Applied Mathematical Sciences, Vol. 3, 2009, no. 20, 959 - 968

Using Cellular Automata to Simulate Epidemic Diseases

S. Hoya White

Department of Applied Mathematics
Universidad de Salamanca
sarahw@usal.es

A. Martín del Rey

Department of Applied Mathematics
Universidad de Salamanca
delrey@usal.es

G. Rodríguez Sánchez

Department of Applied Mathematics
Universidad de Salamanca
gerardo@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 behavior of epidemic spreading.

Mathematics Subject Classification: 68Q80, 92D30

Keywords: Cellular automata, epidemic spreading, SIS model

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 many individuals in an area

at the same time. In this way, the most recent worrying epidemic was the Severe Acute Respiratoy Syndrome (SARS) outbreak in Asia. Consequently, since the publication of the first mathematical epidemic models (see [2, 5]), several mathematical models to study the dynamics of epidemics have been apperared in the literature. Many of them are based on differential equations, which neglect spatial aspects of the epidemic process. 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 [4]). Other mathematical models are based on discrete systems: stochastic interacting particle models, cellular automata models, etc. (see, for example [1, 3, 6, 7]). These simple models eliminate the last mentioned shortcomings, and are specially suitable for computer simulations.

The main goal of this work is to introduce a new cellular automaton model to simulate the spread of a general epidemic. As is mentioned above, cellular automata (CA for short) are simple models of computation capable to simulate complex physical, biological or environmental phenomena (see [8]). Specifically, a two-dimensional CA is formed by a two-dimensional array of identical objects called cells, which are endowed with a state that change 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) determines how local interactions can influence the global behaviour of the system.

The rest of the paper is organized as follows: In Section 2 a review of bidimensional 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 An overview on 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 rule, called local transtition 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 (see Figure 1-(a)): $C = \{(a, b), 1 \leq a \leq r, 1 \leq b \leq c\}$.

The state of each cell is an element of a finite state set, S, in such a way that the state of the cell (a, b) at time t is denoted by $s_{ab}^{(t)} \in S$. The matrix $C^{(t)} = \left(s_{ij}^{(t)}\right)$ is called configuration of the CA at time t. Moreover, $C^{(0)}$ is the initial configuration of the CA.

The neighborhood of a cell (a, b) is the set of cells whose states at time t determine the state of the cell (a, b) at time $t + 1$, by means of the local

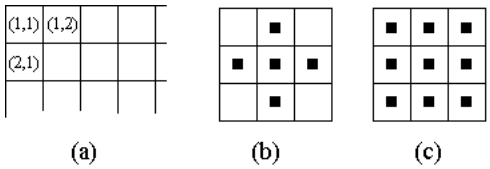

Figure 1: (a) Cellular space (b) Von Neuman neighborhood (c) Moore neighborhood

transition function. Depending on the process to be modelled, one can choose an appropiate neighborhood. Nevertheless, the traditional neighborhoods considered are the Von Neumann neighborhood (see Figure 1-(b)), and the Moore neighborhood (see Figure 1-(c)). Note that the main cell is also considered in its neighborhood. A neighborhood is defined by means of a finite set of indices $V = \{(\alpha_i, \beta_i), 1 \leq i \leq m\} \subset \mathbb{Z} \times \mathbb{Z}$, such that for every cell (a, b), its neighborhood, $V_{(a,b)}$, is the set of m cells given by

$$V_{(a,b)} = \{(a + \alpha_1, b + \beta_1), \ldots, (a + \alpha_m, b + \beta_m) : (\alpha_k, \beta_k) \in V\}.$$

Note that for Moore neighborhood, we have

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

Moreover, we will denote by V^* the set of indices defining the neighborhood of a cell (a, b) in which the main cell is not considered, that is, $V^* = V - \{(0,0)\}$.

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

$$s_{ab}^{(t+1)} = f\left(s_{a+\alpha_1,b+\beta_1}^{(t)}, \ldots, s_{a+\alpha_m,b+\beta_m}^{(t)}\right).$$

As the cellular space is considered to be finite, boundary conditions must be taken into account in order to assure the well-defined dynamics of the CA. Several boundary conditions can be chosen depending on the phenomenon to be simulated. In this work, we will consider null boundary conditions, that is: $s_{ab}^{(t)} = 0$ if $a < 1$ or $a > r$, or if $b < 1$ or $b > c$.

