Deterministic Site Exchange Cellular Automata Models for the Spread of Diseases in Human Settlements

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A cellular automata model that describes as limit cases the spread of contagious diseases modeled by systems of ordinary or partial differential equations is developed. Realistic assumptions in the motion of human populations are considered. A parameter describing the range of that motion is defined. For small (large) values of this parameter, the behavior described by partial (ordinary) differential equation models are reproduced. Emphasis is also placed on the study of those scenarios which the differential equations fail to describe. In the study of these cases some interesting results, including evidence of period doubling behavior, are reported.

1. Introduction

One of the first tasks that faces a theoretician wanting to interpret the time evolution of a complex system is the construction of a model. In real systems, many features are likely to be relevant, but not all of them, however, are included in the model. In spite of this lack, such a simplified description can often be very helpful in developing the necessary intuition for understanding the behavior of real complex systems.

Most models in population dynamics, spread of diseases, rumors, and news are formulated in terms of differential equations, both partial and ordinary. The difference between the types of equations used is strongly related with the assumptions made about the way members of a population move in the spatial region they belong to. If the model assumes a homogeneous mixing of the different classes of individuals

(infected, susceptible, removed, etc.) then, systems of ordinary differential equations are used. If a short-range character of motion is assumed, that is, if the mean length of the motion of members of a population is small with respect to the size of the spatial ensemble where they live, then the spread of some diseases behave as diffusion processes and partial differential equations are used in their modeling. In both cases the assumption of random motion is made for the individuals. This last hypothesis is irreplaceable in the deduction of the equations.

Two objections could be made about these assumptions. First, the diffusive or perfect mixing hypothesis is not quite always fulfilled. In many cities the length of the average daily motion of habitants is too large to produce diffusive behavior and at the same time too small to guarantee perfect mixing. Second, the motion of individuals of some species (human among them) is not random as assumed in the hypothesis of the above mentioned models. At least for human populations it is far from true. A lot of people go to school or to work and later go back home daily, therefore periodic motion seems more praiseworthy than random. Many other species (such as foxes and other mammalians) also have motion routines. Finally, we want to stress that little has been said about those transmission processes that do not fit very well into the mentioned differential models. We mainly address that topic in this work.

The aim of this paper is to develop a cellular automata model, general enough to describe those limit situations well-modeled by systems of ordinary or partial differential equations and also those that the differential models fail to describe, taking into account the peculiarities of motion in human settlements and the main characteristics of contact processes. We also report some interesting properties of the infectives time series in different regimes of the parameters. The model is described in section 2. It depends on two parameters: the mean length of motion path of individuals, λ; and a measure of the strength of the contagion process, p. Tuning the parameter λ we obtain diffusive or perfect mixed behaviors and also describe those scenarios hard to characterize with differential equations. After a validation of the behavior of our model in these limit cases (section 3), we focus our attention on those intermediate scenarios where differential models cannot be used (section 4). In this regime we obtain the central results of our work, studying some properties of the spread of epidemics related with the time series of infectives. Section 5 follows with conclusions.

2. The model

Cellular automata provide simple models for a variety of complex systems containing a large number of identical elements with local interactions. A cellular automaton consists of a lattice with a discrete variable

at each site, evolving at discrete time steps. At a given time, the value of the variable at one site is determined by the values of the variables at the neighboring sites. The neighborhood could include the site itself. The evolution rule is synchronous, that is, all sites are updated simultaneously. Cellular automata are therefore, discrete (in space and time) dynamical systems. For a review of their main properties see, for example, [6, 10, 12].

Site exchange cellular automata are automata networks whose rules consist of two subrules. The first, applied synchronously, is a local rule inspired by Conway's "Game of Life" and describes the local behavior of the transmission processes (contagion process, spread of news and rumors). We call these types of rules *contagion rules* although their validity is far beyond the scope of infective processes. For example, some processes of the diffusion of rumors can be described using these rules. The second, which has been sequentially applied, describes the motion of a fraction of individuals and are called *transport rules*. These models have been extensively studied [1–5] in recent years. In order to enhance the difference between those works and ours we briefly describe them below.

Let Z be the set of the integer numbers and $\Lambda \subseteq Z^2$ be a lattice. The set Λ represents the spatial environment where the population lives. At a time step t a site of Λ is either empty or occupied (representing an individual in some subclass of the population). The way the transport subrules have been used [1–5] is as follows: Each time step, an occupied site is selected at random and swapped with another site (empty or occupied) also selected at random. This operation is repeated $mc(t)N$ times, where N is the total number of sites, $c(t)$ is the density of nonzero sites at time t, and m is a parameter called the *degree of mixing*. It is important to note the stochastic character of the processes. We also want to stress their unrealistic character. These rules fail to explain why in a human population, with rigid motion schedules dictated by daily routine, the spread of epidemics behave under certain conditions as perfect mixed or perfect diffusive.

