemiology is concerned with modeling the spread of infectious disease in a population. The aim is generally to understand the time course of the disease with the goal of controlling its spread. Such models are used, for example, to guide policy in vaccination strategies for childhood diseases.

Mathematical modeling in epidemiology was pioneered by Bernoulli in 1760 in his work demonstrating the effectiveness of the technique of variolation against smallpox (see [1]), although the search for understanding of the dynamics of epidemic spreading goes back to 'Epidemics' by Hippocrates. Nevertheless, the work due to

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0096-3003/$ - see front matter © 2006 Elsevier Inc. All rights reserved.
doi:10.1016/j.amc.2006.06.126

Kermack and McKendrick in 1927 (see [2]) can be considered as the starting point for the design of modern mathematical models. It consists of a SIR model. Specifically, one can consider some types of mathematical models depending on the division of the population into classes. So, we have the SIR models where susceptible (S), infected (I), and recovered (R) individuals are considered. The susceptible individuals are those capable to contracting the disease; the infected individuals are those capable of spreading the disease; and the recovered individuals are those immune from the disease, either died from the disease, or, having recovered, are definitely immune to it. For many infections there is a period of time during which the individual has been infected but is not yet infectious himself; during this latent period the individual is said to be exposed. In this case we have the SEIR model in which the new class of exposed individuals (E) must be considered. Some infections, for example the group of those responsible for the common cold, do not confer any long lasting immunity. Such infections do not have a recovered state and individuals become susceptible again after infection. Then we have the SIS models. Moreover, there are another variants of these models such as the SIRS model or the SEIRS model.

Traditionally, the majority of existing mathematical models to simulate epidemics are based on ordinary differential equations. These models have serious drawbacks in that they neglect the local characteristics of the spreading process and they do not include variable susceptibility of individuals. Specifically, they fail to simulate in a proper way (1) the individual contact processes, (2) the effects of individual behaviour, (3) the spatial aspects of the epidemic spreading, and (4) the effects of mixing patterns of the individuals.

Cellular automata (CA for short) can overcome these drawbacks and have been used by several researches as an efficient alternative method to simulate epidemic spreading (see, for example, [3–13], apart from another works appeared in the life sciences and computing literature). Of special interest are the CA-epidemic proposals modeling the motion of individuals (see, for example [14–16]). Roughly speaking, cellular automata are simple models of computation capable to simulate physical, biological or environmental complex phenomena. Consequently, several models based on such mathematical objects have been appeared in the literature to simulate growth processes, reaction-diffusion systems, self-reproduction models, epidemic models, forest fire spreading, image processing algorithms, etc. (see, for example, [17]). Specifically, a two-dimensional CA is formed by a two-dimensional array of identical objects called cells, which 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 neighbors cells) determines how local interactions can influence the global behaviour of the system.

Usually, when a CA-based model is considered to simulate an epidemic spreading, individuals are assumed to be distributed in the cellular space such that each cell stands for an individual of the population. In this work, a mathematical deterministic model to simulate epidemic spreading is introduced. It is based on cellular automata, and three classes of population are considered: susceptible, infected and recovered. Furthermore, in each cell several individuals are considered instead of only one individual, as is stated in the majority of proposals appeared in the literature, since the proposed model try to simulate epidemic spreading in large regions. Consequently, each cell stands for an square portion of the land and its state is obtained from the fraction of the number of individuals which are susceptible, infected, or recovered from the disease. Moreover, in the proposed model the vaccination process can be considered.

The rest of the paper is organized as follows: In Section 2 the basic results about cellular automata are introduced; the model to simulate the epidemic spreading is presented in Section 3; in Section 4 some simulations using artificially chosen parameters are shown, and, finally, the conclusions are introduced in Section 5.

