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Cellular automata and epidemiological models with spatial dependence

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Abstract

We present a cellular automata model developed to study the evolution of an infectivity nucleus in several conditions and for two kinds of epidemiologically different diseases. We analyse the role of the model parameters, concerning the epidemiological and demographic aspects of the problem, and of the evolution rules in relation to the spread of such infectious diseases, the arising of periodic temporal modulations related to the infectivity and recovery fronts, and the evolution of travelling waves. Among the obtained results we find analogies to endemic situations and pandemics.

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1. Introduction

The literature on epidemic models is extensive, pointing to different aspects related to the subject. A great deal of work involves phenomenological description of a certain epidemic situation [1–5]. There are several models dealing with the evolution of the densities of the population groups involved, most of them being zero-dimensional. Such models have studied, among several epidemical features, the existence of threshold values for the spread of an infection [6,7], the asymptotic solution for the density of infected individuals [8–10], the effect of stochastic fluctuations on the modulation of an epidemic situation [11], and, in some spatially dependent models, the geographic spread of an epidemic [12,13].

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When describing an infection transmitted through person-to-person contact, the population is generally classified into three groups: the S group, representing the portion of the population that has not been affected by the disease but can be infected in case of contact with a sick person; the I group corresponding to the group of individuals already infected by the disease and who are also responsible for its transmission to the susceptible group, and last the removed group R, related to those who recovered from the disease and became permanently or temporarily immune or, eventually, those who died from the illness and not from other causes. Among these infectious diseases there are two main groups: those which confer immunity to the recovered individual, most of them virus agent diseases (measles, chickenpox, mumps, HIV, poliomyelitis) [14]; and those which do not confer immunity and in which, the individual once recovered is susceptible again to infection; among them we find the bacterial agent diseases (meningitis, plague, venereal diseases) and the protozoan agent diseases (malaria) [14]. As discussed above, several models have been proposed to explain different epidemical cases. The *SIR* model is applied to those diseases that do confer immunity, and the cycle of a typical individual involves the susceptible (S), the infected (I), and the recovered and immune (R) stages. The *SIS* model deals with those diseases that do not confer immunity and the R stage is not considered. A model may or may not take into account the vital dynamics of the population, depending on the characteristic times of the development of the epidemic. This aspect of the models is expressed by the inclusion of natality and mortality terms in the equations [14].

Historically, several examples of epidemic travelling waves are known. Among them is the plague, or the Black Death [1,2], as it was known in Middle Ages Europe, and which reduced the population of the continent to a quarter of its value at the beginning of the epidemic. Other examples are the influenza pandemic in the early 20th century [3], and the spread of cholera in Asia and East Europe during the 1960s [5].

In this work we deal with diseases transmitted through person-to-person contact. We analyse the propagation of an infective nucleus under several conditions and for both *SIS* and *SIR* groups. Instead of a system of partial differential equations we consider a cellular automata with discrete time steps and matrix elements configuring a network. The rules proposed for the evolution of the automata determine the state of an element in terms of it own state and those of its neighbours at earlier time steps [15].

Among the reasons for considering a cellular automata instead of differential equations is that the computational time involved is considerably shorter. Besides, some epidemiological features can be included in a more direct way through this approach, such as the possibility of an external agent, and different stages during the infective period among others. The last possibility is considered through the division of the infected period into three phases: *incubation*, when the individual is already infected and infectious but presents no symptoms, *proper infection*, when the individual is infectious and shows the symptoms, and *latency*, when the infected individual is not infectious anymore but still has the symptoms. Finally, this model allows us to consider several boundary conditions and drawings and to model the actual shape of a certain geographical site in an easy way.

The local aspect of the interaction between the individuals is taken into account through the definition of an interaction radius. This means that an element of the network is allowed to interact with a limited number of neighbours within a bounded region.

In order to analyse the seasonal behaviour of certain epidemics we include the possibility of periodic rules governing the evolution of the automata. We look for the possibility of an endemic situation, permanent in time and restrained in area, or a pandemic, a violent growing epidemic wave. One of our goals is to analyse the possibility of spatio-temporal patterns in the behaviour of an epidemic, as pointed out in [4], after some research on an isolated population, inhabiting an island.

In the next section we present the aspects related with the cellular automata, and then we introduce the rules corresponding to different kinds of diseases. Next we describe the numerical results and finally we draw some conclusions.

2. The cellular automata

We consider a two-dimensional network represented by a matrix, its elements, time dependent, being identified by a pair of subindexes. The state of each element is univocally determined by two fields: $\pi_{ij}(t)$ and $u_{ij}(t)$. The field $\pi_{ij}(t)$ is related to the epidemiological state of the element. The number of allowed states depends also on the kind of disease considered and is described in more detail below. The field $u_{ij}(t)$ accounts for the state of the neighbourhood of certain element and is related to the probability of transitions from the present state of an individual to another, that is, to the contact and recovery rate, to put it within an epidemiological context. The rules governing those transitions depend on the features of the disease to be modelled. In this work we consider two main cases: diseases that confer immunity and those that do not. In the following subsections we describe the valid evolution rules for each case as well as considerations of particular validity.

2.1. Model without immunity

This model assumes that the disease does not confer immunity to infected individuals after recovery. Each healthy individual is susceptible of being infected through contact with sick ones. After the infectious period, the infected individual recovers and is included in the S group again. It can be shown that the inclusion of vital dynamics in this model does not affect the main results [16].