In order to fill this gap we devise another type of transport subrule. Let $\Lambda \subseteq Z^2$, whose vertices are occupied by members of the population. Let $\Omega = \{0, 1, \ldots, p\}^{\Lambda}$ be the set of elements of the form $(a_{(i,j)})_{(i,j)\in\Lambda}$ where $a \in \{0, 1, \ldots, p\}$ represents to which subclass they belong, and (i, j) is a position in the lattice. For example, in an epidemic process these subclasses could be empty, susceptible, infective, or removed. The set Ω contains all the possible configurations over the lattice Λ. Hence we call it the *configuration space*.

Let $\tau : \Omega \to \Omega$ be a function which satisfies the following conditions.

(a) Let $(\tau(a_{(i,j)}))_{(i,j)\in\Lambda}$ be the image of the element $(a_{(i,j)})_{(i,j)\in\Lambda}$ under the application of τ. Then if $a_{(i_0,j_0)} \neq 0$, one of the following two conditions hold.

(a1) $\tau(a_{(i_0,j_0)}) = 0$.

(a2) $\tau(a_{(i_0,j_0)}) = a_{(i_0,j_0)}$.

(b) For every $x_1, x_2 \in \Lambda$, with $x_1 \neq x_2$ such that $a_{x_1}, a_{x_2} \neq 0$, then $\tau(a_{x_1}) \neq \tau(a_{x_2})$.

The above statements deserve an explanation. Condition (a) means that every nonempty element of the lattice can only be moved to an empty site (a1) or remain in the original position (a2). It means that we are considering the possibility that some members of the population do not change their position with time. This is a very natural assumption. In fact, a fraction of all members in a human settlement (old men and women, housekeepers, etc.) do not change their spatial position with time.

Condition (b) means that two occupied sites cannot go to the same empty site. These are reasonable statements. We observe in nature that elements of a population, by means of motion, could be placed close together but never one over the other.

We call the function τ a *transport rule*. We emphasize the synchronous character of this type of rule. In Figure 1 we show a schematic representation of a transport rule.

Let $X \in \Omega$ be an element of Ω. We denote by $O(X)$ the subset of Λ with nonempty positions, that is, $O(X) = \{x \in \Lambda, a_x \neq 0\}$, and by $N(X)$ the number of elements of $O(X)$. We define the number

$$\lambda = \frac{1}{N(X)} \sum_{x \in O(X)} \rho(x, \tau(x)) \tag{1}$$

where $\rho(x, \tau(x))$ is the euclidean distance between the nonempty position x and its destination by means of τ. We call λ the *mean length of motion path* of individuals. Note that once a transport rule τ is selected, the value of λ is calculated straightforwardly. Further, different transporta-

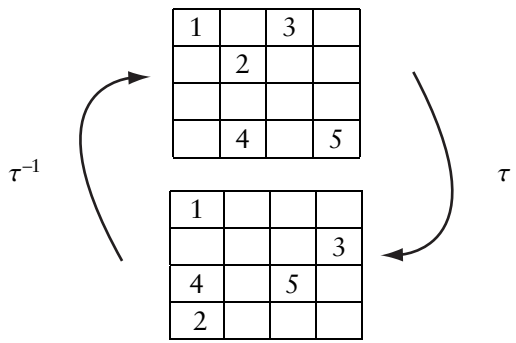


Figure 1. A schematic transport rule. Notice that some sites remain unchanged.

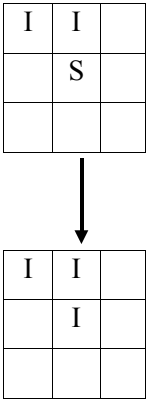


Figure 2. A schematic contagion rule.

tion rules should have the same λ. We only use transport rules with uniform probability distribution functions for distances, that is,

$$\lambda \approx \frac{d_{\min} + d_{\max}}{2}$$

where $d_{\min}, d_{\max}$ are the minimum and maximum distances allowed, respectively. Obviously $d_{\min} = 0$. Transportation rules having different probability distribution functions for distances are also important and interesting. In fact, in human settlements people are averse to move far away from their home (sometimes it is impossible to avoid) to develop their social routines. However, in this work we only focus on the above-mentioned transportation rules.

Let us now define the kind of local rules which describe the transmission process (e.g., contagion, spread of rumors, etc.). Although we use concepts related with epidemic processes, the validity of the definitions explored below are far beyond this narrow framework.

Let us take $\Omega = \{0, 1, 2\}^{\Lambda}$, where 0 means empty, 1 susceptible, and 2 infective. Nonempty susceptible sites become infective by contact, that is, a susceptible may become infective with a probability p if and only if it is in the neighborhood of an infective. More precisely, during one time step, the probability that a susceptible having n infected neighbors become infected is $1 - (1 - p)^n$. This hypothesis neglects latent periods, that is, an infected susceptible becomes immediately infective. In Figure 2 we show a situation as described.

