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Oscillations in an epidemiological model based on asynchronous probabilistic cellular automaton

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ABSTRACT

Consider a contagious disease affecting a host population composed of two groups with distinct habits. At each time step, each individual of this population can be in one of two states: susceptible (S) or infective (I). Here, a SIS epidemic model based on cellular automaton (CA) is proposed to study the disease spreading in such a population. In this model, the state transitions are described by probabilistic rules and each group has its own schedule to update the states of its individuals. We also propose a set of difference equations (DE) to analyze this population dynamics and we show how these two approaches (CA and DE) can be equivalent. We noticed that oscillations can be found in the composition of the group with more active social life, but not in the composition of the other group.

1. Introduction

One of the main goals of eco-epidemiological studies is to determine if a contagious disease will become endemic or not in a host population. When a pathogen endemically remains, either it persists at an approximately constant level or its prevalence oscillates as the time passes (e.g. Anderson and May, 1992; Hethcote and Levin, 1989). Here, we examine this issue by assuming that the host population is formed by two groups with different social and personal behaviors. These differences are expressed in the infection rate, in the cure rate, and in the schedule for updating the status of the individuals regarding the progress of the infection. Thus, this work is supported by a risk-structured model (e.g. Anderson and May, 1992; Rock et al., 2014), an approach commonly used to analyze the spread of sexually transmitted diseases, such as human immunodeficiency virus (e.g. Jacquez et al., 1988; Piqueira et al., 2004), human papillomavirus (e.g. Alsaleh and Gumel, 2014; Tobin and Comiskey, 2013), and *Chlamydia trachomatis* (e.g. Althaus et al., 2012; Sharomi and Gumel, 2011).

Models based on cellular automaton (CA) have been employed for investigating the propagation of contagious disease (e.g. Ahmed et al., 1998; Boccara et al., 1994; Doran and Laffan, 2005; Ferreri and Venturino, 2013; Fuentes and Kuperman, 1999; Ilnytskyi et al., 2016; Schimit and Monteiro, 2009; Silva and Monteiro, 2014; Sirakoulis et al., 2000; Slimi et al., 2009; Yakowitz et al., 1990; Yang et al., 2015), because features of the host population and its interaction with the contagious pathogen can be naturally taken into account and conveniently programmable in digital computers. Some of these features include the spatial locations of the individuals, their social activities, their movements, the network of social contacts, disease control policies, the state transitions related to the stages of the disease. Also, the parameters of these CA models do have biological meaning. However, this approach has a drawback: usually, the long-term behavior can be determined only by running numerical simulations. In order to try to analytically predict the attractor of this dynamical system, equivalent models written in terms of differential/difference equations have been derived. In fact, since the 1980s (e.g. Omohundro, 1984; Toffoli, 1984; Vichniac, 1984), equivalences between cellular automata and differential/difference equations have been investigated (e.g. Ahmed et al., 1998; Boccara et al., 1994; Ferreri and Venturino, 2013; Fuentes and Kuperman, 1999; Ilnytskyi et al., 2016; Monteiro et al., 2006; Schimit and Monteiro, 2009; Silva and Monteiro, 2014; Sirakoulis, 2004; Slimi et al., 2009; Yang et al., 2015).

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The epidemiological model proposed here is written in terms of a probabilistic CA with asynchronous update. Therefore, the state transitions of the cells composing the lattice are determined from probabilistic rules and the states of all cells are not updated at the same time steps (e.g. Fatés, 2014; Manzoni, 2012). In addition, to explain the long-term behavior found in these simulations, an equivalent model written as a set of difference equations (DE) is analyzed.

This manuscript is organized as follows. The CA model and the corresponding numerical simulations are presented in Section 2. The DE model is examined in Section 3. It is also shown how the parameter values of the DE approximation must be estimated to reproduce the results obtained from the CA simulations. In Section 4, the possible relevance of this work is stressed.

2. The CA model

Suppose that the individuals of the host population live in a square matrix composed of $n \times n = N$ cells with periodic boundary conditions (that is, the bottom edge of the matrix contacts the top edge and the right edge contacts the left edge; thus, a torus embedded in a three-dimensional space is formed from a two-dimensional lattice in order to eliminate edge effects). Each cell corresponds to an individual. The neighborhood of a cell is formed by its eight surrounding cells, which is usually known as Moore neighborhood of unit radius (e.g. Wolfram, 1994). Due to the chosen boundary condition, all cells have the same number of neighbors.

At each time step t, each cell is in one of two states: susceptible S_i or infective I_i, with $i = 1, 2$ denoting the type of behavior. Recovery from the studied disease does not induce protective immunity. There are $N_1 = (1-\mu)N$ individuals belonging to the group-1 and $N_2 = \mu N$ individuals in the group-2, with $0 \le \mu \le 1$. These two types of individuals are randomly and homogeneously distributed over the space. They stay in their groups along the simulation; hence, $N_i = S_i(t) + I_i(t) =$ constant. The assumption $N_1 + N_2 = N =$ constant is appropriate for modeling infections spreading quickly and/or populations in which the deaths are balanced by the births.

The states of the cells belonging to the group-1 are updated at each time step; the states of the cells belonging to the group-2 are updated at each β time steps. Obviously, if $\beta = 1$, then the states of all cells would be synchronously updated throughout a simulation. If $\beta > 1$, the CA is said to be asynchronous. In this model, it is also supposed that individuals of the group-2 interact with individuals of both groups only at each β time steps. Thus, the group-2 is formed by individuals with a less active social life, when compared to individuals of the group-1.