A very important type of CA, suitable for simulating some ecological and biological systems, are those whose local transition functions are as follows:

$$s_{ab}^{(t+1)} = g\left(\sum_{(\alpha,\beta)\in V} \mu_{\alpha\beta}^{(a,b)} s_{a+\alpha,b+\beta}^{(t)}\right), \tag{1}$$

where $g\colon \mathbb{R} \to S$ is a suitable discretization function, and $\mu_{\alpha\beta}^{(a,b)} \in \mathbb{R}$ are the specific parameters of the system to be simulated.

3 The model

The proposed model is based on the use of a two-dimensional cellular endowed with a local transition function of the form (1). Each cell stands for a square area of the land in which the epidemic is spreading. Moreover, it is assumed that the population distribution is homogeneous, *i.e.*, all cells have the same population at every step of time.

The state of each cell at each time step is obtained from the fraction of the number of individuals of the cell which are infected by the epidemic, that is, $s_{ab}^{(t)}$ is a suitable discretization of

$$\frac{\text{infected population of } (a,b)}{\text{total population of } (a,b)}. \tag{2}$$

At time $t = 0$, the last expression is exactly the state for each cell. Consequently, $s_{ab}^{(t)} \in [0,1]$ for every t. As $s_{ab}^{(t)}$ is a real number and the state set is finite, we must discretize such value in order to obtain an element of S. In this work a state set with 11 elements will be considered:

$$S = \{s_0 = 0, s_1 = 0.1, \ldots, s_9 = 0.9, s_{10} = 1\}. \tag{3}$$

Furthermore, it is supposed that the state of each cell at a particular time step depends on the states of its eight nearest cells and the cell itself at the previous time step. Hence, the neighborhood considered is the Moore neighborhood.

Taking into account the definition of S, we will consider the following discretization function:

$$g\colon \mathbb{R} \to S, \quad x \mapsto g(x) = \begin{cases} 0, & \text{if } x < 0 \\ \frac{[10x]}{10}, & \text{if } 0 \le x \le 1 \\ 1, & \text{if } x > 1 \end{cases} \tag{4}$$

where $[x]$ stands for the $round\,(x)$ function.

These states change according to the following local transition function:

$$s_{ab}^{(t+1)} = g\left(\left(1 - P(t)\right) s_{ab}^{(t)} + \left(1 - s_{ab}^{(t)}\right) \left[\varepsilon s_{ab}^{(t)} + \sum_{(\alpha,\beta)\in V^*} \mu_{\alpha\beta}^{(a,b)} s_{a+\alpha,b+\beta}^{(t)} \right] \right),$$

where g is the discretization function given in (4).

The function $P(t)$ stands for the recovering process of the infected cells, *i.e.*, it measures the population of the cell that has recovered from the disease after

a time step. The explicit expression of the function $P(t)$ must be determined according to the epidemic to be modeled. For the sake of simplicity, in our work we suppose that this function is a polynomial of degree n.

Moreover, the real parameters ε and $\mu_{\alpha\beta}^{(a,b)}$ represent the main characteristics of the epidemic and the environment. Obviously, ε and $\mu_{\alpha\beta}^{(a,b)}$ are determined by the epidemic to be modeled.

Specifically, the real parameter $\mu_{\alpha\beta}^{(a,b)}$ involves three factors: The connection factor, $c_{\alpha\beta}^{(a,b)}$, the movement factor, $m_{\alpha\beta}^{(a,b)}$, between the cell (a,b) and its neighbor cell $(a+\alpha, b+\beta)$, and the virulence of the epidemic, $v \in [0,1]$. As a consequence, $\mu_{\alpha\beta}^{(a,b)} = c_{\alpha\beta}^{(a,b)} \cdot m_{\alpha\beta}^{(a,b)} \cdot v$, for every cell (a,b) and every $(\alpha,\beta) \in V^*$.

As is mentioned above, it is suppose that the way on infection is the contact between two individuals. Then, the non-infected individuals located at the cell (a,b) can be infected by the infected individuals of the main cell (a,b), or by the infected individuals located at a neighbor cell, $(a+\alpha, b+\beta)$, that have travelled to the cell (a,b).