2. Overview of cellular automata

Bidimensional cellular automata are discrete dynamical systems formed by a finite number of $r \times c$ identical objects called cells which are arranged uniformly in a two-dimensional cellular space. Each cell is endowed with a state (from a finite state set Q), that changes at every step of time accordingly to a local transition rule. In this sense, the state of a particular cell at time t depends on the states of a set of cells, called its neighborhood, at the previous time step $t - 1$. More precisely, a CA is defined by the 4-uplet (C, Q, V, f), where C is the cellular space:

$$C = \{(i,j),\ 1 \leqslant i \leqslant r,\ 1 \leqslant j \leqslant c\}; \tag{1}$$

Q is the finite state set whose elements are the all possible states of the cells; $V = \{(\alpha_k, \beta_k), 1 \leqslant k \leqslant n\} \subset Z \times Z$, is the finite set of indices defining the neighborhood of each cell, such that the neighborhood of the cell (i,j) is

$$V_{ij} = \{(i + \alpha_1, j + \beta_1), \ldots, (i + \alpha_n, j + \beta_n)\}. \tag{2}$$

Moreover, $V^* = V - \{(0,0)\}$. Finally, the function f is the local transition function:

$$s_{ij}^t = f(s_{i+\alpha_1, j+\beta_1}^{t-1}, \ldots, s_{i+\alpha_n, j+\beta_n}^{t-1}) \in Q, \tag{3}$$

where s_{ij}^t stands for the state of the cell (i,j) at time t.

As is mentioned above, the cells are represented as identical square areas forming the cellular space (see Fig. 1(a)). The most important types of neighborhoods are Von Neumann neighborhood (see Fig. 1(b)) given by the cell itself and the four cells placed at north, south, east and west, and Moore neighborhood (see Fig. 1(c)), formed by the cell itself and its eight nearest cells.

The set of indices for Von Neumann neighborhoods is the following:

$$V = \{(0,0), (-1,0), (0,1), (1,0), (0,-1)\}, \tag{4}$$

whereas for Moore neighbourhoods V is defined as follows:

$$V = \{(0,0), (-1,0), (-1,1), (0,1), (1,1), (1,0), (1,-1), (0,-1), (-1,-1)\}, \tag{5}$$

As is mentioned above, the CA evolves deterministically in discrete time steps, changing the states of the cells by means of the local transition function f. As the cellular space is considered to be finite, boundary conditions must be considered in order to assure a well-defined dynamics of the CA. These boundary conditions depends on the process to be simulated; in this work, we will use null boundary conditions, that is, $s_{ij}^t = 0$ if $(i,j) \notin C$.

3. The proposed model

In this section, we introduce the mathematical model based on cellular automata, to simulate the spreading of a general epidemic. It is suppose that the ground where the epidemic is spreading stands for the cellular space of the CA, and it is divided into identical square areas, each of them represent a cell of the CA. Different cells will have different populations: differing densities and different 'across cell' traversal or mobility properties. Moreover, the cellular space is considered to be large enough to ensure that the epidemic spreading affects only to the central region. Indeed, in this case, the null boundary conditions stand for absorbing boundary conditions.

The main features of the epidemic and the environment where it is spreading are the following:

- The epidemic is not lethal and no birth, immigration or emigration is considered; consequently, the total amount of population is constant. As a consequence, the population of each cell is always the same.
- The population distribution is inhomogeneous, that is, the total population living in each cell is different, and the total population of the cell (i,j) is N_{ij}.

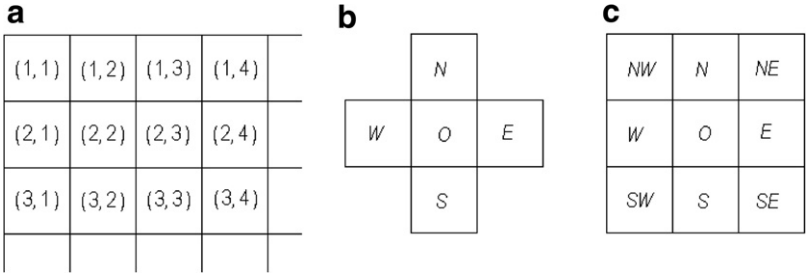

Fig. 1. (a) Rectangular cellular space. (b) Von Neumann neighborhood. (c) Moore neighborhood.