The equations for a typical adimensional *SIS* model can be written as

$$\frac{\partial S}{\partial t} = -\lambda IS + \gamma I + \mu - \mu S \,,$$

$$\frac{\partial I}{\partial t} = \lambda IS - \gamma I - \mu I \,, \tag{1}$$

where λ is the contact rate, γ the recovery rate and μ a constant related to the vital dynamics. As the following relation holds

$$S + I = 1 \,, \tag{2}$$

Eqs. (1) can be reduced to only one equation for I,

$$\frac{\partial I}{\partial t} = (\lambda - (\gamma + \mu))I - \lambda I^2 \,. \tag{3}$$

The solution for this equation is

$$I(t) = \begin{cases} \frac{\exp[(\gamma+\mu)(\lambda-1)t]}{\lambda[\exp[(\gamma+\mu)(\lambda-1)t]-1]/(\lambda-1)+1/I_0} & \text{for } \lambda \neq 1 \,, \\ \frac{1}{\lambda t+1/I_0} & \text{for } \lambda = 1 \,, \end{cases} \tag{4}$$

where $\omega = \lambda/(\mu + \gamma)$ is defined as the effective contact rate, representing the average number of adequate contacts during the period of infectiousness, and I_0 is an initial value. The asymptotic solution of this equations is

$$I(t)_{t\to\infty} = \begin{cases} 1 - 1/\omega & \text{if } \omega > 1 \,, \\ 0 & \text{if } \omega \leqslant 1 \,. \end{cases} \tag{5}$$

The cellular automata takes into account the same terms that are present in Eq. (1) but in a somehow different language. First, we note that the infected period is divided into three stages: incubation, the infection proper and latency. Each time an individual is infected he goes through these three stages, staying on average in each one a defined period of time. We call the characteristic time of the incubation stage t_i, that corresponding to the properly infected stage t_p and t_l that of the latency stage. This formulation allows us to include cases where the incubation or the latency stages could be neglected in a general way . The rules governing the evolution of the cellular automata are

$$\pi_{ij}(t+1) = \begin{cases} \pi_{ij}(t) + 1 & \text{if } 0 < \pi_{ij}(t) < t_i + t_p + t_l \,, \\ \pi_{ij}(t+1) = 0 & \text{if } \pi_{ij}(t) = t_i + t_p + t_l \,, \\ \pi_{ij}(t+1) = 0 & \text{if } \pi_{ij}(t) = 0 \text{ and } u_{ij}(t+1) < h \,, \\ \pi_{ij}(t+1) = 1 & \text{if } \pi_{ij}(t) = 0 \text{ and } u_{ij}(t+1) \geqslant h \end{cases} \tag{6}$$

for $\pi_{ij}(t)$, where h is a random number within the interval $[0, 1]$ and with probability distribution $p(h)$, that is defined for each case, while the equation for $u_{ij}(t)$ is

$$u_{ij}(t+1) = \frac{1}{N}\left(\sum_{fn} I_{ij}(t)\, e^{-1} + \sum_{sn} I_{ij}(t)\, e^{-2} + \sum_{tc} I_{ij}(t)\, e^{-3} + \cdots \right). \tag{7}$$

The successive terms correspond to sums over the first, second and further neighbours respectively and N corresponds to a normalization constant,

$$N = \frac{1}{4(e^{-1} + e^{-2} + e^{-3} + \cdots)} \,.$$

This series is truncated according to the choice of the interaction radius. The field $I_{ij}(t)$ is defined as

$$I_{ij}(t+1) = \begin{cases} F(\pi_{ij}(t)) & \text{if } \pi_{ij}(t) \geqslant 1 \ , \\ 0 & \text{if } \pi_{ij}(t) = 0 \ , \end{cases} \tag{8}$$

where $F(t)$ is a positive real function $F(t): (0, t_i + t_p + t_l) \rightarrow \Re^+$. The role of $F(t)$ is to assign to a certain individual an effective infectiousness as a function of its state, letting us distinguish between non-infectious and infectious individuals. Besides, the varying infective power during the three stages of the infected period is assigned by $F(t)$, time dependent.

Many infectious diseases, including measles, mumps, rubella, chickenpox, poliomyelitis, diphtheria, pertussis, gonorrhea and influenza, have been observed to show periodicity and other oscillatory behaviours. For example, there were yearly outbreaks of chickenpox and mumps from 1929 to 1970 in New York City [17]. In order to take into account this type of behaviour we also consider periodic modulations of $p(h)$, accounting for seasonal oscillations of the incidence of some diseases over the population, that is highly related with an oscillatory behaviour of the contact rate. In the corresponding case for the *SIS* model, the solution of Eqs. (1) shows that the number of infected and of susceptible individuals oscillates around the stationary value for the case when λ is a constant [14]. We can see that $F(t)$ and $p(h)$ in the automata rules play the role played by ω in the *SIS* model.