The time evolution of this SIS (susceptible-infective-susceptible) model is ruled by the following set of probabilities of state transitions. First, consider the group-1. At the time steps $t = h\beta$, with $h = 1, 2, 3$, and so on, there is a probability $P_1(\nu_1) = 1-e^{-k_1\nu_1}$ of a S_1-individual being infected by an I_1-individual, in which ν_1 is the number of I_1-neighbors (of course, $0 \le \nu_1 \le 8$). Note that $P_1(0) = 0$. Note also that $P_1(\nu_1)$ increases with ν_1; hence, the higher the number of I_1-neighbors, the higher the chance of a S_1-individual becoming sick. If S_1 was not infected by I_1-neighbors, then there is a probability $P_2(\nu_2) = 1-e^{-k_2\nu_2}$ of S_1 being infected by an I_2-individual, in which ν_2 is the number of I_2-neighbors. The parameters k_1 and k_2 express the pathogen infectivity combined with the habits of each group. Also, each I_1-individual has a probability P_3 per time step of becoming cured and, consequently, susceptible again. At the time steps $t \neq h\beta$, the state transitions of the individuals of the group-1 only are ruled by the probabilities $P_1(\nu_1)$ and P_3.

Now, consider the group-2. At the time steps $t = h\beta$, there is a probability $P_2(\nu_2)$ of a S_2-individual being infected by an I_2-individual; and, if this infection did not occur, then there is a probability $P_1(\nu_1)$ of S_2 being infected by I_1. The probability of I_2 being cured is P_4. At the time steps $t \neq h\beta$, the states of the individuals of this group are not updated.

In Figs. 1–7, $n = 200$ (therefore, the host population is composed of $N = 40{,}000$ individuals) and the initial condition is $I_1(0)/N = 0.005(1-\mu)$ and $I_2(0)/N = 0.005\mu$ (consequently, $S_1(0)/N = 0.995(1-\mu)$ and $S_2(0)/N = 0.995\mu$). Thus, in $t = 0$, there are 0.5% of infective individuals and 99.5% of susceptible individuals. The attractors shown in these figures are indeed global attractors (because they are reached from any initial condition with $1 \le I_1(0) + I_2(0) \le N$).

The curves exhibited in Figs. 1(a)–6(a) represent the average results obtained in 20 simulations. The standard deviations of both average curves are about 2%.

Figs. 1(a) and 2(a) present the time evolutions of $I_1(t)/N$ (thick line) and $I_2(t)/N$ (thin line) by taking $\beta = 5$, $k_1 = k_2 = 0.3$, and $P_3 = P_4 = 0.5$. Note that both groups have the same parameter values regarding their interactions with the pathogen. In 1(a), $\mu = 0.1$; in 2(a), $\mu = 0.9$. Observe that, by increasing μ (the proportion of the group-2), I_2 increases and I_1 decreases. This result is not surprising: the higher the amount of individuals belonging to the group-i, the higher the number of $I_i(t)$ as the time passes. However, it is interesting to note that $I_2(t)$ tends to a steady state for both values of μ and $I_1(t)$ to a steady state or a periodic solution, depending on μ.

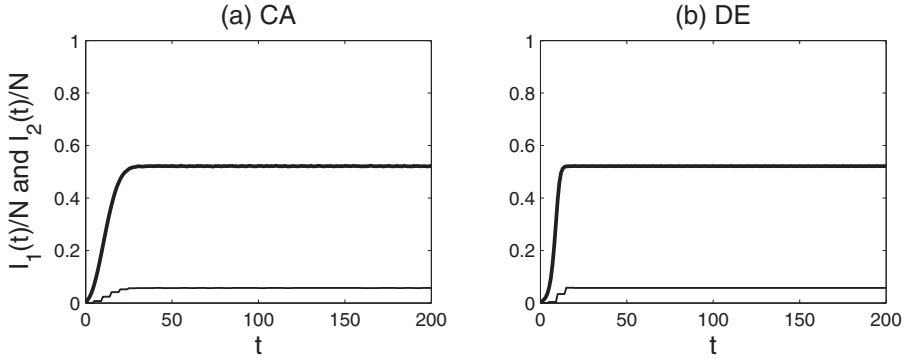

Fig. 1. Time evolutions of $I_1(t)/N$ (thick line) and $I_2(t)/N$ (thin line) obtained from the CA model (a) and from the DE model (b). In both approaches, the initial condition is $I_1(0)/N = 0.005(1-\mu)$ and $I_2(0)/N = 0.005\mu$. In (a), the curves represent average values of 20 simulations with the CA model by taking $\mu = 0.1$, $N = 40{,}000$, $\beta = 5$, $k_1 = k_2 = 0.3$, and $P_3 = P_4 = 0.5$. In (b), the curves were obtained by numerically solving the DE model after estimating the six parameters by using Eqs. (5)–(10). In this case, $aN \simeq 1.32$, $b \simeq 0.50$, $cN \simeq 2.00$, $d \simeq 0.50$, $pN \simeq 0.05$, and $qN \simeq 1.07$. In both approaches, $I_1/N \to 0.52$ and $I_2/N \to 0.06$.