Ecological Complexity 17 (2014) 40–45

Contents lists available at ScienceDirect

Ecological Complexity

journal homepage: www.elsevier.com/locate/ecocom

Self-sustained oscillations in epidemic models with infective immigrants

H.A.L.R. Silva [a], L.H.A. Monteiro [a,b,*]

[a] Universidade Presbiteriana Mackenzie, Escola de Engenharia, Rua da Consolação, n. 896, 01302-907 São Paulo, SP, Brazil
[b] Universidade de São Paulo, Departamento de Engenhariade Telecomunicações e Controle, Av. Prof. Luciano Gualberto, travessa 3, n. 380, 05508-900 São Paulo, SP, Brazil

ARTICLE INFO

Article history:
Received 23 April 2013
Received in revised form 23 July 2013
Accepted 7 August 2013
Available online 13 September 2013

Keywords:
Epidemic
Hopf bifurcation
Migration
Ordinary differential equation
Probabilistic cellular automaton
Recurrence

ABSTRACT

A relevant issue related to eco-epidemiological studies concerns the demographic mechanisms that can lead to self-sustained oscillations in the composition of a host population subject to infection. In particular, why does the prevalence of some contagious diseases oscillate over time? Here, we address this question by using susceptible-infective-recovered-empty models including migration of infective foreigners and variable population size. These models are described in terms of ordinary differential equations (ODE) and also in terms of probabilistic cellular automaton (PCA), in which each cell is connected to others either by a regular lattice or by a random graph favoring local contacts. Each cell in the PCA model can be either empty or occupied by a single individual. The amount of neighbors per cell affects the value of the basic reproduction number R_0, which is, in fact, a bifurcation parameter. We show that, by varying the amount of neighbors per cell (and consequently R_0), the number of infective individuals can start to exhibit periodic behavior, which corresponds to a Hopf bifurcation in the ODE model. This bifurcation gives rise to a self-sustained oscillation and it can only occur if the immigration rate of infective individuals is above a critical value. We also investigate how the sum of new infections, within the considered time window, depends on the number of neighbors per cell.

1. Introduction

The time evolution of many eco-epidemiological systems is modeled by differential equations, which can have periodic solutions. An attracting periodic solution, in this context, means that the sizes of the different groups composing the modeled system regularly oscillate in a permanent regime (e.g. Anderson and May, 1991; Malchow et al., 2008; Murray, 2003). This dynamic behavior can be a consequence of considering delays and/or periodic coefficients in the differential equations (e.g. Hethcote and van den Driessche, 1995; Olinky et al., 2008; Schwartz, 1992). However, self-sustained oscillations can also be found in constant-parameter models without any periodic input (e.g. Mukherjee, 2010; Pada Das et al., 2011; Sharp and Pastor, 2011).

An important issue in epidemiology is to understand why the prevalence of some contagious diseases oscillates in a periodic manner with time, while others chronically persist at an approximately constant level (e.g. Anderson and May, 1991; Bauch, 2008; Grassly and Fraser, 2006; Hethcote and Levin, 1989). Are the oscillations in prevalence caused by variations in demographic features of the host population (for instance, variations in the age structure, population size or in its spatial aggregation)? Are these oscillations stimulated by seasonal fluctuations in the disease propagation (for instance, fluctuations in the infection rate due to cyclic weather changes)? Are they an effect of attempts to control pathogen propagation (via, for instance, the implementation of regular vaccination campaigns)? Are they an outcome of immunity loss (that is, the prevalence oscillates because the corresponding infection does not confer permanent immunity after recovery)? Are they induced by antigenic drift due to mutations (which certainly contributes for the recurrence of influenza epidemics)? Questions like these have been addressed in theoretical works using cellular automata (e.g. Boccara et al., 1994; Johansen, 1996; Schimit and Monteiro, 2011; Slimi et al., 2009; Sun et al., 2010) and differential equations (e.g. Breban et al., 2009; Casagrandi et al., 2006; Castellazzo et al., 2012; Magal and Ruan, 2010; Roberts and Tobias, 2000; Zhang et al., 2012).

In this work, we propose an epidemic model based on probabilistic cellular automaton (PCA) to investigate the emergence of oscillations in the prevalence of contagious diseases. In

* Corresponding author at: Universidade Presbiteriana Mackenzie, Escola de Engenharia, Rua da Consolação, n. 896, 01302-907 São Paulo, SP, Brazil. Tel.: +55 11 2114 8711; fax: +55 11 2114 8600.
E-mail addresses: henrique.silva@mackenzista.br (H.A.L.R. Silva), luizm@mackenzie.br, luizm@usp.br (L.H.A. Monteiro).