Let $\epsilon_p : \Omega \to \Omega$ be the function (which depends on probability p) representing the above mentioned transmission process. We call the *contagion process* the time trajectory of an element $X \in \Omega$ by the application of $\pi = \tau^{-1} \circ \epsilon_p \circ \tau \circ \epsilon_p$:

$$\{\pi_n\}_{n \in N} = \{\pi^n(X)\}_{n \in N}. \tag{2}$$

We also consider along this line a more general situation, in which the disease has a finite duration, that is, a susceptible, which becomes infective at time t, again becomes susceptible at time $t + d$. Then, we could define a function $\epsilon_p^d : \Omega \to \Omega$ representing this transmission process. Hence, a function $\pi^d = \tau^{-1} \circ \epsilon_p^d \circ \tau \circ \epsilon_p^d$ could be defined and also a contagion process similar to that described in equation (2). Note also that $\lim_{d\to\infty} \epsilon_p^d - \epsilon_p$.

Although this set up seems completely deterministic, it has a stochastic component. The function ϵ^p[1] could assign *in each realization* different elements $\epsilon_p(X) \in \Omega$ to every element $X \in \Omega$. Hence, equation (2) should be understood as the set of realizations of the contagion process. The configuration X is the initial conditions of the process and encloses all the information at time $t = 0$. Models in ordinary or partial differential equations also use some information encoded in X in the form of initial and/or boundary conditions. Note that the application of π represents the daily exposure of the elements of the population. They could be infected or not at their original sites (ϵ_p), later they move (τ) to their destination, being exposed or transmitting the disease there (ϵ_p), and later go back to their original positions (τ^{-1}). We oversimplify the process by assuming that the only situations where contagion takes place are at their original position or destination, that is, homes, schools, jobs, or any other social activities for human populations. In this case, exposure to epidemics during transportation (e.g., subway, bus, etc.) is neglected here. Similar assumptions can be made in animal diseases. Consider, for instance, rabies epidemic among the foxes. Rabies is a viral infection of the central nervous system. It is transmitted by contact and is invariably fatal. As stated in [7] foxes acquire the disease mainly during hunting hours or at their dens.

3. Limit cases

If our model correctly embodies the main features of the motion process in a population and the contact process of epidemics, it should describe as particular cases the limit situations modeled by systems of partial and ordinary differential equations. In this section we show how, for different values of the parameter λ defined in equation (1), we could obtain the extreme behaviors of perfect mixing and perfect diffusion. All the simulations referenced in this section were done with a lattice Λ of size $L = 150$, in other words, 150×150 sites with half of them nonempty. The initial distribution of the nonempty sites is always like black squares on a chess board. Therefore, we have a population of 11250 members. The transmission process $\epsilon_p(\epsilon_p^d)$ will be as described in

[1] For the remainder of section 2, the assertions made about ϵ_p are also valid for ϵ_p^d.

section 2: there will be only susceptibles and infectives. We always start the simulation with only one infective placed at the center of the lattice in order to avoid side effects. For all the simulations, the transportation rule does not change.

3.1 Large values of λ

If the parameter λ is large enough, then a perfect mixed behavior could be observed. Figure 3 shows the pattern of infectives for several different values of t. The length of an average path is $\lambda = 75$. Because the lattice size is 150, the value of λ is extremely high and perfect mixed behavior is easily observed. A classical differential equation model for this scenario is:

$$\begin{cases} \dfrac{dI(t)}{dt} = \alpha I(t)(11250 - I(t)) \\ I(0) = 1 \end{cases} \tag{3}$$

where $I(t)$ represents the number of infectives at instant t and α is a constant related with the morbidity of the disease. The higher the α value, the higher the growth rate of $I(t)$. We have observed a strong relationship between the constant α and the probability p of the transmission process ϵ_p. In Figure 4 is shown a graph with the averaged values of infectives obtained with 10 simulations with the same probability ($p = 0.31$) and the theoretical curve obtained from the Cauchy problem (equation (3)) with the corresponding values of α (in this case $\alpha = 0.678$). In Figure 5 is shown a graph of α *versus* p. Each point of the curve is also obtained from 10 simulations with the same value of p. The time interval was $[0, 250]$. In each simulation the values of infectives $I_0, I_1, \ldots, I_{250}$, were introduced in a linear regression using equation (3) to obtain the value of α. The process was repeated 10 times and the average is plotted.

