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Letter to the Editor

On the effects of the spatial distribution in an epidemic model based on cellular automaton

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ABSTRACT

Several theoretical studies on disease propagation assume that individuals belonging to different groups regarding their health conditions are homogeneously distributed over the space. This is the well-known homogenous mixing assumption, which supports epidemiological models written in terms of ordinary differential or difference equations. Here, we consider that the host population infected by a contagious pathogen is composed by two groups with distinct traits and habits, which can be homogeneously mixed or not. The pathogen propagation is modeled by using an asynchronous probabilistic cellular automaton. Our main goal is to examine how a heterogeneous spatial distribution of these groups affects the endemic state. We noted that homogeneous distribution favors the occurrence of oscillations in the population composition. Surprisingly, we found out that the propagation dynamics of the heterogeneous distribution can also be described by a set of ordinary difference equations.

1. Introduction

Theoretical studies on epidemiology aim to predict and control the spreading of infectious pathogens. One of the key issues is to determine the evolution of the number of infected individuals as the time passes (e.g. Anderson and May, 1992; Keeling and Rohani, 2008).

Since the seminal work by Kermack and McKendrick (1927), many epidemiological studies based on ordinary differential or difference equations rely on the homogeneous mixing assumption (e.g. Anderson and May, 1992; Keeling and Rohani, 2008). Thus, these studies assume that spatial heterogeneities related to the geographical locations of individuals and to the topological structure of the network of social contacts can be neglected (e.g. Turnes and Monteiro, 2014). Obviously, this is a strong simplification.

Cellular automaton (CA) has been employed as an alternative approach to model the propagation of contagious diseases (e.g. Yakowitz et al., 1990; Boccara et al., 1994; Ahmed et al., 1998; Fuentes and Kuperman, 1999; Sirakoulis et al., 2000; Doran and Laffan, 2005; Monteiro et al., 2006; Slimi et al., 2009; Silva and Monteiro, 2014; Ilnytskyi et al., 2016; Chaves and Monteiro, 2017). In CA models, the spatial features of the host population are naturally considered, because the neighborhood of each individual can be easily taken into account.

We already used a probabilistic CA with asynchronous update to study the disease spread in a host population composed by two groups with distinct traits and habits (Chaves and Monteiro, 2017). Thus, each group had its own infection rate, cure rate, and level of social activity. In this former work; however, the two groups were homogenously mixed. Here, our goal is to examine how the endemic state is affected when one of these groups remains confined to a given spatial region of the CA lattice. Interestingly, we found out that even for this heterogeneous distribution, a mean-field approximation written as a set of ordinary difference equations (DE) can reproduce the dynamics observed on the CA lattice.

This paper is organized as follows. In Section 2, the CA model and the corresponding DE model are described. In Section 3, the results obtained from numerical simulations are presented. In Section 4, these results are discussed from an epidemiological standpoint.

2. The models

In the CA model (Chaves and Monteiro, 2017), the host population is represented by a square lattice with $n \times n = n^2 = N$ individuals with periodic boundary conditions. Each individual is in contact with its eight surrounding neighbors, which is usually called as Moore neighborhood of unit radius (e.g. Wolfram, 1994).

Assume that cure does not confer immunity to the corresponding pathogen. Therefore, a recovered individual is susceptible, in the sense that such an individual can be infected again. Thus, each individual, at each time step t, is in one of two states: susceptible S_i or infected I_i, with $i = 1, 2$ denoting its group. The group-1 is composed by $N_1 = (1 - \mu)N$ (with $0 \leq \mu \leq 1$) individuals and their states are updated at each time step. The group-2 is composed by $N_2 = \mu N$ individuals and their states are updated at β time steps (with $\beta > 1$). Note that the group-2 is formed by individuals with less active social life, as compared to the group-1.

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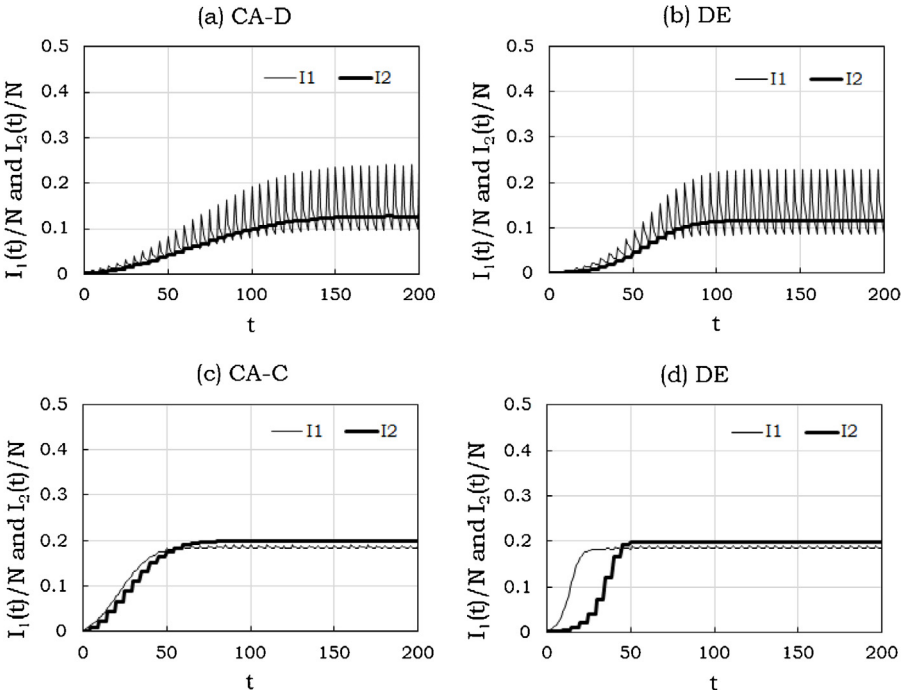