1476-945X/$ – see front matter © 2013 Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.ecocom.2013.08.002

this model, the population size is not constant, the contact network is either regular or random, and the immigration of infective individuals is taken into account. Migration can be a host behavior crucial for the spreading of infectious diseases (e.g. Brauer and van den Driessche, 2001; Niro et al., 2012; Wallace and Fitch, 2008) and is the key process in our study.

In epidemic models with immigration, it is usually supposed that the immigrant flow is constant (e.g. Brauer and van den Driessche, 2001; Enatsu et al., 2012; Guo and Li, 2012; Li et al., 2006); therefore, the disease is never eliminated from the host population, which is not realistic. Here, we suppose that the density of infective individuals outside the spatial region represented by the PCA network is the same as in this region. Thus, the immigration rate is taken as proportional to the quantity of infective individuals found in the PCA network. Thanks to this (strong) assumption, the disease-free steady state exists, which is a solution that can be observed in the real world (e.g. Bartlett, 1957; Monteiro et al., 2006a).

By analyzing the mean-field approximation written in terms of ordinary differential equations (ODE), we derive conditions for a Hopf bifurcation to occur; that is, the birth of a limit cycle (an isolated closed trajectory corresponding to a periodic solution) in the state space of the ODE model. We also investigated how the number of neighbors per cell influences the total of new infections in a chosen time window.

This manuscript about oscillatory behavior in the spreading of a transmissible disease is organized as follows. In Section 2, the model based on PCA is described and results obtained from numerical simulations are presented. In Section 3, the results are analytically explained from the equivalent ODE model. In Section 4, the conclusions are discussed.

2. PCA model and numerical results

Models formulated in terms of PCA have been proposed for studying the propagation of contagious diseases (e.g. Ahmed et al., 1998; Doran and Laffan, 2005; Ferreri and Venturino, 2013; Fuentes and Kuperman, 1999; Sirakoulis et al., 2000; Yakowitz et al., 1990). In our model, individuals live in a square matrix formed by $n \times n = N$ cells with periodic boundary conditions. Each cell represents either an empty space (E) or an individual that can be in one of three states: susceptible (S), infective (I) or recovered (R). The time evolution of this SIRE (susceptible-infective-recovered-empty) model is ruled by the following set of probabilities of state transitions. At each time step, there is a probability $P_{S\to I}(v) = 1 - e^{-kv}$ of a S-individual being infected, in which v is the number of connections with distinct infective neighbors and k is a parameter expressing the pathogen infectivity (Schimit and Monteiro, 2009, 2012). Each I-individual has probability $P_{I\to R}$ per time step of becoming cured, probability $P_{I\to E}$ per time step of dying due to the infection, and probability $P_{E\to I}$ per time step of recruiting an infectious foreigner for occupying an empty cell (as noted in Section 1). At each time step, a R-individual may die with probability $P_{R\to E}$ and a S-individual may be born in an empty cell with probability $P_{E\to S}$. When individuals die at time step t, the corresponding cells become empty at $t + 1$. Note that the number of individuals can vary, but this number plus the amount of empty cells remains constant and equal to $N = n^2$. The states of all cells are simultaneously updated throughout a simulation.

Here, a regular lattice and a random graph are employed to represent the connections among the cells. In both topologies, the m neighbors of a cell pertain to the square matrix of size $2r + 1$ centered on such a cell, in which r is the neighborhood radius. In the regular lattice, the m neighbors are all the $(2r + 1)^2 - 1$ cells contained in this matrix. For instance, the case $r = 1$ including all 8 surrounding cells is known as Moore neighborhood of unit radius (e.g. Wolfram, 1994). In the random graph, from each cell, p connections start and arrive at other cells pertaining to its neighborhood matrix (two or more connections between the same two cells are allowed). The cells with radius equal to i compose the layer i, with $i = 1, 2, \ldots, r$, and the probability q_i of creating a link between a cell and any cell pertaining to the layer i of its neighborhood matrix is given by $q_i = 2(r + 1 - i)/[r(r + 1)]$. Observe that q_i diminishes with i. For instance, for $r = 2$, then $q_1 = 2/3$ and $q_2 = 1/3$; that is, the probability of connecting a cell to any of the 8 cells forming the layer $i = 1$ is 66.7%, and to any of the 16 cells forming the layer $i = 2$ is 33.3%. For $(2r + 1)^2 - 1 \gg p$ (that is, the number of available cells to establish a link is much greater than the number of links starting from each cell), the number m of neighbors of each cell is approximately $2p$. This network (Monteiro et al., 2006b; Schimit and Monteiro, 2009) is mainly locally connected like graphs called "small-worlds" (Watts and Strogatz, 1998), because the average clustering coefficient $\langle C\rangle$ is "high" (that is, $\langle C\rangle \gg m/N$) and the average shortest path length $\langle l\rangle$ is "small" (that is, $\langle l\rangle < \ln(N)$).