3.2 Small values of λ

With small values of λ the cellular automata model behaves as perfect diffusive. In Figure 6 the pattern of infectives is shown for several different values of t. Notice the formation of a wave front, which grows until it covers the entire lattice. This is in agreement with other results. In [7] a diffusion term was added into the rate equation of infectives in the model proposed by Kermack and McKendrick (see [8] for details) in order to take into account the dispersion of rabid foxes. The new system of equations admitted traveling wave front solutions. Note that if only susceptibles and infectives exist, then an elementary differential equation model for this scenario is:

$$\frac{\partial u(x, y, t)}{\partial t} = D\nabla^2 u(x, y, t) + \alpha u(x, y, t)(1 - u(x, y, t)) \tag{4}$$

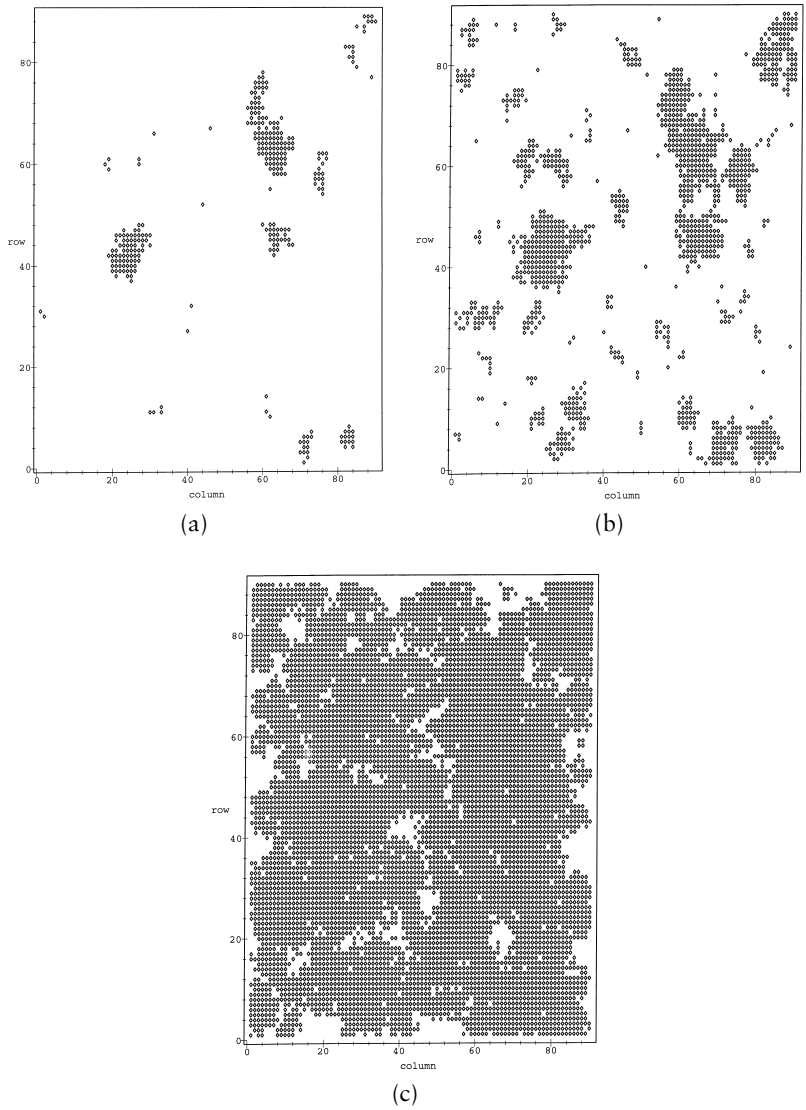


Figure 3. Deployment of contagion process for $\lambda = 75$ for several values of t: (a) corresponds to $t = 10$, (b) to $t = 30$, and (c) to $t = 50$.

where $u(x, y, t)$, represents the density of infectives in position (x, y) at instant t. The constant D is the diffusion coefficient and the constant α is related with the morbidity of the disease. The influence of λ in the behavior of D is in accordance with our perception of what a diffusion process is. As λ increases, the spread of infectives produces spatial patterns that are hard to describe as a diffusion process. This fact is

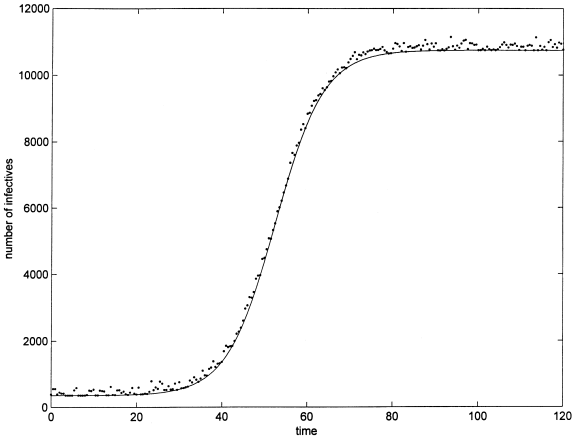


Figure 4. Averaged values of infectives from 10 simulations with the same value of probability ($p = 0.678$) and the analytical solutions of the Cauchy problem (equation (3)) with the corresponding values of α (in this case $\alpha = 0.31$).