2.2. Model with immunity

This model assumes that the disease under study confers immunity to infected individuals after recovery. Once the infected individual recovers, he is included in the R group temporarily or permanently. The inclusion of vital dynamics changes qualitatively the behaviour of the system [16]. In this subsection we discuss two different cases. First, we consider the simplest situation, when after the infective stage, the individual recovers and stays immune for a lifetime. This situation is associated with the *SIR* model. The equations for a typical zero-dimensional *SIR* model can be written as

$$\begin{aligned} \frac{\partial S}{\partial t} &= -\lambda IS + \mu - \mu S \ , \\ \frac{\partial I}{\partial t} &= \lambda IS - \gamma I - \mu I \ , \\ \frac{\partial R}{\partial t} &= \gamma I - \mu R \ , \end{aligned} \tag{9}$$

where the recovered individuals are considered no longer as a (positive) contribution for the S group, but instead for the R one. Eqs. (9) can be written as a set of two equations for I and S, because the one for R is not coupled to them. Clearly, the behaviour of this model is more complex than the one studied above.

The rules governing the evolution of the cellular automata for the present case are the following:

$$\pi_{ij}(t+1)=\begin{cases}\pi_{ij}(t)+1 & \text{if } 0<\pi_{ij}(t)<t_i+t_p+t_l\ ,\\ -1 & \text{if } \pi_{ij}(t)\geqslant t_i+t_p+t_l\ ,\\ 0 & \text{if } \pi_{ij}(t)=0 \text{ and } u_{ij}(t+1)<h\ ,\\ 1 & \text{if } \pi_{ij}(t)=0 \text{ and } u_{ij}(t+1)\geqslant h\ .\end{cases}$$

The field $u_{ij}(t)$ adopts the same form as before

$$u_{ij}(t+1)=\frac{1}{N}\left(\sum_{fn} I_{ij}(t)\,e^{-1}+\sum_{sn} I_{ij}(t)\,e^{-2}+\sum_{tc} I_{ij}(t)\,e^{-3}+\cdots\right) \tag{10}$$

and

$$I_{ij}(t+1)=\begin{cases}F(\pi_{ij}(t)) & \text{if } \pi_{ij}(t)\geqslant 1\ ,\\ 0 & \text{if } \pi_{ij}(t)\leqslant 0\end{cases} \tag{11}$$

as in the former case, $F(t)$ is a positive real function $F(t):(0,t_i+t_p+t_l)\to\Re^+$ and $F(t)=0$ for x outside the interval $(0,t_i+t_p+t_l)$. A more general case is represented by the *SIRS* model, when the immune period is finite and of length t_r. After this period of time the individual becomes susceptible again. In such cases the rules governing the cellular automata are

$$\pi_{ij}(t+1)=\begin{cases}\pi_{ij}(t)+1 & \text{if } 0<\pi_{ij}(t)<t_i+t_p+t_l\ ,\\ -1 & \text{if } \pi_{ij}(t)=t_i+t_p+t_l\ ,\\ \pi_{ij}(t)-1 & \text{if } -t_r\leqslant\pi_{ij}(t)<0\ ,\\ 0 & \text{if } \pi_{ij}(t)<-t_r\ ,\\ 0 & \text{if } \pi_{ij}(t)=0 \text{ and } u_{ij}(t+1)<h\ ,\\ 1 & \text{if } \pi_{ij}(t)=0 \text{ and } u_{ij}(t+1)\geqslant h\ .\end{cases}$$

When $t_r\to\infty$ we have the former case (*SIR* models) as a particular situation; when $t_r=0$ we are in the case related to *SIS* models and when $t_r\neq 0$ the situation is associated with *SIRS* models.

In contrast with the case analysed in the previous subsection, we have to distinguish between cases with or without vital dynamics. In the present work we do not include vital dynamics. This feature will be taken into account in a future work.

3. Numerical results

In this section we present the results obtained for both cases. The main goal was different in each model. While the main feature to be analysed in models without immunity, which are described first, is the asymptotic mean density of the infected individuals, I_a, and the existence of threshold values, in models with immunity the

objective is to find numerically travelling pulses of epidemic. In each case we draw a comparison with the results obtained through ordinary differential equations [16]. In all the simulations we have considered 1000 realizations over a 100×100 lattice with the initial condition

$$u_{ij}(0) = 0 \quad \forall i, j \tag{12}$$

and

$$\pi_{ij}(0) = \begin{cases} t_i & \text{if } \sqrt{(50-i)^2 + (50-j)^2} \leqslant 5 \ , \\ 0 & \text{elsewhere} \ . \end{cases} \tag{13}$$

In order to study the effect of the occupation number on the evolution of an initial focus, we take a random distribution with a given density over the whole lattice with the exception of the sites occupied by the initial focus, taken as the same identical initial condition in all the cases. We consider Dirichlet boundary conditions.

3.1. Models without immunity

As pointed out above, the main interest in this case is to analyse the asymptotic level of infected individuals as the relevant parameters of the problem vary in certain range. As a first step and recalling Eqs. (6) and (8), we choose $t_i = t_p = t_l = 2$, $p(h) \equiv 1$, and

$$F(\pi_{ij}(t)) = \begin{cases} f & \text{if } \pi_{ij}(t) > 0 \ , \\ 0 & \text{if } \pi_{ij}(t) = 0 \end{cases} \tag{14}$$

thus studying the effect on I_a of f only. We consider interactions between first neighbours, neglecting further contributions. A deeper analysis shows that the main results are only affected quantitatively but not qualitatively by this approximation. The numerical results obtained in this step show clearly, as can be seen in Fig. 1, a phase transition for a certain critical value f_c. Below this value the infection cannot be sustained and the epidemic gradually fades. But for values above f_c the epidemic evolves, the infected individuals density, $I(t)$, reaching a stationary mean value. The analysis of the behaviour of I_a as a function of f shows the transition to a new phase. Near f_c, $I_a(f)$ can be fitted with a critical-like power-law curve

$$I_a = A|f - f_c|^{\alpha} \ .$$

The procedure for obtaining the best fit is well described in [18].