In the first situation, that is, when all individuals considered belong to the cell (a,b), the infection process is given by the parameter $\varepsilon \in [0,1]$, which represents the portion the non-infected individuals of the cell at time t infected by the infected population of the cell at the same time.

In the second situation, that is, when the non-infected individuals of (a,b) are infected by the infected individuals of the neighbor cells, some type of connection (by airplane, by train, by car, etc.) between two neighbor cells must be considered in order to permit the epidemic propagation from a neighbor cell to the main cell. This connection is given by the coefficients $c_{\alpha\beta}^{(a,b)}$, such that if there is some connection between $(a+\alpha, b+\beta)$ and (a,b), then $c_{\alpha\beta}^{(a,b)} = 1$, and if there is not connection between these two cells, then $c_{\alpha\beta}^{(a,b)} = 0$.

The parameter $m_{\alpha\beta}^{(a,b)}$ gives the probability of an infected individual belongs to the cell $(a+\alpha, b+\beta)$ to be moved to the cell (a,b). As a consequence, $m_{\alpha\beta}^{(a,b)} \in [0,1]$.

4 Simulations

The cellular space in the next simulations will be formed by a two-dimensional array of 40×40 cells and, as is mentioned above the state set is formed by 11 elements (see (3)). To represent the state of each cell, a color code is used and it is shown in Figure 2. Moreover, we also suppose that $\varepsilon = 0.4$, $m_{\alpha\beta}^{(a,b)} = 0.4$ for all (a,b) and $(\alpha,\beta) \in V_{(a,b)}$, $v = 0.4$, and $P(t) = 0.2t + 0.2$. Note that for the sake of simplicity, these parameters are artificially chosen.

Moreover, the size of the time step must be considered according to the main characteristic of the epidemic and the environment.

Figure 2: Color codes

First of all, let us consider the case in which each cell is connected with all of its neighbor cells, that is $c_{\alpha\beta}^{(a,b)} = 1$ for all (a,b) and $(\alpha,\beta) \in V^*$. If the initial configuration is formed by all cells with state 0 except for three cells which are infected as follows: $s_{10,10}^{(0)} = 0.8, s_{20,20}^{(0)} = 1, s_{30,30}^{(0)} = 0.5$, then, the evolution of the epidemic spreading obtained from the CA is shown in Figure 3 (the evolution goes from left to right, and from top to botton). Note that in this case, after 11 iterations, the epidemic disappeared.

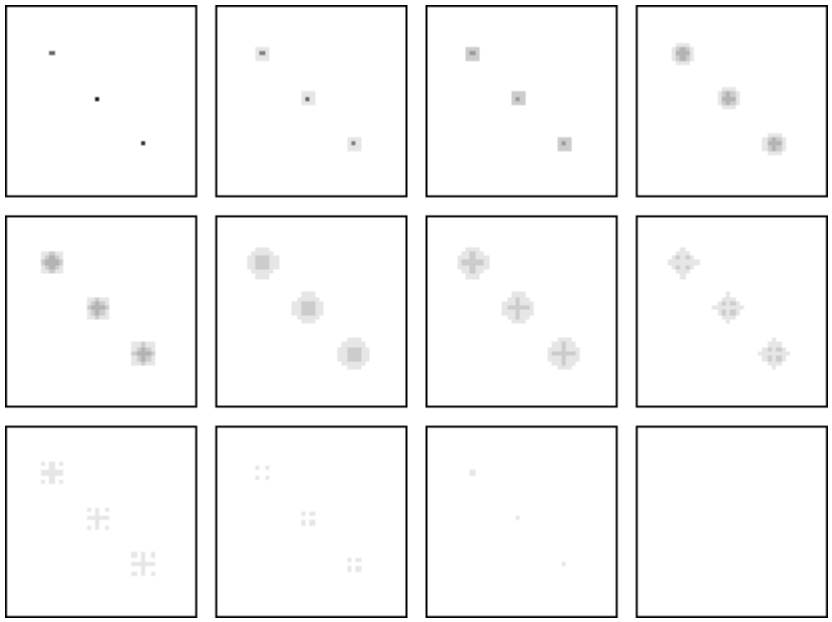

Figure 3: Simulation with all neighbor cells connected

Furthermore, suppose that the state of each cell at time $t = 0$ is randomly chosen, then the evolution is shown in Figure 4. In this case, the epidemic disappeared after 20 iterations. Moreover, the frequency of the different states in the evolution of the CA is shown in the following table, and the percentage of infected population evolves as is shown in Figure 5.