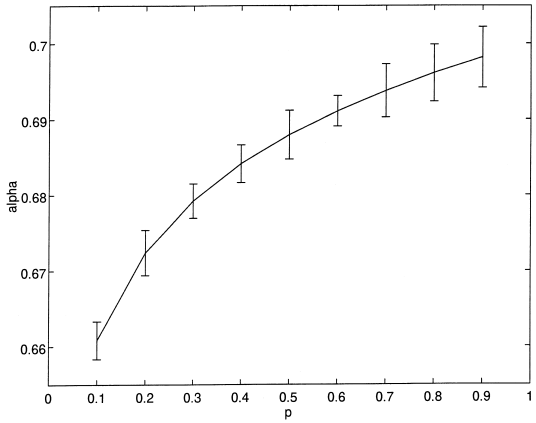
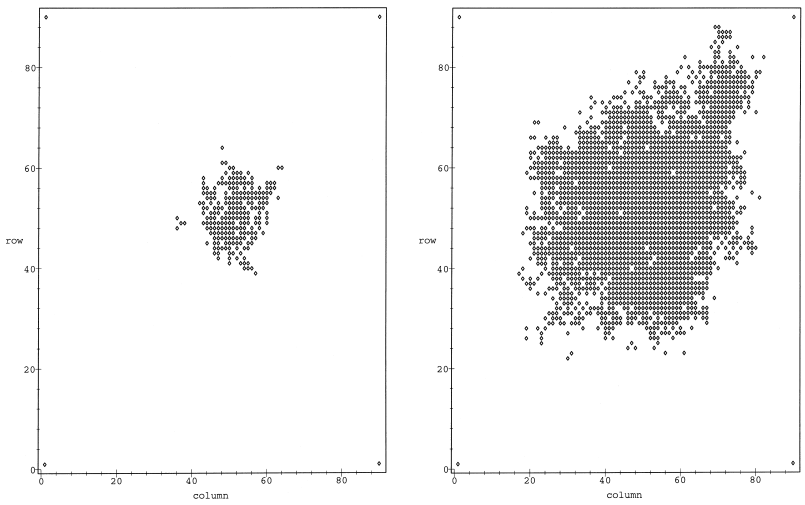


Figure 5. Rate of increase of infectives α *versus* the probability of contagion rule p.

reflected in the behavior of D with respect to λ. In Figure 7 is shown a graph of D *versus* λ. The scale in the horizontal axis represents fractions of $L/2$. For example, 0.4 represents $\lambda = 30$. Each point of the curve is obtained with 10 simulations using the same value of λ. The procedure to construct those points is as follows. In order to find an approximation of $u(x, y, t)$, the lattice Λ was divided into 15×15 squares of 10×10 sites. On each square the number of "infected" sites were counted and divided by 100. This is an approximation of $u(x_i, y_i, t)$ in the square centered at the point (x_i, y_i) at time $t \in [0, 250]$. All these values of



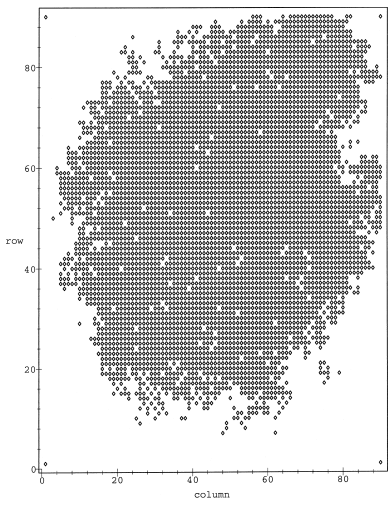


Figure 6. Deployment of contagion process for $\lambda = 15$ for several values of t: (a) corresponds to $t = 10$, (b) to $t = 30$, and (c) to $t = 50$.

u are introduced in a nonlinear weighted regression using equation (4) to obtain D and α. We use a weighted procedure in order to enhance the contribution of those squares with higher density. This process was repeated 10 times and the average values were plotted. Notice that as λ grows the size of the error bars get larger. This fact is a consequence of the diffusive regimen breaking. For values of $\lambda > 30$, the process is no longer diffusion.

