Expert Systems With Applications 48 (2016) 1–8

Contents lists available at ScienceDirect

Expert Systems With Applications

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

Epidemiological modeling with a population density map-based cellular automata simulation system

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ARTICLE INFO

Keywords:
Cellular automata
SEIR-IBM model
Epidemic spread model
Disease spread model
Mathematical modeling
Square lattice

ABSTRACT

We present a new numerical, two-dimensional cellular automata framework for simulation of the spread of an infectious disease in a region with non-homogenous spatial population distribution. For the simulation the real map of the population density in Poland is used, where the sources of the infection are located. Presented model is a combination of SEIR and IBM models complemented with additional factors like variable population density, death, birth and some stochastic parameters to reflect the more realistic population dynamics. In proposed model the states of individuals are tracked through time like in IBM model and the evolution of the whole system is described by SEIR transition function which determines how cells interact with their neighbours, influencing global behavior of the system. Presented model requires less complicated input than IBM models and is less expensive computationally. We explore influenza as the contagious disease in our map-based simulation. The results of the simulation show the spreading-rate of the disease and can be used to describe possible actions for preventing pandemic.

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1. Introduction

Epidemiology is an interdisciplinary field, in which many specializations like medicine, social sciences or economics are important components of the discussion. In our paper we tried to describe how epidemiology can be complemented with computational techniques combined with empirical data.

Traditionally, differential equation models have been used to describe the spreading of a contagious disease (Murray, 1993). Usually, an epidemic model belongs to one of the following types: *SIR, SIS, SEIR* or *SEIRS*, where particular letters denote one of the separate groups of the whole population (*S*usceptible, *I*nfective, *E*xposed and *R*ecovered), and the time evolution of these groups is modeled by the set of differential equations (Liu, Jin, & Liu, 2006; Milne, Fermanis, & Johnston, 2008; Pfeifer et al., 2008). However this mathematical approach has some serious drawbacks. It neglects external infections due to traveling individuals. It does not include variable susceptibility of individuals, and complex boundary and initial conditions (Achmed & Agiza, 1998).

Cellular automata (CA) can overcome these above drawbacks. CA is specified by lattice of cells which states can reflect initial and boundary conditions and the evolution in time of the CA can include the dynamics of the individuals. Initially CA was a model used to analyze the dynamics of micro-scale particle, such as thermodynamic rule, spin-glass model, etc. Wolfram showed that CA models are often based on rules that are simpler than complex mathematical equations and produce comparable results (Wolfram, 1983), therefore, CA may serve as a framework which can be applied for study complex natural phenomena in a way that is conceptually clearer and more realistic than conventional mathematical systems (Itami, 1994). Since then, CA has been also used by several researchers as an alternative method of modeling macro-scale phenomena, e.g. epidemics and building spatial and time discrete models of dynamical systems in which the contagious disease can spread (White & Sanchez, 2007; White, del Rey, & Sanchez, 2009). Alternatively to the above applications, CA can also be used to imitate the dynamics of similar, but not natural phenomena e.g. spreading of infectious diseases through internet: Peng, Wang, and Yu has used CA for building a model describing the propagation of worms in smartphones, where worms are self-replicating computer viruses, which can propagate through computer networks without any human intervention (Peng et al., 2013).

Most numerical models are based on the use of two-dimensional CA, where each cell stands for a square portion of the environment. Simulation region in two-dimensional CA consists of an array

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http://dx.doi.org/10.1016/j.eswa.2015.08.018
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Nomenclature

