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Impact of time delay on the dynamics of SEIR epidemic model using cellular automata

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HIGHLIGHTS

- We presented two dimensional cellular automata model to simulate *SEIR* epidemic spread with time delay.
- The existence and stability of disease free equilibrium is examined.
- The behavioral patterns of disease dynamics are explored.
- The critical value of delay, beyond which there are notable variations in diffusion patterns, is computed.
- The basic reproductive number of the system and its relationship with the vital parameters has been investigated.

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ABSTRACT

The delay of an infectious disease is significant when aiming to predict its strength and spreading patterns. In this paper the *SEIR* (susceptible–exposed–infected–recovered) epidemic spread with time delay is analyzed through a two-dimensional cellular automata model. The time delay corresponding to the infectious span, predominantly, includes death during the latency period in due course of infection. The advancement of whole system is described by *SEIR* transition function complemented with crucial factors like inhomogeneous population distribution, birth and disease independent mortality. Moreover, to reflect more realistic population dynamics some stochastic parameters like population movement and connections at local level are also considered. The existence and stability of disease free equilibrium is investigated. Two prime behavioral patterns of disease dynamics is found depending on delay. The critical value of delay, beyond which there are notable variations in spread patterns, is computed. The influence of important parameters affecting the disease dynamics on basic reproduction number is also examined. The results obtained show that delay plays an affirmative role to control disease progression in an infected host.

1. Introduction

Epidemiology being a cornerstone of the public health, deals with the incidence and pervasiveness of diseases in large populations with focus on detection of their sources, causes and preventive measures against them. Mathematical models are very significant means for predicting, analyzing and controlling of an epidemic spread. Models such as *SI*, *SIS*, *SIR*, *SIRS*, *SEIRS* which exhibits the change in state among the susceptible (S), exposed (E), infected (I) and recovered (R) populations have been developed and studied in past [1–4], to understand the disease spread pattern and to anticipate the outcomes of introducing public health interference measures to minimize its spread.

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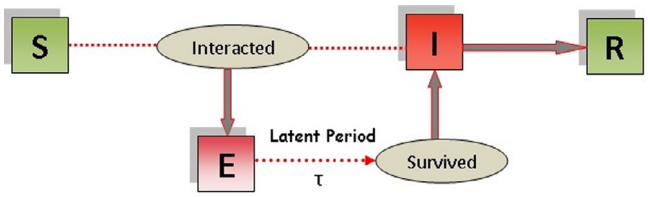

Fig. 1. Schematic of *SEIR* model with time delay.

More recently, researchers have shown their concerned on epidemiological models with latent or incubation period [5–8], since plentiful diseases, such as influenza, tuberculosis, have a passive course, during which the individual is said to be infected but not infectious. For example, measles has a latent period of 8–13—days, the incubation time for canine madness can be anything from a few months to years once the patient has the virus [9] and incubation period of diphtheria is 2–5 days (range, 1–10 days) [10].

Predominantly, the inclusion of the incubation period gives rise to models with the consideration of the difference in time between being exposed (E) to infection and becoming infectious (hence, *SEIR*) as well as in being infectious and dying ('delays'). So, the consideration of the occurrence of time delay (death during latency period) has a crucial biological meaning in epidemic models [9,11–15]. In epidemics with some fixed delay, the dynamic behavior of the disease at time t depends on the state of the system at time prior to delay period [16]. Yet, all these models are based on differential equation approach, hence, fail to capture spatial and temporal variations in the risk of transmission of infection with time delay and are not suitable for computational implementation. Moreover there is little understanding of how spread of *SEIR* epidemic with delay is affected by inhomogeneous population distribution, population migration and connections at local level.

An important attempt to achieve this objective has been the Cellular automata (*CA*) based approach, which reflect the heterogeneity of real surroundings by giving due consideration to individual properties and raising spatial and time discrete models of dynamical systems. *CA* have been successfully used as a substitutive method of modeling ecological phenomena, ecohydraulics systems, epidemics, earthquakes, tumor growth, fluid flow, biological pattern formation, civil development, avalanches, traffic jams, galaxy formation, parallel computers, image processing and much more [17–26]. Generally, when *CA* environment is applied in epidemiology, individual based modeling (*IBM*) technique is adopted [27,28]. In this technique, each individual of the population is represented by a cell in the cellular space and individual characteristics are reflected with lapse of time. Alternatively to the above technique, state of the cell can be obtained from the fraction of the number of individuals which are susceptible, infected, or recovered from the infection [23,29].

Understanding the critical impact of delay of an infectious disease and to assimilate structural parameters, lately, a more realistic cellular automata based *SEIRS* probabilistic model has been developed [30]. However, some infections, for example measles, rubella, diphtheria, confer long lasting immunity. Such infections give immunization upon recovery from infection, and individuals does not become susceptible again, accordingly abstracting the population into *SEIR* compartment [31].

Alternatively, this paper investigates the impact of delay on disease spreading by using a deterministic *CA* based *SEIR* epidemic model. This model imitate the spread of *SEIR* disease in both time and space at local level [23,32], considering time delay from exposed to infectious period. The state of a cell evolve over a sequence of discrete time steps on the basis of a set of deterministic rules based on the environment under consideration and from the portion of the number of individuals which are susceptible, exposed, infected, or recovered from the disease. Spatial and stochastic parameters like infections in a cell, death rate, birth rate, mobility of other infective individuals and connectivity within cells have been considered.

The rest of the paper is presented as follows: In Section 2 the proposed model is discussed in detail; stability of disease-free equilibrium is discussed in Section 3; Section 4 consists of simulations using relevant parameters and in Section 5 discussion based on conclusion has been included.

2. Description of the model

We consider a population, divided into four epidemiological classes, S, E, I and R with an assumption that the global ground where the epidemic is escalating stands for the two dimensional cellular lattice of the *CA*. The lattice is fragmented into identical square areas to capture the interactions taking place at local level. Each of these similar regions represents a cell of the *CA* with each cell having varying populations: varying denseness and varying crosswise cell mobility properties. Fig. 1 shows the movement within the classes in model with time delay. The exposed class originates due to interactions within susceptible and infected class. Only those members of exposed class who survives the latency period moves to infected class.

CA as discussed in [33,34] can be defined by the 4-uplet (L, K, V, f) where L is the lattice cellular space:

$$L = \{(i, j) : 1 \leq i \leq m, 1 \leq j \leq n\}. \tag{1}$$

Here (i, j) represents the location in the ith row and jth column; m and n are row and column count respectively. K is the finite state set whose members are the all possible states of the cells; V is the function affiliating a cell to its neighborhood. The neighborhood function $V = V_{i,j}$ with a distance of r units for the cell (i, j) is defined as

$$V = \{(\alpha, \beta) \in L \setminus \{(i, j)\} : |i - \alpha| \leq r \wedge |j - \beta| \leq r\}. \tag{2}$$