- It is suppose that the way of infection is the contact between the infected individual and the healthy individual.
- Once the healthy individuals have contracted the infection and have recovered from it, they acquire immunity. That is, they are definitely immune to the disease and consequently they will not be susceptible individuals.
- People can move from one cell to another (if there is some type of way of transport), that is, the individuals are able to go outside and come back inside their cells during each time step.
- It is suppose that when an infected individual arrives at a cell, the number of healthy individuals contacted by him/her is the same independently of the total amount of population of the 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; set $I_{ij}^t \in [0,1]$ the portion of the infected population of the cell at time t who can transmit the disease to the healthy ones; and let $R_{ij}^t \in [0,1]$ be the portion of recovered individuals of (i,j) from the disease at time t, that will be permanently immunised. As is stated above, the population of each cell is constant, consequently: $S_{ij}^t + I_{ij}^t + R_{ij}^t = 1$.

Moreover, set DS_{ij}^t, DI_{ij}^t, and DR_{ij}^t suitable discretizations of the fractions of the susceptible, infected and recovered population of the cell at time t, respectively, to get elements of the finite state set Q. In this work, we will consider the state set $Q = K \times K \times K$, where:

$$K = \{0.00, 0.01, 0.02, 0.03, \ldots, 0.99, 1\}, \tag{6}$$

which is formed by 101 elements. Consequently, the discretization used is:

$$DI_{ij}^t = \frac{[100 \cdot I_{ij}^t]}{100}, \quad DR_{ij}^t = \frac{[100 \cdot R_{ij}^t]}{100}, \quad DS_{ij}^t = 1 - DI_{ij}^t - DR_{ij}^t, \tag{7}$$

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

Then, the state of the cellular automata used in the model is the three-uplet $s_{ij}^t = (DS_{ij}^t, DI_{ij}^t, DR_{ij}^t) \in Q$.

The main goal of the model is to compute the factors S_{ij}^t, I_{ij}^t and R_{ij}^t. The local transition function used is the following:

$$I_{ij}^t = (1-\varepsilon) \cdot I_{ij}^{t-1} + v \cdot S_{ij}^{t-1} \cdot I_{ij}^{t-1} + S_{ij}^{t-1} \cdot \sum_{(\alpha,\beta)\in V^*} \frac{N_{i+\alpha,j+\beta}}{N_{ij}} \cdot \mu_{\alpha\beta}^{(i,j)} \cdot I_{i+\alpha,j+\beta}^{t-1}, \tag{8}$$

$$S_{ij}^t = S_{ij}^{t-1} - v \cdot S_{ij}^{t-1} \cdot I_{ij}^{t-1} - S_{ij}^{t-1} \cdot \sum_{(\alpha,\beta)\in V^*} \frac{N_{i+\alpha,j+\beta}}{N_{ij}} \cdot \mu_{\alpha\beta}^{(i,j)} \cdot I_{i+\alpha,j+\beta}^{t-1}, \tag{9}$$

$$R_{ij}^t = R_{ij}^{t-1} + \varepsilon \cdot I_{ij}^{t-1}. \tag{10}$$

where $V^* = V - \{(0,0)\}$, and the real parameter $\mu_{\alpha\beta}^{(i,j)}$ is defined as the product of three factors: $\mu_{\alpha\beta}^{(i,j)} = c_{\alpha\beta}^{(i,j)} \cdot m_{\alpha\beta}^{(i,j)} \cdot v$, where $c_{\alpha\beta}^{(i,j)}$ and $m_{\alpha\beta}^{(i,j)}$ are the connection factor and the movement factor between the main cell (i,j) and its neighbour cell $(i+\alpha, j+\beta)$, respectively, and $v \in [0,1]$ is the virulence of the epidemic. Moreover, the parameter $\varepsilon \in [0,1]$ stands for the portion of infected individuals which recover from the disease at each time step.