The next step is to consider the incubation and latency times, setting $f_i = f_l = 0$. For this case

$$F(\pi_{ij}(t)) = \begin{cases} f_i & \text{if } 0 < \pi_{ij}(t) \leqslant t_i \ , \\ f_p & \text{if } t_i < \pi_{ij}(t) \leqslant t_i + t_p \ , \\ f_l & \text{if } t_i + t_p < \pi_{ij}(t) \leqslant t_i + t_p + t_l \ . \end{cases}$$

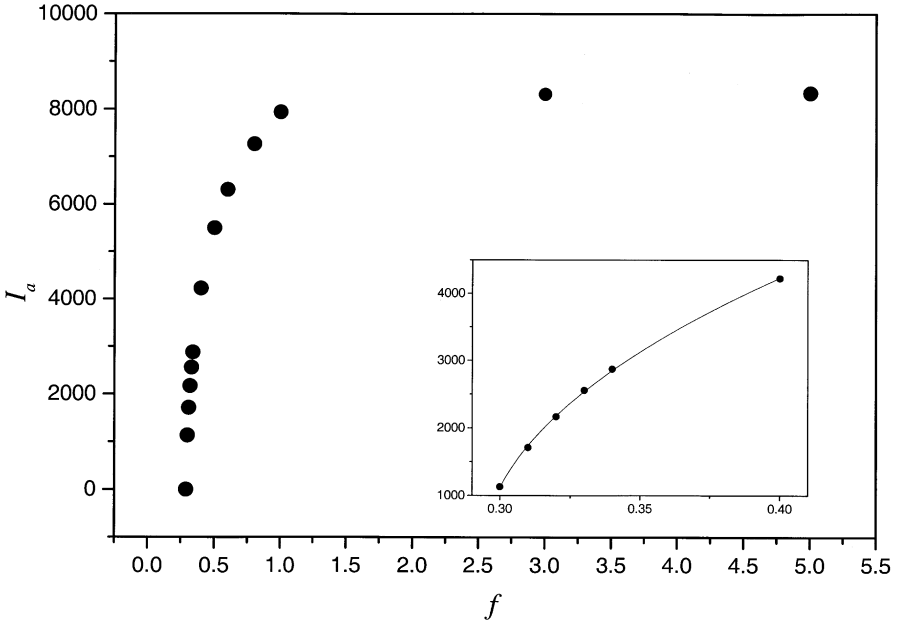

Fig. 1. Asymptotic density of infected individuals as a function of f. In the inset we show the scaling of the data with a power-like curve $A|f - f_c|^{\alpha}$. With $A = 14540 \pm 5.2 \times 10^2$, $f_c = 0.2916 \pm 0.6 \times 10^{-3}$ and $\alpha = 0.534 \pm 1.7 \times 10^{-2}$.

The results are very similar to those obtained in the former case, as can be seen in Fig. 2. The main differences are the displacement of the critical value f_c and a different value for the critical exponent α.

Now we study the case when $p(h)$ is periodically modulated, accounting for a seasonal oscillation of the incidence of some diseases over the population. We define

$$p(h) = \begin{cases} 1 & \text{if } t \in \left[nT; \left(n + \frac{1}{2}\right) T\right) , \\ \frac{3}{2} h^{1/2} & \text{if } t \in \left[\left(n + \frac{1}{2}\right) T; (n+1)\, T\right) , \end{cases}$$

where T is the period of this oscillation and $t_i = t_p = t_l = 2$, $F(t)$ is defined as in Eq. (14), and $n = 0, 1, 2, \ldots$. In this way we obtain a $p(h)$ modulated in time. While the constant value represents a stage or a period over which the infectiousness of the disease remains fixed, the shape of $p(h)$ within the interval $[(n + \frac{1}{2})T; (n + 1)T)$ corresponds to another for which, due to a set of epidemiological related factors, the disease turns more infective. The selection of $p(h)$ is not unique and the one used in this work was chosen in order to show in a more apparent way the effects of such modulation.