On the other hand, let us suppose that the conections between the cells are given by the following graph (see Figure 6), where each vertix stands for a cell and each edge between two vertices stands for a conection between these two cells. Note that in this case, the cellular space is of 13×13. If the parameters

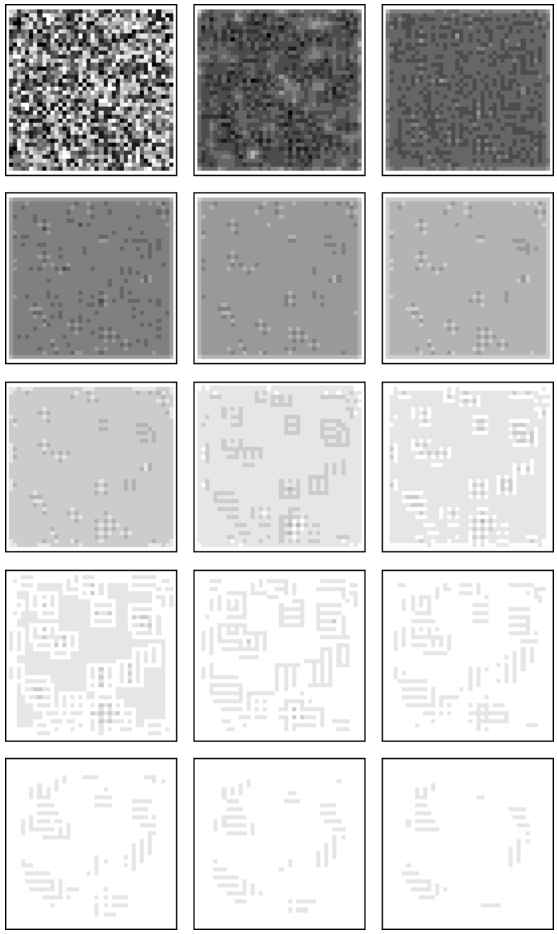

Figure 4: Simulation with a random initial configuration: Configurations $C^{(0)}$–$C^{(14)}$

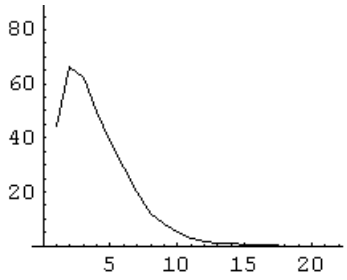

Figure 5: Evolution of the percentage of infected population

of the CA are $P(t) = 0.1t + 0.1$, $\varepsilon = 0.4$, $m_{\alpha\beta}^{(a,b)} = 0.8$, $\forall (a,b), \forall (\alpha,\beta) \in V^*$ and $v = 0.6$, then the evolution of the epidemic spreading is given in Figure 7.

t	s_0	s_1	s_2	s_3	s_4	s_5	s_6	s_7	s_8	s_9	s_{10}
0	82	174	156	164	184	150	151	143	166	151	79
1	0	0	1	3	27	105	557	628	238	37	4
2	0	0	0	1	7	171	933	488	0	0	0
3	0	0	0	4	205	1251	136	4	0	0	0
4	0	0	4	197	1329	70	0	0	0	0	0
5	0	0	209	1334	57	0	0	0	0	0	0
6	0	77	1444	79	0	0	0	0	0	0	0
7	27	1316	253	4	0	0	0	0	0	0	0
8	349	1186	64	1	0	0	0	0	0	0	0
9	762	806	32	0	0	0	0	0	0	0	0
10	1203	392	5	0	0	0	0	0	0	0	0
11	1336	264	0	0	0	0	0	0	0	0	0
12	1424	176	0	0	0	0	0	0	0	0	0
13	1477	123	0	0	0	0	0	0	0	0	0
14	1516	84	0	0	0	0	0	0	0	0	0
15	1550	50	0	0	0	0	0	0	0	0	0
16	1572	28	0	0	0	0	0	0	0	0	0
17	1586	14	0	0	0	0	0	0	0	0	0
18	1593	7	0	0	0	0	0	0	0	0	0
19	1598	2	0	0	0	0	0	0	0	0	0
20	1600	0	0	0	0	0	0	0	0	0	0

Table 1: Frequency of the states

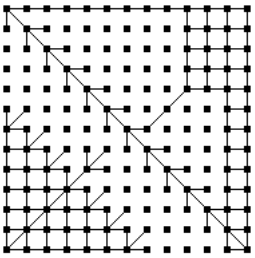

Figure 6: Connections between the cells

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. 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 (that is, a non-homogeneous population environment), or the effect of vaccination of the population, virus mutation, etc. Furthermore, it is also interesting to consider non-constant connections factors.