Eqs. (8) and (10) reflect that every loss in the infected population is due to a gain in the recovered population, while every gain in the infected population is due to a loss in the susceptible population. Roughly speaking, the Eq. (8) can be interpreted as saying that the portion of infected individuals of a cell (i,j) at a particular time step t is given by the portion of infected individuals which have not been recovered from the disease (first sum of the summation) and by 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) taking into account the virulence of the disease. Moreover, some susceptible individuals of the cell (i,j) can be infected by infected individuals of the neighbour cells which have travelled to the cell (third sum of the summation). Obviously, it depends on some parameters involving the virulence, the nature of the connections between the cells, the possibilities of an infected individual to be moved from one cell to another, and the relation between the population of the cells. Furthermore, Eq. (10) gives the portion of recovered individuals of the cell (i,j) at time t as the number of recovered individuals of the cell at the previous time step plus the

fraction of infected individuals of the cell which have been recovered in one step of time. Finally, Eq. (9) gives the portion of susceptible individuals of the cell (i,j) at time step t as the portion of susceptible individuals at time $t-1$ which have not been infected.

Note that, as a simple calculus shows: $S_{ij}^t + I_{ij}^t + R_{ij}^t = 1$, for every cell (i,j) and every time step t.

As is mentioned above, the way of infection of the epidemic to be modeled is the contact between two individuals (an infected and a healthy individual). Consequently, 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 traveled 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 the Eq. (8). In the other case, given by the second sum of the summation of (8), when the infection is carried out by individuals belonging to neighbour cells, some type of connection between the cells must be exist in order to allow the epidemic spreading. In this work, we will consider three ways of transport: by airplane, by train and by car or bus. This connection is given by the coefficients $c_{\alpha\beta}^{(i,j)}$ such that:

$$c_{\alpha\beta}^{(i,j)} = \begin{cases} 1, & \text{if there exist the three ways of transport between the cells,} \\ 0.6, & \text{if there are two ways of transport between the cells,} \\ 0.3, & \text{if there is only one way of transport between the cells,} \\ 0, & \text{if there is not any way of transport between the cells,} \end{cases} \tag{11}$$

The movement factor $m_{\alpha\beta}^{(i,j)} \in [0,1]$ 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). Note that this parameter is different from the connection factor since it depends on the infected individuals and the other one (the connection factor) depends on the existing transport infrastructures between the cells considered. Moreover, the movement factor must be given by the main features of the disease to be modeled.

Finally, it is very important to decide whether or not the outbreak disease occurs. In this sense, we will obtain the values of the parameters for which the epidemic spread from one cell to its neighbor cells. Suppose that in the initial configuration there is only one cell with infected individuals: O, and set N its north neighbor cell. Then the infected individuals of N at time step $t=1$ is given by the following expression:

$$I_N^1 = \frac{N_C}{N_N} \cdot c_O^N \cdot m_O^N \cdot v \cdot I_O^0, \tag{12}$$

since $I_N^0 = 0$ and $S_N^0 = 1$.

In our model, we suppose that there are infected individuals in the cell N at a particular time step t when $DI_N^t \in Q - \{0\}$, that is, when $DI_N^t \geqslant 0.01$. Consequently, the following equation must hold:

$$DI_O^0 \geqslant \frac{N_N}{100 \cdot N_C \cdot c_O^N \cdot m_O^N \cdot v} = \rho. \tag{13}$$

As a consequence, the number of infected individuals necessary to extend the epidemic out to the cell depends on the values of the parameters c_O^N, m_O^N and v. In Table 1 some examples are shown for the case in which the population is the same in all cells.