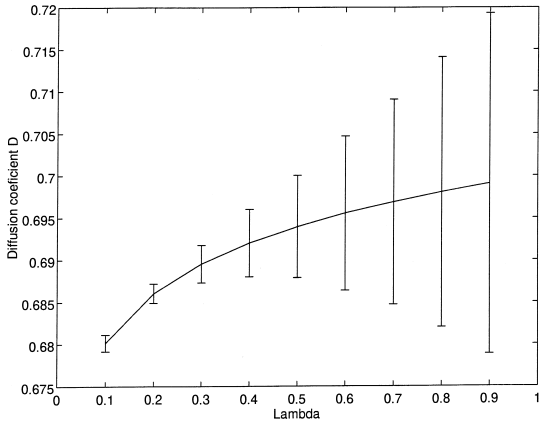


Figure 7. Diffusion coefficient D *versus* λ.

3.3 The influence of duration of disease

We consider now the above limit cases with the additional assumption that the disease has duration d. The influence of this assumption on the behavior of infection processes with values of λ in the "intermediate" zone, that is to say, too large to produce a diffusive regime and too small to produce perfect mixing behavior, will be discussed in detail in section 4. Let us consider a transmission process ϵ_p^d as defined in section 2. In both limit cases the behavior of infectives curve $I(t)$ is as follows: For certain values of T in the interval $[0, T]$ the function $I(t)$ is strictly increasing. For $t > T$ the function $I(t)$ remains almost constant at the height H. Note that this plateau means an equilibrium between the production of new infectives and the arrival of old infectives until the end of the disease. This equilibrium should be understood in a nonstrict sense, because the number of infectives fluctuates around some value as we show below. The amplitude of fluctuations strongly depends on λ and takes its smallest values for λ in the limit cases. For the values of λ in the intermediate zone the amplitude of fluctuations is large. Some interesting properties of this behavior are studied in section 4.

We study the influence of λ and p on the values of T and H. In Figure 8 is shown the infectives time series for different values of λ and some fixed value of p. Notice that as λ increases, the value of T first decreases and later increases. The turning point of T corresponds to some value of λ in the intermediate zone. Figure 9 shows the behavior of T *versus* λ for different values of p. The upper curve corresponds to $p = 0.1$, the intermediate curve corresponds to $p = 0.3$, and the lower to $p = 0.7$. Each point of the curves represents the average of 10 simulations. Hence the increase of p produces in the above described behavior the uniform descent of the curves. These results are a

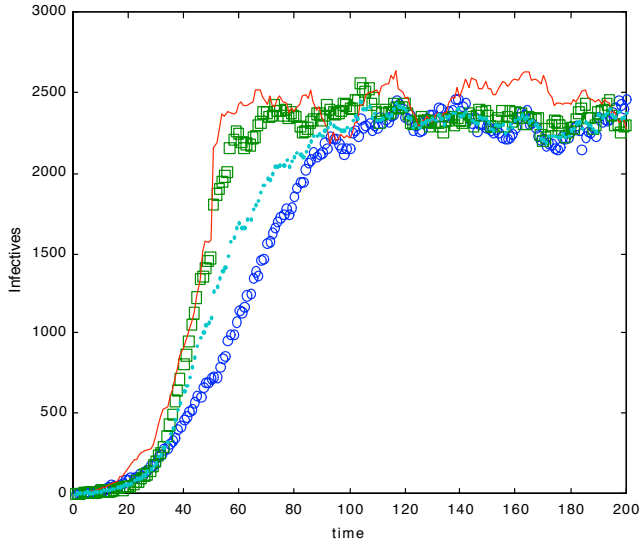


Figure 8. Different infectives time series: $\lambda = 7$ (circle); $\lambda = 24$ (square); $\lambda = 41$ (line); and $\lambda = 60$ (dot). The value of p is fixed at $p = 0.1$.

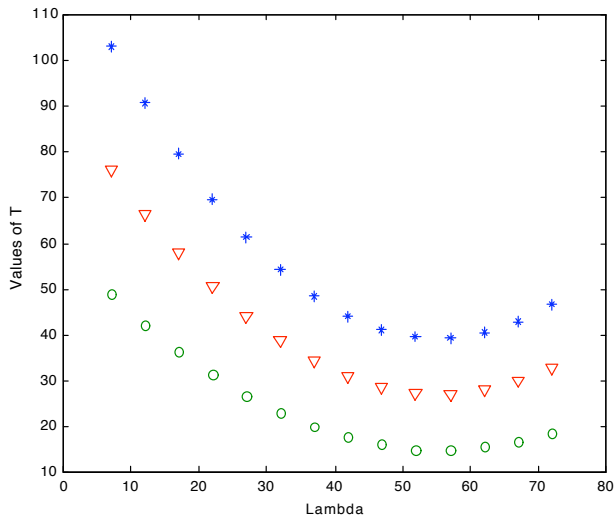


Figure 9. The behavior of T *versus* λ for different values of p. The upper curve corresponds to $p = 0.1$, the intermediate curve corresponds to $p = 0.3$, and the lower to $p = 0.7$.