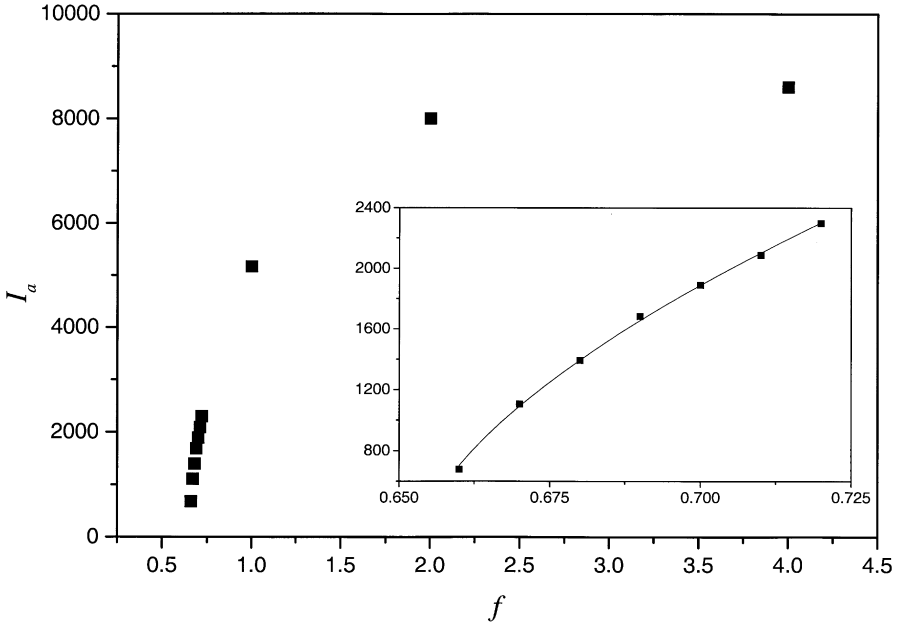

Fig. 2. Asymptotic density of infected individuals as a function of f. In the inset we show the scaling of the data with a power-like curve $A|f - f_c|^{\alpha}$. With $A = 10530 \pm 6.2 \times 10^2$, $f_c = 0.6513 \pm 0.5 \times 10^{-3}$ and $\alpha = 0.569 \pm 1.8 \times 10^{-2}$. *SIS* model with latency.

We analyse the oscillatory behaviour of I, as a function of a given initial occupation of the lattice, d_i. We want to compare our results with the observed oscillatory behaviour of the evolution of certain diseases and its relation with the population density of the infected areas. Indeed, in [4] the authors describe the different structures adopted by the seasonal behaviour of measles in communities with different population densities. These communities are divided into three groups according to their density being high, medium or low. In all the cases the occurrence of temporal peaks of infection is observed, but with different properties. In large communities the epidemic waves show a periodic behaviour, with peaks mounted over a constant background which represents the endemic stable level. We will refer to this background level as the endemic background density. In the smallest communities the behaviour is completely irregular and discontinuous, and the occurrence of peaks is almost exclusively associated to external factors. Within intermediate communities the behaviour of the epidemic presents periodic but isolated peaks, that is, without endemic periods between them. Based on these results we define two quantities to be analysed: (a) the relative endemic background of the epidemic I_0, as the ratio between the endemic background density and the initial density or occupation number, d_i; and (b) the relative amplitude of the peak I_p, calculated as the standard deviation of the oscillatory density over the initial density d_i.

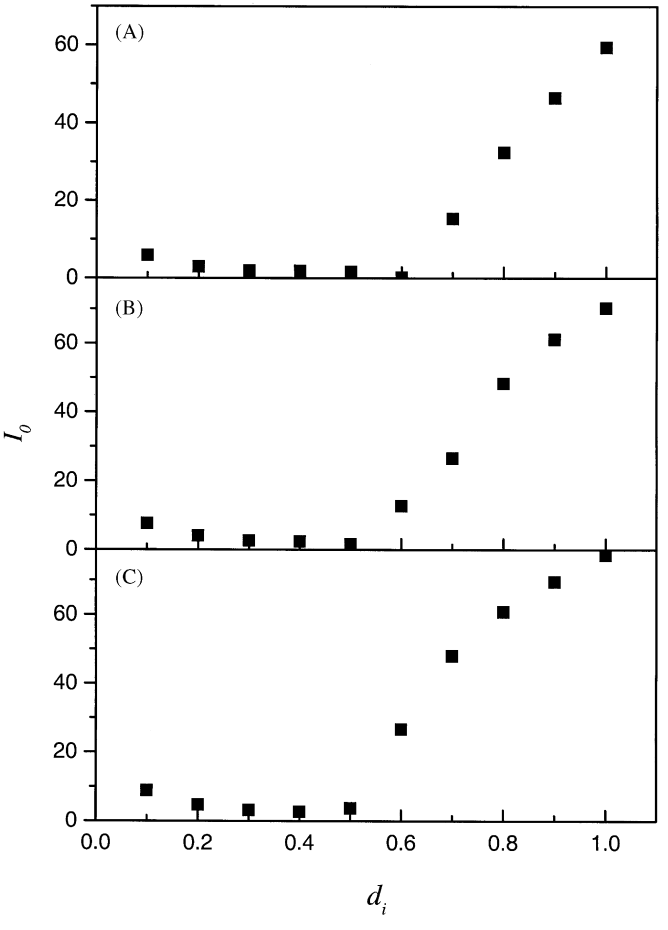

Fig. 3. Relative endemic background value, I_0, vs. d_i for three different values of f. (A) $f = 0.8$, (B) $f = 0.9$, (C) $f = 1.0$. *SIS* periodic model.

The first result obtained is that for the period of oscillation chosen (100 time steps), the epidemic cannot be sustained when $f < 0.8$. In Fig. 3 we show the behaviour of I_0 as a function of d_i for different values of f. For $f \geqslant 0.8$ we find that as the occupation of the lattice, d_i, increases from 0 to 0.6, I_0 decreases. This tendency is reversed for $d_i > 0.6$, when I_0 begins to grow until a saturation level for $d_i = 1$. This fact shows a particular behaviour of the system around $d_i = 0.6$, that is revealed in a much clearer way when we analyse the non-monotonic behaviour of I_p. As can be seen in Fig. 4, I_p has a high peak near $d_i = 0.6$ that can be associated to a phase transition. Previous works [19], have shown that local epidemic models with immunization are in the same universality class as percolation cluster growth models. The percolation threshold for square lattices is equal to 0.593 [20]. In our periodic *SIS* model, though there is no real immunization, an effective immunization can be achieved in those periods when the probability of infection is very low.