On the other hand, if

$$DI_O^0 < \rho, \tag{14}$$

then the evolution of the infected population is restricted to the main cell O, and the number of infected individuals becomes zero if $I_O^t < I_O^{t-1}$ for every t. Consequently, as Eq. (14) holds then a simple calculus shows that for every t:

$$I_O^t = (1-\varepsilon) \cdot I_O^{t-1} + v \cdot S_O^{t-1} \cdot I_O^{t-1} < I_O^{t-1}, \tag{15}$$

if and only if $S_O^{t-1} < \varepsilon/v$. As a consequence, $I_O^t = I_O(t)$ is a decreasing function which tends to 0 if $S_O^0 < \varepsilon/v$, or equivalently, if

Table 1
Minimum values of infected individuals located at the cell O at time $t = 0$ necessary to produce the epidemic spreading to another cells

Connection factor	Movement factor	Virulence	DI_O^0
$c = 1$	$m = 1$	$v = 1$	0.01
		$v = 0.6$	0.02
		$v = 0.3$	0.03
	$m = 0.6$	$v = 1$	0.02
		$v = 0.6$	0.03
		$v = 0.3$	0.06
	$m = 0.3$	$v = 1$	0.03
		$v = 0.6$	0.06
		$v = 0.3$	0.11
$c = 0.6$	$m = 1$	$v = 1$	0.02
		$v = 0.6$	0.03
		$v = 0.3$	0.06
	$m = 0.6$	$v = 1$	0.03
		$v = 0.6$	0.05
		$v = 0.3$	0.09
	$m = 0.3$	$v = 1$	0.06
		$v = 0.6$	0.09
		$v = 0.3$	0.19
$c = 0.3$	$m = 1$	$v = 1$	0.03
		$v = 0.6$	0.06
		$v = 0.3$	0.11
	$m = 0.6$	$v = 1$	0.06
		$v = 0.6$	0.09
		$v = 0.3$	0.19
	$m = 0.3$	$v = 1$	0.11
		$v = 0.6$	0.19
		$v = 0.3$	0.37

$$\Omega = \frac{vS_O^0}{\varepsilon} < 1, \tag{16}$$

where Ω is the threshold quantity called the basic reproductive number.

Furtheremore, if Eq. (16) does not hold, then the infected population of the cell increases and when it exceeds the constant ρ the epidemic will be spread from the main cell to its neighbour cells.

4. Simulations

The cellular space in the next simulations will be formed by a two-dimensional array of 50×50 cells. In the simulations we represent the proportion of infected individuals of each cell by means of a gray level code is used running from white color for the state 0 to black color for state 1. For the sake of simplicity, we will use the following artificially chosen parameters: $v = 0.6$, $\varepsilon = 0.4$, $m_{\alpha\beta}^{(i,j)} = 0.5$, for every cell (i,j). The initial conditions consist of only one cell with infected individuals, namely $(25, 25)$ with $s_{25,25}^0 = (0.7, 0.3, 0)$. Moreover, in the simulations, six configurations of the CA are shown: Those at time steps $t = 0, 5, 10, 15, 20, 25$.

We will consider two cases: (1) Each cell is connected with all of its neighborhoods with the same parameter: $c_{\alpha\beta}^{(i,j)} = 1$ for every cell (i,j), and $(\alpha, \beta) \in V$; (2) The connection between the cells is not constant.

(1) Suppose that the population in each cell is the same, that is, $N_{ij} = 100$ for every cell (i,j). Then, the simulation obtained with Von Neumann neighborhoods is shown in Fig. 2 and the simulation computed with Moore neighborhoods is shown in Fig. 3. Note that the successive epidemic fronts (regions of spread at different times) are circular as is expected, where the starting point of the epidemic is in the center of these circular fronts. The evolutions of the number of susceptible, infected and recovered individuals are shown in Fig. 4. Initially, only the central cell has infected individuals, specifically the 30% of population (30 persons). As is shown in Fig. 4, the number of infected individuals increases from $t = 1$ to $t = 44$ with Von Neumann

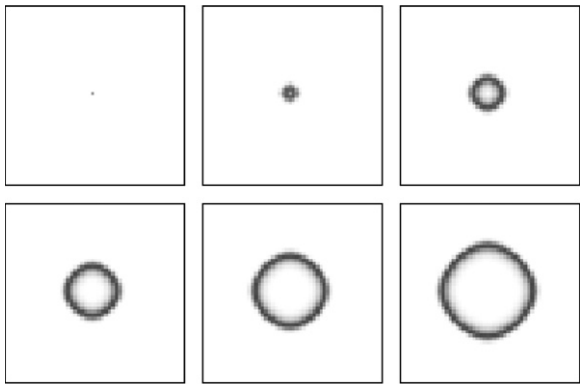

Fig. 2. Simulations with constant connection factors and Von Neumann neighborhoods.