As examples, Figs. 1 and 2 exhibit the time evolutions of the normalized concentrations of S, I and R-individuals in the regular lattice for $k = 0.25$, $P_{I\to R} = 0.5$, $P_{I\to E} = 0.1$, $P_{E\to I} = 0.8$, $P_{R\to E} = 0.005$, $P_{E\to S} = 0.05$ and $n = 500$ (therefore, $N = 2.5 \times 10^5$), from the initial condition $S(0)/N = 0.4975$, $I(0)/N = 0.0025$ and $R(0)/N = 0$ (consequently, $E(0)/N = 0.5$; thus, at $t = 0$, half of the cells are unoccupied). In Fig. 1, $m = 80$ ($r = 4$) and the concentrations tend to a periodic oscillation; in Fig. 2, $m = 528$ ($r = 11$) and a stationary solution is attained. In a time window of 5000 time steps, the total of new infections (transitions $S \to I$) for $m = 80$ is 3.04×10^6; for $m = 528$, 2.36×10^6.

Table 1 presents the asymptotic behaviors in simulations with the regular network for $r = 1, 2, \ldots, 11$ (recall that $m = (2r + 1)^2 - 1$). Limit cycle is found for $48 \le m \le 224$; for $m \le 24$, a disease-free steady state is reached; for $m \ge 288$, an endemic steady state is observed when $t \to \infty$. Table 2 lists the behaviors in the random network with $r = 50$ and $m/2 \simeq p = 1, 2, 5, 10, 20, \ldots, 80$. In this network, the attractor is a limit cycle for $4 \le m \le 60$. These numerical results can be analytically explained by analyzing the

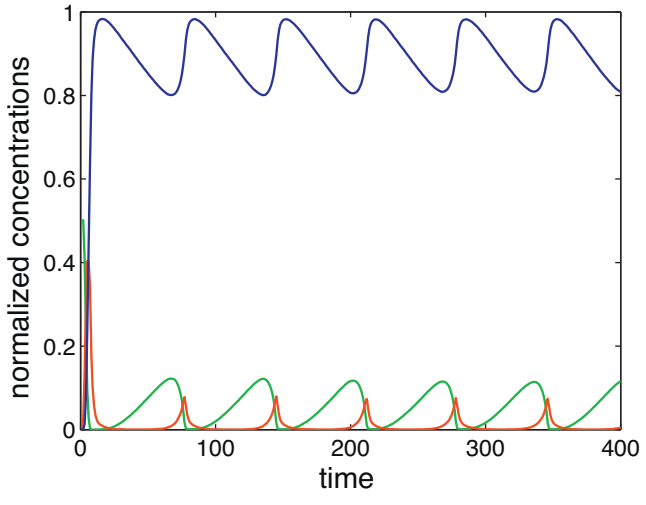

Fig. 1. Time evolutions of $S(t)/N$ (green), $I(t)/N$ (red) and $R(t)/N$ (blue) obtained from a PCA simulation in the regular graph ($E(t)/N$ is not shown; however, $E(t)/N = 1 - S(t)/N - I(t)/N - R(t)/N$). The parameter values are $k = 0.25$, $P_{I\to R} = 0.5$, $P_{I\to E} = 0.1$, $P_{E\to I} = 0.8$, $P_{R\to E} = 0.005$, $P_{E\to S} = 0.05$, $n = 500$ and $m = 80$ (that is, Moore neighborhood with $r = 4$). The initial condition is $S(0)/N = 0.4975$, $I(0)/N = 0.0025$, $R(0)/N = 0$ and $E(0)/N = 0.5$. The system exhibits self-sustained oscillation in permanent regime.