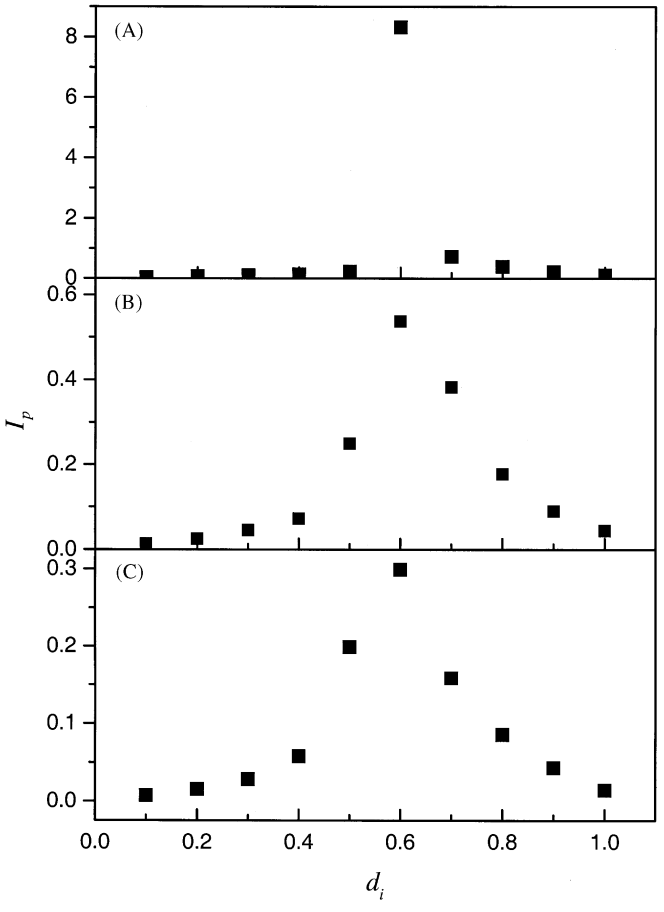

Fig. 4. Asymptotic peak amplitude over initial density vs. d_i, for three different values of f (as in Fig. 3). *SIS* periodic model.

3.2. Models with immunity

Here we analyse both the presence of travelling waves and the asymptotic level of the density of individuals never affected by the epidemic, for different sets of values for the relevant parameters. We also study the behaviour of the front velocity as a function of d_i. As in the former case we begin with $t_i = t_p = t_l = 2$, $p(h) \equiv 1$, and

$$F(\pi_{ij}(t)) = \begin{cases} f & \text{if } \pi_{ij}(t) > 0 \,, \\ 0 & \text{if } \pi_{ij}(t) \leqslant 0 \,. \end{cases}$$

Again, we consider interactions between first neighbours, neglecting further contributions.

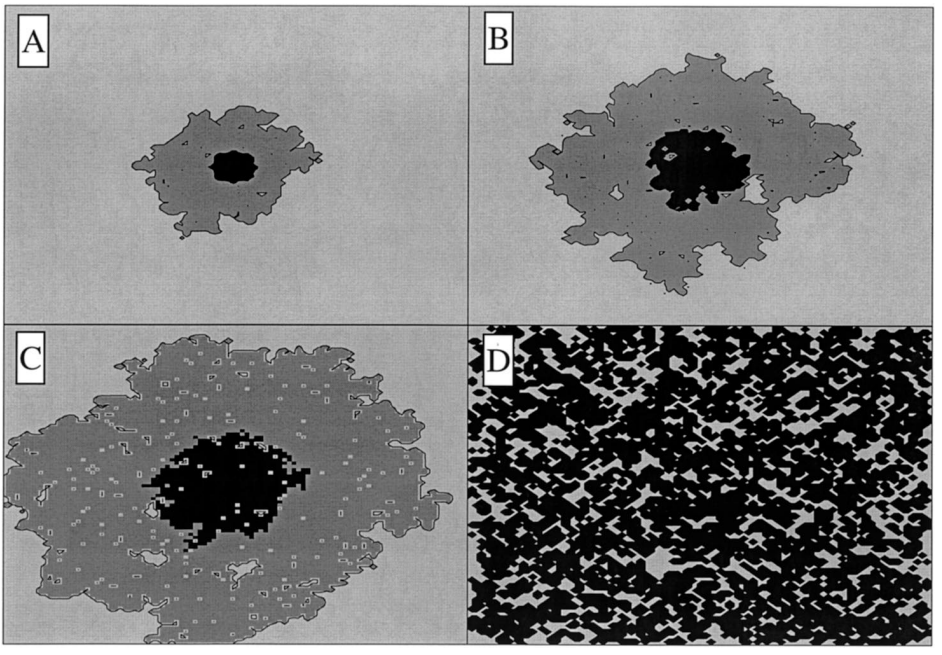

Fig. 5. (A–C) Three consecutive stages of the evolution of the cellular automata for the *SIR* model, separated by 100 time steps. Light gray, dark gray and black indicate susceptible, infected and removed individuals respectively. (D) Asymptotic state of the cellular automata for the *SIS* model. Gray and black indicate susceptible and infected individuals respectively.