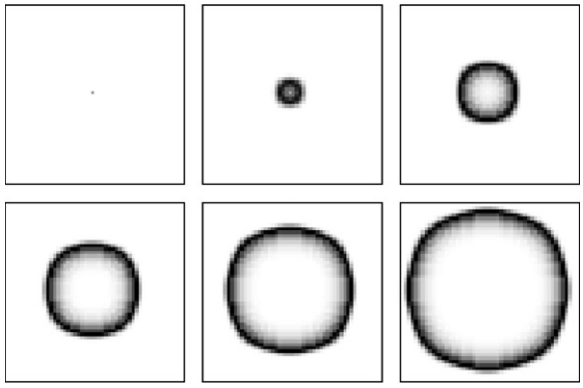

Fig. 3. Simulations with constant connection factors and Moore neighborhoods.

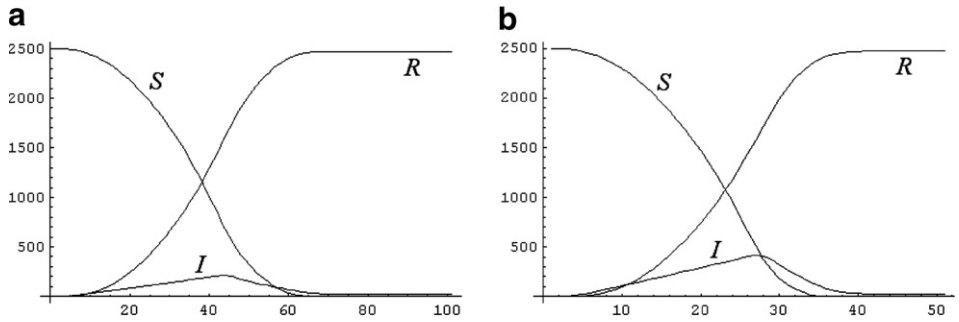

Fig. 4. Evolution of the susceptible, infected and recovered individuals for Von Neumman neighbourhoods (a), and Moore neighbourhoods (b).

neighbourhoods and from $t = 1$ to $t = 27$ with Moore Neighborhoods. Furthermore, the number of susceptible individuals decreases as the number of recovered individuals increases.

On the other hand, suppose that the population is not constant in all the cells according to $N_{ij} = e^j$. Note that in this case most of the population is concentrated in the eastern cells and it decreases uniformly to the western cells. Then the evolution of the CA with Moore neighborhoods is shown in Fig. 5. In this case, note that the epidemic will rapidly propagate through the western cells and the maximum values of the states of the cells are obtained, precisely, in these western cells given by higher gray level colors.

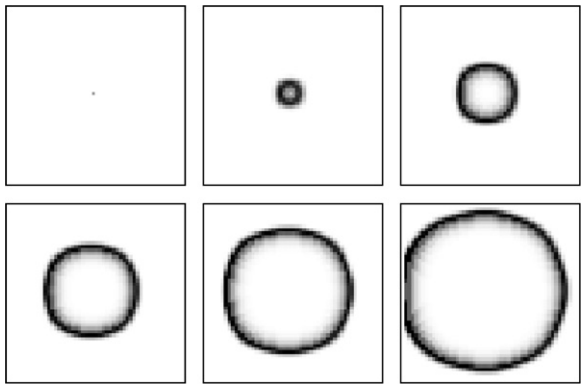

Fig. 5. Simulations with constant connection factors, Moore neighbourhoods and inhomogeneous distribution of the population.