Fig. 1. In (a) and (c): $N = 40{,}000$, $\mu = 0.3$, $\beta = 5$, $k_1 = 0.2$, $k_2 = 1$, $P_1 = 0.9$, and $P_2 = 0.5$. In (b): $a_{11}N \simeq 1.23$, $a_{12}N \simeq 2.15$, $a_{21}N \simeq 2.45$, $a_{22}N \simeq 0.39$, $b_1 \simeq 0.89$, and $b_2 \simeq 0.50$. In (d): $a_{11}N \simeq 1.74$, $a_{12}N \simeq 0.06$, $a_{21}N \simeq 4.95$, $a_{22}N \simeq 0$, $b_1 \simeq 0.90$, and $b_2 \simeq 0.50$.

This SIS model is ruled by the following probabilistic rules (Chaves and Monteiro, 2017). The probability per time step of a S_i-individual being infected by an I_i-individual is $Q_i(v_i) = 1 - e^{-k_i v_i}$ (with $k_i > 0$); if this infection does not occur, the probability per time step of such a S_i-individual being infected by an I_j-individual (with $j \neq i$) is $Q_j(v_j) = 1 - e^{-k_j v_j}$ (with $k_j > 0$). In these expressions, v_i is the number of I_i-neighbors and k_i expresses the infectivity of the pathogen concerning the group-i. The probability of an I_i-individual being cured, per time step, is the constant P_i. These rules are applied for all individuals for $t = h\beta$ (with $h = 1, 2, 3$ and so on). For $t \neq h\beta$, the rules are applied by supposing that the population is composed only by the group-1 (that is, the group-2 is supposed to be inactive). Also, each individual remains in its group during the course of an epidemic.

Inspired by Kermack and McKendrick (1927), a mean-field approximation of our SIS model can be written as (Chaves and Monteiro, 2017):

$$\text{for } t \neq h\beta$$
$$I_i(t+1) = I_i(t) + \{a_{ii}[N_i - I_i(t)]I_i(t) - b_i I_i(t)\}(2-i), \quad i = 1, 2 \tag{1}$$

$$\text{for } t = h\beta$$
$$I_i(t+1) = I_i(t) + \sum_{j=1}^{2} a_{ij}[N_i - I_i(t)]I_j - b_i I_i(t), \quad i = 1, 2 \tag{2}$$

in which a_{ij} is related to the infections of S_i-individuals due to the contact with I_j-individuals ($i,j = 1, 2$) and b_i is related to the recovery of I_i-individuals. Observe that for $t \neq h\beta$, the number of I_2-individuals is not altered (that is, $I_2(t+1) = I_2(t)$).

An equivalence between CA and DE can be obtained by estimating the parameter values of this DE model from a CA simulation by using the following expressions (Chaves and Monteiro, 2017):

$$a_{ij} \simeq \frac{\Delta I_i(t)_{S_i \xrightarrow{I_j} I_i}}{S_i(t) I_j(t)}, \quad i, j = 1, 2 \tag{3}$$

$$b_i \simeq \frac{\Delta I_i(t)_{I_i \to S_i}}{I_i(t)}, \quad i = 1, 2 \tag{4}$$

in which $\Delta I_i(t)_{S_i \xrightarrow{I_j} I_i}$ is the number of state transitions $S_i \to I_i$ between t and $t+1$ due to infection by I_j (for $i, j = 1, 2$) and $\Delta I_i(t)_{I_i \to S_i}$ is the number of state transitions $I_i \to S_i$ between t and $t+1$ (that is, the number of I_i-individuals in t that will be recovered at $t+1$). Thus, by counting these state transitions in a CA simulation, the parameter values of the DE model can be computed in order to reproduce the dynamical behavior observed in such a CA simulation.

Note that in our study the values of a_{ij} (the infection rate constant) and b_i (the inverse mean duration of infection) can be affected by the social behavior of the individuals. This feature is supported by the following reasoning. SIS models are convenient to describe the propagation of infections caused, for instance, by the bacteria *Chlamydia trachomatis* and *Neisseria gonorrhoeae* (e.g. Kretzschmar et al., 1996; Jenkins et al., 2013). These sexually transmitted diseases are commonly treated with antibiotics, but cure does not confer immunity. In our study, distinct values of b_i in the DE model (P_i in the CA rules; in fact, $b_i = P_i$, as shown by Chaves and Monteiro, 2017) can reflect distinct levels of adherence to the treatment against the pathogen. For instance, $b_1 > b_2$ means that the group with more active social life is more likely to seek treatment (e.g., by taking antibiotics). Similarly, different values of a_{ij} in the DE model (k_i and k_j in the CA rules) can reflect differences in taking preventive measures against the pathogen propagation (e.g., the frequency of condom use).