The observed behaviour of the system is qualitatively different from that of the case analysed previously. The numerical results show the presence of travelling waves and the occupation of almost all the lattice by the removed group once the epidemic wave has passed through. In Fig. 5 we can observe four stages of the evolution of the epidemic, showing the epidemic wave and the presence of islands of susceptible individuals, places that have not been affected by the epidemic. This fact is due to the random process that governs the evolution rules of the cellular automata. The density of such islands, S_a, depends strongly on f, a fact developed in Fig. 6, where we plot the asymptotic density of susceptible individuals, representing those not reached by the infection. The presence of a phase transition for a certain critical value f_c is again observed. Above this value the stationary mean number of unaffected individuals goes to zero. But for values below f_c the value of S_a grows, reaching a stationary mean value bounded by $S_a(0)$, the initial number of susceptible individuals. The analysis of the behaviour of S_a versus f shows a phase transition. Near f_c, $S_a(f)$ can be fitted with a critical-like power-law curve

$$S_a = S_a(0) - A|f - f_c|^{\alpha} .$$

The observed tail in the inset of Fig. 6 can be easily explained. We consider an initial infective nucleus that grows initially infecting some individuals who after some time

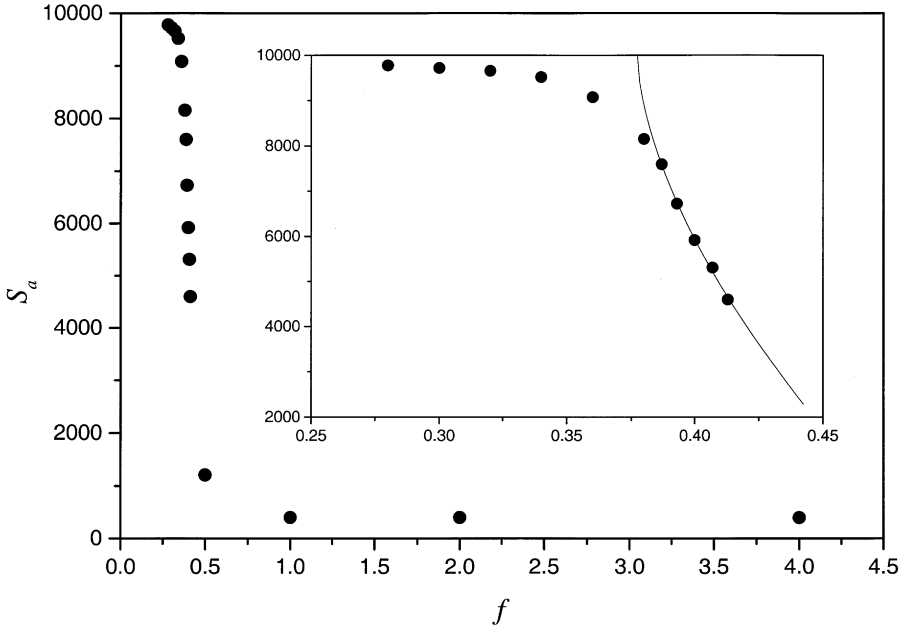

Fig. 6. Asymptotic density of the unaffected individuals as a function of f. In the inset we show the scaling of the data with a power-like curve $S_a(0) - A|f - f_c|^{\alpha}$. With $A = 40859 \pm 13563$, $f_c = 0.3771 \pm 6.7 \times 10^{-3}$ and $\alpha = 0.61 \pm 0.11$.

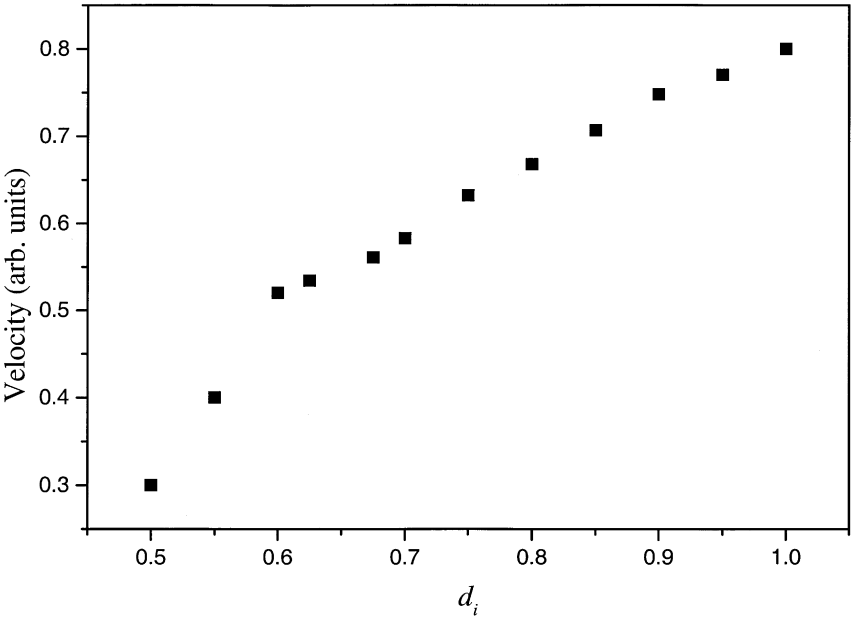

Fig. 7. Propagation velocity of the epidemic travelling wave as a function of d_i.

become immune. Depending on the values chosen for the parameters the nucleus will grow or collapse but there is always a small number of infected individuals as a result of the initial transient condition. These infected individuals account for the tail in Fig. 6.