(2) In the second case we assume that there is not constant connections between each cell and its neighborhoods. Furthermore, for the sake of simplicity, we suppose that the cellular space is divided into four artificial areas with different connection factors as follows:

$$\text{Area I}: C_1 = \{(i,j) \in C:\ 1 \leqslant i \leqslant 25,\ 1 \leqslant j \leqslant 25\};\ \text{if}\ (i,j) \in C_1\ \text{then}\ c_{\alpha\beta}^{(i,j)} = 0.6.$$

$$\text{Area II}: C_2 = \{(i,j) \in C:\ 1 \leqslant i \leqslant 25,\ 26 \leqslant j \leqslant 50\};\ \text{if}\ (i,j) \in C_2\ \text{then}\ c_{\alpha\beta}^{(i,j)} = 1.$$

$$\text{Area III}: C_3 = \{(i,j) \in C:\ 26 \leqslant i \leqslant 50,\ 1 \leqslant j \leqslant 25\};\ \text{if}\ (i,j) \in C_3\ \text{then}\ c_{\alpha\beta}^{(i,j)} = 0.$$

$$\text{Area IV}: C_4 = \{(i,j) \in C:\ 26 \leqslant i \leqslant 50,\ 26 \leqslant j \leqslant 50\};\ \text{if}\ (i,j) \in C_4\ \text{then}\ c_{\alpha\beta}^{(i,j)} = 0.3.$$

Then, the simulations obtained are shown in Fig. 6. Note that the epidemic disease does not spread through area C_3 as the connection factor is 0. Moreover, the greater speed of the spreading is obtained in area C_2 as the connection factor is equal to 1. In the other areas, C_1 and C_4, the rate speed of the epidemic spreading is, obviously, slower.

Finally, if the population depends on the cell considered (taking into account the formula stated above), the epidemic spreading is modeled as in Fig. 7. Note that the infected population grows rapidly in the west of the cellular space.

Also, the effect of population vaccination can be considered in this model. In this case, a vaccination parameter, $\omega \in [0,1]$, must be considered in the local transition functions of the model. Such parameter stands for the portion of susceptible infected individuals at each time step which are vaccinated. Consequently, we have:

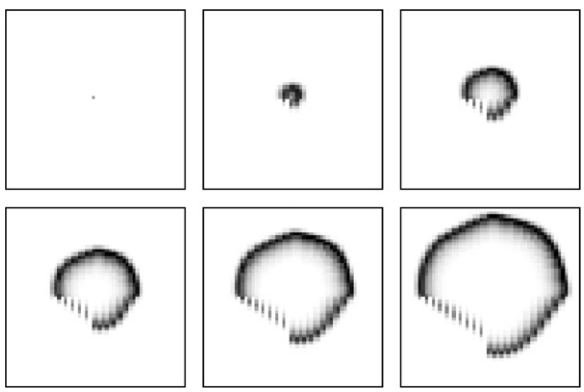

Fig. 6. Simulations with non constant connection factors and Moore neighbourhoods and homogeneous distribution of the population.

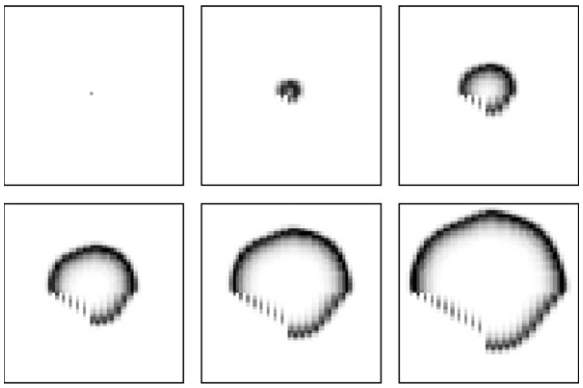

Fig. 7. Simulations with non constant connection factors and Moore neighbourhoods and inhomogeneous distribution of the population.