a	Fixed length in days of the exposed state (E)
b	Fixed length in days of the infective state (I)
c	Number of columns
c_v	Variation coefficient of the infection probability
C	Cellular space
$d(ij)$	Index of the closest dense cell at the Moore distance greater than one
E	Number of exposed individuals which are infected but not yet infectious
$E^t_{ij\|d}$	Number of exposed individuals in the (i, j)–th cell at the time t in the d–th day of this stage
f	Local transition function
i, i_0	Cell's row index
I	Number of infective individuals which are capable of transmitting the disease
$I^t_{ij\|d}$	Number of infective individuals in the (i, j)–th cell at the time t in the d–th day of this stage
j, j_0	Cell's column index
n^t_{ij}	Vector of numbers of individuals who move out of the (i, j)–th cell at the time t
N^t_{ij}	Number of individuals in the (i, j)–th cell at the time t
$N^t_{ij\to xy}$	Number of individuals commuting from the cell (i, j) to the cell (x, y) at the time t
Q	Finite set of states
p^t_{ij}	Probability that a susceptible individual in the (i, j)–th cell at the time t will be infected
r	Number of rows
R	Number of recovered individuals which are permanently immune
R^t_{ij}	Number of recovered individuals in the (i, j)–th cell at the time t
R_0	Basic reproduction number
s^t_{ij}	Vector representing (i, j)-th cell's state at the time t
$s^t_{ij\to xy}$	Vector of individuals commuting from the cell (i, j) to the cell (x, y) at the time t
S	Number of susceptible individuals which are able to contract the disease
S^t_{ij}	Number of susceptible individuals in the (i, j)-th cell at the time t
t	Time in days
V	Function mapping the cell to its neighboring cells
V^*	Updated (considering cells without population) function mapping the cell to its neighboring cells
β	Contact rate between individuals in a cell
δ	$1/\delta$ is the mean latent period for the disease (in SEIR model)
γ	$1/\gamma$ is the mean infectious period (in SEIR model)
μ	Birth and death rate (in SEIR model)
μ_b	New births per one individual per time step
μ_d	Natural deaths per one individual per time step
μ_{vm}	Mortality rate per time step, i.e. probability of a death of an infected individual
ϕ_h	Fraction of a healthy population commuting outside of their cell
ϕ_s	Fraction of an infected population commuting outside of their cell
ϕ_c	Fraction of commuters commuting outside of their neighborhood. There is an assumption that they commute to the closest dense cell (a cell with population above 50, 000) at the Moore distance greater than one

(lattice) of identical cells, which are endowed with the state (*S, I, E, R*) that changes with discrete steps of time according to a rule called transition function (White et al., 2009). The evolution of the state of a particular cell in CA depends on the states of the neighboring cells and these local interactions determine the spreading of the disease in the system. Usually it is supposed that the distribution of the population is homogeneous which means that all cells in the lattice have the same population and the whole size of the population is constant (White et al., 2009). Moreover, commonly used models assume that individuals are not mobile and the epidemic spread occurs across individuals fixed in their positions (Moreno, Gmez, & Pacheco, 2003; Yang & Wang, 2007).

Another approach presented Situngkir (2004) who used map-based simulation and simplified data of transportation through sea for modeling influenza disease in Indonesia. Jin and Liu (2006) used CA for simulations in spatial heteregenous hosts mixing and the natural birth rate and he compared the results with homogenous cases.

Many of existing models employ individual based model (IBM) in CA environment. In the model by Lopez, Burguerner, and Giovanini (2014) each individual of the population is represented by a cell in the lattice of CA . This way of modeling an epidemic situation allows to individually define the characteristic of each individual but it does not reflect the real density of the population. In the model by Sirakoulis, Karafyllidis, and Thanailakis (2000) the state of the cell is obtained from the fraction of the number of individuals which are susceptible, infected, or recovered from the disease . This approach shows the epidemic propagation during the population movement within the cells but does not specify anything about inhomogeneous and mixing of susceptible, infective, and recovered accordingly. Similar model extended for patchy population has been given by Athithan, Shukla, and Biradar (2014) who introduced heterogeneity and population movement into the model by Sirakoulis et al., (2000).

Our model combines presented solutions and introduces several new improvements. This model allows us to capture the individual heterogeneity as well as a realistic behavior of individual contacts. Presented solution is based on inhomogeneous distribution across lattice - we constructed simulation region according to the real population density map of Poland and the accuracy of map projection can be easily changed by changing the lattice resolution. In addition to heterogeneity we introduce new spatial and stochastic parameters to reflect the real processes related to population dynamics. In our model we approximate all other possible types of infection (infections in a cell, death and birth rate, mortality rate) by a semi-empirical mathematical parameters, which is an extension of models from Jin and Liu (2006) Situngkir (2004) White and Sanchez (2007) White et al. (2009). Additionally, we model the possibility of infection of an individual due to mobility of other infective individuals, which is in agreement with the model by Min-Hua, Duan-Ming, Gui-Jun, Yan-Ping, and Xin-Yu (2008), but we introduce daily commutes of individuals with variable ratio of commuters depending on whether they're healthy or infected. The state of the cell is obtained from the fraction of the number of individuals which are susceptible, infected, or recovered from the disease.

Consequently we present novel, complete SEIR model to simulate the epidemic spread based on CA. It is expected that incorporating several improvements i.e. real density of the population, parameters reflected the dynamics of population and different types of infection in one numerical system, we can get the results consistent with the behavior of a real epidemic.

2. Disease modeling frameworks

One of the most popular models of the infectious diseases is the classical *SEIR* model (Aron & Schwartz, 1984). In this model, the whole population is divided into four compartments which describe separated groups of individuals: susceptible which are able to contract

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