Finally we analyse the velocity of propagation of the travelling wave for several values of d_i. We plot the radius of the infective front as a function of time obtaining a curve that can be linearly fitted, with low errors in the coefficients resulting in a well-defined value for the velocity. The results are shown in Fig. 7, where two regimes for the behaviour of the velocity can be observed.

4. Conclusions

There are so many aspects to be studied when considering an epidemiological model that the analysis must be necessarily limited. We have chosen to analyse different epidemiological features in each one of the models presented in this work. When presenting the rules governing the cellular automata we said that the function $F(t)$ was related to the infectiousness of the epidemic. In the subsequent analysis we have defined $F(t)$ as a Heaviside like function adopting different constant values, f, and also varying according to the stage of the infective period being that of latency or of incubation. This form for $F(t)$ is the simplest choice but it allowed us to perform the desired analysis. A more complex form for $F(t)$ can be chosen in order to model the evolution of a specific epidemic. First, we have studied the *SIS* model. We have analysed the effect of the value of f on the stationary mean density of infected individuals, first setting the incubation and latency times equal to zero and then considering the infective period as divided in three stages of equal length. Actually, the latent stage can be considered as an effective short time immunity. Its effect is to lower the value of the stationary mean infective density for a given value of f. From the point of view of our model, the effect of the incubation period is not so apparent, but by choosing an adequate and more specific shape for $F(t)$ during the incubation period, the essential features of a particular epidemic could be represented through the cellular automata. Also, the incubation period can be more relevant if mobility is added to the model and the individuals have the ability to recognize the infected ones presenting symptoms, and move away from them. In that case, to be studied in a future work, the lapse of time between the moment when one individual becomes infectious and that when he can be recognized as sick could be of non-negligible importance. The existence of a threshold for the propagation of an epidemic is a well-known result when modelling a disease by the *SIS* model. We have found that the threshold value for f depends on the duration of the infective period and on the existence or not of latentcy and incubation stages. The curve obtained for the stationary mean density of infected individuals near that threshold value shows the existence of a phase transition. In that region the data has been fitted with a critical-like power-law curve. This aspect is more related to cellular automata than with epidemics, but we have analysed the effect of latency and incubation on the critical exponent value, which is larger when those are considered.

Next we studied the effect of periodic modulation on the rules governing the cellular automata. The main goal here was comparing our results with those obtained for the periodic behaviour of measles in [4] from direct observation on an isolated population. In that work the authors describe the structures adopted by the epidemic depending on the density of the population of a certain community. According to its population density, communities can be divided into three main groups, the periodic evolution of an epidemic adopting distinct features in each one. While in densely populated communities the epidemic waves show a periodic behaviour, with peaks mounted over an endemic background, in the smallest communities the behaviour is completely irregular and discontinuous attributing the occurrence of peaks almost exclusively to external factors. The behaviour in intermediate communities can be associated with an intermediate behaviour between that of the former two cases, with periodic but not continuous peaks, without endemic periods between them. We have defined two quantities: the relative endemic background of the epidemic, I_0, and the relative amplitude of the peak, I_p. We have found that there is a transition near $d_i = 0.6$, that may be associated with the percolation threshold for square lattices, equal to 0.593 [20]. The endemic background grows for $d_i > 0.6$, while the amplitude of the peaks becomes lower. This means that as the initial density increases, the ratio between the endemic background and the peaks decreases. This result agrees with that presented in [4]. For densely populated communities the endemic value is relatively high with small peaks mounted over it, while for smaller communities the relative amplitude of the peaks is much higher, the endemic value being almost zero. The results for $d_i < 0.6$ show that while the endemic background remains low, the amplitude of the peaks tends to zero monotonically as d_i decreases, leading us to state that a high peak can appear only due to an external fluctuation. The peak around $d_i = 0.6$ in Figs. 3 and 4 is of bounded amplitude due to the finite size of the lattice. Further analysis should be done in order to determine the possibility of a divergence around this point for bigger lattices.

Though the same analysis done for the *SIS* model can be also done for the *SIR* model we decided to study numerically other features not found in the former. We analysed the surge of travelling waves of infection and their propagation velocity. We studied the case when the acquired immunity is permanent. The non-deterministic behaviour of the cellular automata due to the random character of h leads to the appearance of islands of susceptible individuals that have not been affected at all by the epidemic. The stationary mean density of these individuals as a function of f has been plotted in Fig. 6, behaving like I_s in the *SIS* model. We have found an apparent change near $f = 0.37$. In that region the curve was fitted with a critical-like power-law curve. The remaining tail can be attributed to the transient evolution of the cellular automata from the initial condition. In the first steps the initial infectious nucleus evolves infecting its neighbours and then growing or collapsing depending on the values chosen for the parameters. Due to this initial stage there is always a remainder of removed individuals.

Finally, we have studied the propagation velocity of the travelling wave as a function of d_i. We have found that the velocity increases monotonically, but shows two different regimes. It grows at a high rate for $d_i < 0.6$, and more slowly for $d_i > 0.6$. From the

point of view of the epidemic aspects this result agrees with the fact that the epidemic nucleus generally appears in a densely populated area, advancing at a certain speed and reaching other regions with lower population density as its propagation velocity decreases [4]. Taking into account that we are modelling the problem through cellular automata, the change of regime in the behaviour of the velocity around $d_i = 0.6$ may be associated with the threshold percolation density for square lattices [20].

The vital dynamics and mobility, considering diffusive and transport terms, will be included in a future work.

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