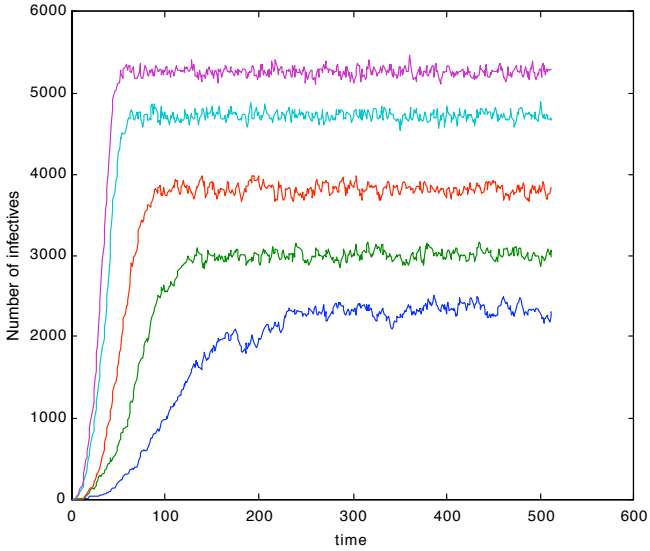


Figure 10. The infectives time series for different values of p and $\lambda = 7$. From the lowest to the highest curve: $p = 0.05$, $p = 0.09$, $p = 0.3$, $p = 0.4$, and $p = 0.7$.

consequence of the percolation of the network of infectives and are in agreement with recent results about percolation in networks (see [13]). Notice that all curves reach their minimum at "intermediate" values of λ in agreement with the results shown in Figure 8.

Figure 10 shows the time series of infectives for different values of p and for some fixed value of λ. Notice that as p increases the values of H also increase.

The results of sections 3.1 and 3.2 show that our cellular automata model could capture the main features of extreme cases described by differential equations, reported in the literature. In the following section we study the properties of the "intermediate" zone for the values of λ.

4. The behavior for intermediate values of λ

In this section we report our findings on the behavior of the site exchange cellular automata model for values of λ that are too large to yield diffusive behavior and too small to produce perfect mixing. That is what we called the "intermediate zone" in the previous section. Note that for these values of parameter λ the differential equation models fail to describe the behavior of the process.

Let us consider a transmission process ϵ_p^d as defined in section 2. Figure 11 shows the time series of infectives for $d = 5$, $p = 0.4$, and $\lambda = 45$. Notice that there is no plateau H as that described in section 3.3.

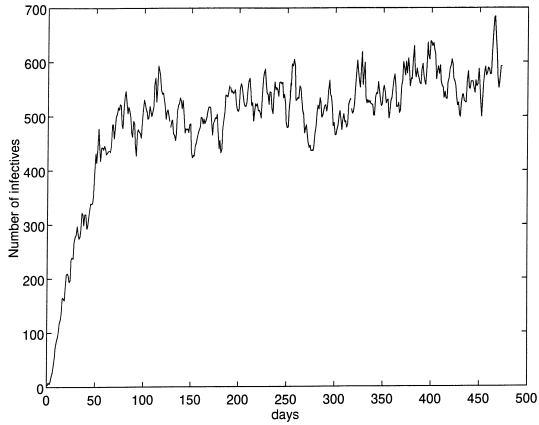


Figure 11. Times series of infectives for $\lambda = 45$ and $d = 5$.

We report a scaling property for this time series. Let us denote by $s_t = |I_{t+1} - I_t|$ the absolute value of the difference between two consecutive elements of the time series. Let $N(s)$ be the cumulative number of s, that is, how many times the value s appears in the $\{s_t\}$ series. We observe that

$$N(x) \propto \frac{1}{S^{\delta}}. \tag{5}$$

This means that a small difference between two consecutive values in the time series of infectives has a higher probability of appearing than a larger one. We also found a strong correlation between δ and p. Figure 12 shows the graph of $N(s)$ *versus* s for a "realization" of the automata model, not for averaged values. In all cases studied the fitness of $N(s)$ to a power function was good. In the inbox plot of Figure 12 are shown the averaged (over 10 simulations) values of δ *versus* the corresponding values of p. We confer a great practical value to the last result. It could allow the estimation of probability p in real epidemic processes having an accurate record of daily reported cases for several apparitions of the disease.