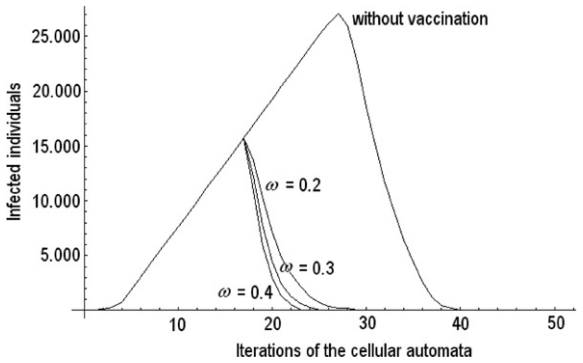

Fig. 8. Evolution of the infected population with different vaccination rates.

$$I_{ij}^{t} = (1-\varepsilon)\cdot I_{ij}^{t-1} + v\cdot S_{ij}^{t-1}\cdot I_{ij}^{t-1} + S_{ij}^{t-1}\cdot \sum_{(\alpha,\beta)\in V^{*}} \frac{N_{i+\alpha,j+\beta}}{N_{ij}}\cdot \mu_{\alpha\beta}^{(i,j)}\cdot I_{i+\alpha,j+\beta}^{t-1}, \tag{17}$$

$$S_{ij}^{t} = S_{ij}^{t-1} - \omega\cdot S_{ij}^{t-1} - v\cdot S_{ij}^{t-1}\cdot I_{ij}^{t-1} - S_{ij}^{t-1}\cdot \sum_{(\alpha,\beta)\in V^{*}} \frac{N_{i+\alpha,j+\beta}}{N_{ij}}\cdot \mu_{\alpha\beta}^{(i,j)}\cdot I_{i+\alpha,j+\beta}^{t-1}, \tag{18}$$

$$R_{ij}^{t} = R_{ij}^{t-1} + \varepsilon\cdot I_{ij}^{t-1} + \omega\cdot S_{ij}^{t-1}. \tag{19}$$

Finally, in Fig. 8 the evolution of infected individuals is shown when the vaccination process is considered. We suppose that the initial configuration is formed by only one cell with infected individuals: the cell $(25,25)$, with $s_{25,25}^{0} = (0.7, 0.3, 0)$. Moreover $v = 0.6$, $\varepsilon = 0.6$, $m_{\alpha\beta}^{(i,j)} = 0.5$, $c_{\alpha\beta}^{(i,j)} = 1$ for every cell (i,j). Four different values of the vaccination rate are considered: $\omega = 0, 0.2, 0.3, 0.4$ and the vaccination process affects to the susceptible individuals of all cells starting at $t = 16$. Note that as ω increases, the number of infected individual decreases.

5. Conclusions

In this work a theoretical 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 population is divided into three classes: susceptible, infected and recovered individuals, consequently, the proposed model can be considered as a SIR-type model. Its main features are the following:

- The total amount of population in the cellular space is constant. Nevertheless, it can not be uniformly distributed between the cells.

- The local transition function is very simple and several epidemiological and environmental parameters are involved.
- The vaccination effect is considered.

The main characteristic of this model is the definition of the state of each cell as a three-uplet formed by a suitable discretization portion of its population which is susceptible, infected and recovered at each time step, together with the definition of the local transition function involving these parameters.

The simulations obtained using artificially chosen parameters seem to be in agreement with the expected behaviour of a real epidemic.

The proposed model can serve as a basis for the development of another algorithms to simulate real epidemics. Consequently, further work aimed at testing its performance against real data. Obviously, in real simulations one has to take care with the scale and an appropriate size of the cells must be used in order to obtain an efficient simulation.

Unfortunately, the proposed model presents some shortcomings, e.g.: small world effect (non local effect) which are crucial to model SARS, Foot and mouth disease and Aviation Flu, and seasonality effects which are crucial to model measles.

Acknowledgements

This work has been partially supported by the Consejería de Sanidad (Junta de Castilla y León, Spain) under grant SAN/1052/SA29/05, and by "D. Samuel Solórzano Barruso" Memorial Foundation (University of Salamanca, Spain) under grant FS/3-2005.

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