ACKNOWLEDGEMENTS. This work has been supported by the Consejería de Sanidad of Junta de Castilla y León (Spain) under grant SAN673 / SA23 / 08.

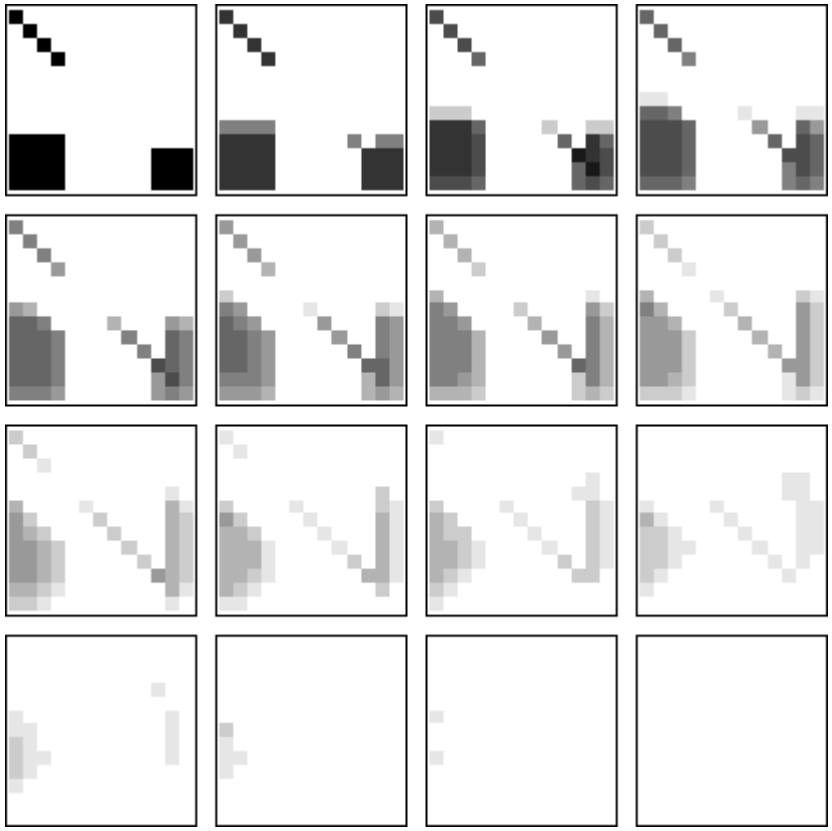

Figure 7: Simulation with variable connections

References

[1] C. Beauchemin, J. Samuel and J. Tuszynski, A simple cellular automaton model for influenza A viral infections, *J. Theor. Biol.,* **232** (2005), 223 - 234.

[2] W. O. Kermack and A. G. McKendrick, Contributions to the mathematical theory of epidemics, part I, *Proc. R. Soc. Edin. A,* **115** (1927), 700 - 721.

[3] M. L. Martins, G. Ceotto, S. G. Alves, C. C. B. Bufon, J. M. Silva and F.,F. Laranjeira, A cellular automata model for citrus variegated chlorosis, *Physica A,* **295** (2001), 42 - 48.

[4] D. Molisson, The dependence of epidemic and population velocities on basic parameters, *Math. Biosci.,* **107** (1991), 255-287.

[5] R. Ross, *The prevention of malaria,* 2nd edition, Murray, London, 1911.

[6] J. Satsuma, R. Willox, A. Ramani, B. Grammaticos and A. S. Carstea, Extending the SIR epidemic model, *Physica A,* **336** (2004), 369–375.

[7] 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), 209-223.

[8] T. Toffoli and N. Margolus, *Cellular Automata Machines. A New Environment for Modeling,* MIT Press, Cambridge, MA, 1987.

Received: November, 2008