We also studied periodic properties of the infectives time series. Our main tool was frequency domain analysis. Fourier spectra (see [11]) are widely used in time series analysis, because the visual representation in the frequency domain can more easily reveal patterns which are harder to discern in the primary data, for example, intricate periodical behavior. We use here a Fourier transform of infectives time series to detect periodical features of that function. From now on, we call *power spectra* of infectives time series to the product of a Fourier transform of

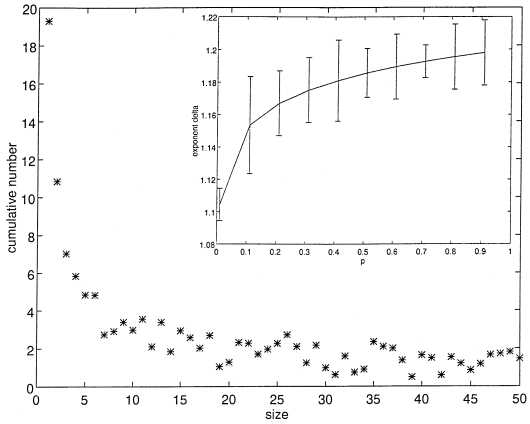


Figure 12. $N(s)$ *versus* s. Notice the power law behavior. In the inbox the exponent of the power law fit with respect to p is plotted.

that function by its complex conjugate:

$$\hat{S}(k) = \theta \left| \sum_{t=1}^{L} I(t) e^{-i2\pi(k/L)t} \right|^2 \tag{6}$$

where θ is a constant related with the sample frequency and L is the number of data available for $I(t)$.

We studied the changes in the periodic behavior of $I(t)$ with respect to the order parameter $\mu = pd/\lambda$. We calculate the power spectrum of several infectives time series with $\mu \in [0.06, 0.6]$. The results are presented in Figure 13, where behavior resembling a period-doubling scenario can be seen. It is well known that period doubling behavior is a possible route to chaotic behavior. Evidence of this feature in real contagion processes have been reported in the literature.

5. Conclusions

We have developed a cellular automata model for the spread of epidemics, rumors, and news in a population of moving individuals. Our model depends on a parameter λ, which represents the mean length of motion of individuals in a population. We reproduced, with a suitable tuning of this parameter, the limit cases of perfect mixing and perfect diffusion often described by systems of ordinary and partial differential equations, respectively. We also studied those cases which the differential equation models fail to describe. Our results prove that an important magnitude in the characterization of dynamics of epidemics in human populations is the length of the average motion of individuals in the population. In the cases where no immunization occurs, another impor-

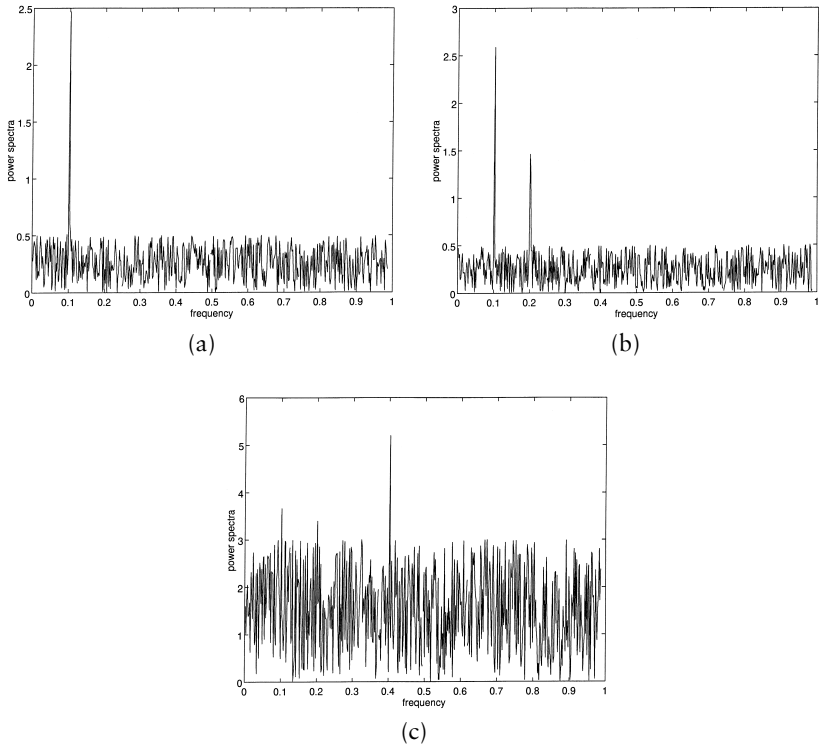


Figure 13. Power spectrum of infectives time series for different values of μ: (a) corresponds to $\lambda = 0.0699$, (b) to $\lambda = 0.3257$, and (c) to $\lambda = 0.5896$. Notice the evidence of period doubling as λ increases.

tant quantity is the duration of the disease. With intermediate values of λ and a suitable tuning of p and d we reported some evidence of period doubling behavior and other interesting properties.

Acknowledgments

The authors are thankful to G. Cocho, O. Miramontes, and P. Miramontes for their helpful comments as well as all the suitable suggestions of an anonymous referee. This work was partially (R. M.) supported by CONACYT, Mexico.

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