

Towards Computational Epidemiology : Using Stochastic Cellular Automata in modeling spread of diseases

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Abstract

In this paper, we propose the use of the Stochastic Cellular Automata paradigm to simulate an infectious disease outbreak. The simulator facilitates the study of dynamics of epidemics of different infectious diseases, and has been applied to study the effects of spread vaccination and ring vaccination strategies. Fundamentally the simulator loosely simulates the SIR (Susceptible Infected Removed) and SEIR (Susceptible Exposed Infected Removed) models. The global stochastic cellular automata presented here incorporates the underlying distances for interactions and can be extended to include the demographic characteristics of a region.

1 Introduction

Nowadays, the problem of emerging and re-emerging diseases like influenza and SARS, have caused increased attention towards public health in general and epidemiology specifically. With the ever-increasing population and ability to travel longer distances in shorter time, the spread of communicable diseases in a society has been accelerated [16, 17]. Growing diversity of the population, and globalization are leading towards increasing interaction among individuals. Constant exposure to public health threats is raising people's concern and necessitates pro-active action towards preventing disease outbreaks. Further, greater emphasis on infectious diseases and epidemics is rooted in the imminent threat arising from bioterrorism. As a result, Public Health professionals have been focusing on identifying the factors in the social, physical and epidemiological environment which aid the faster spread of diseases.

As the significance of Public Health is being recognized, the role of epidemiologists has become more prominent. Epidemiology deals with the study of cause, spread, and control of diseases and the goal for epidemiologists is to implement

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mechanisms for surveillance, monitoring, prevention and control. Epidemiological studies may require large data sets of disease outbreaks which are often spatially and/or temporally distributed. It is in fact ironic that, for epidemiologists to study the dynamics of different diseases, it is vital for an outbreak to occur. Epidemiologists have been studying and analyzing data sets using primarily statistical tools. In the vast variety of infectious diseases, specific expertise is needed for every disease. In order for the epidemiologists to prepare for a sudden outbreak of an infectious disease or a bioterrorism attack, the need for simulation arises. Hence, it is imperative to develop new tools that take advantage of today's computational capabilities, and help epidemiologists to analyze and understand the progression of an epidemic in a given geographic region with specific demographic characteristics. The computational tools also enhance the quality of information, accelerate the generation of answers to specific questions and facilitate prediction. For this purpose we propose the use of Stochastic Cellular Automata paradigm to simulate an infectious disease outbreak. The development of this tool brings together expertise in statistics, mathematics and high performance computing.

1.1 Cellular Automata

In the domain of computational tools, the Cellular Automata paradigm has been used for several decades [14]. Nevertheless, in the field of modeling epidemics, this paradigm has rarely been utilized to its full potential [1, 10, 14, 8]. A cellular automata as defined by Lyman Hurd, is a discrete dynamical system, where space, time, and the states of the system are distinct [15]. An automaton is best exemplified by representing a point in space as a cell C_i surrounded by other cells, thereby defining the neighborhood H_i of C_i . The cells are most often arranged to constitute a regular spatial lattice. See Figure 1.

In general, we can define a cellular automaton of any dimension. Most frequently, one, two, and three dimensional automata have been used in science. For a one dimensional automaton, $|H_i| = 2$, that is, cell C_i has a left and a right neighbor (disregarding any edge conditions). A two dimensional automaton is best represented as a regular spatial lattice or grid. Here, cell $C_{i,j}$ is surrounded by cells that form its neighborhood $H_{i,j}$. Traditionally, there are two possible sizes of $C_{i,j}$'s neighborhood in a two dimensional automaton, namely, $|H_{i,j}| = 4$ in the *von Neumann neighborhood* and $|H_{i,j}| = 8$ in the *Moore neighborhood* [15]. Specifically, the cells in the *von Neumann neighborhood* are $C_{i+1,j}$, $C_{i-1,j}$, $C_{i,j+1}$, $C_{i,j-1}$. The *Moore neighborhood* is defined by cells $C_{i+1,j}$, $C_{i-1,j}$, $C_{i,j+1}$, $C_{i,j-1}$, $C_{i+1,j+1}$, $C_{i-1,j-1}$, $C_{i-1,j+1}$, $C_{i+1,j-1}$.

At a particular time t , each cell C of the automaton is said to be in a specific state $s(t)$, which depends on the specific application. $s(t) \in S$ where S is state space of the cellular automaton. In the simplest case, cells are assuming binary states $(0,1)$. For more complex applications, any size of discrete (and even continuous)

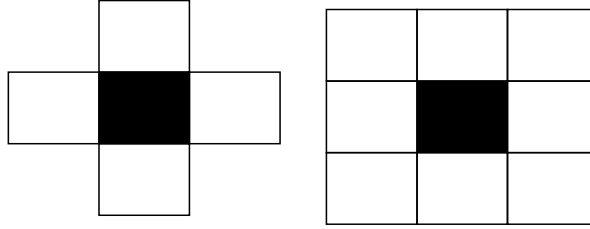


Figure 1: von Neumann and Moore Neighborhood

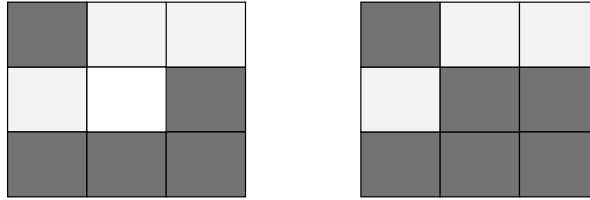


Figure 2: Cellular Automata Update from time step $t-1$ to t

state space can be defined. The state of cell $C_{i,j}$ at time t is determined by the state of its neighborhood $H_{i,j}$ at time $t-1$. Formally, $s_{i,j}(t) = F(H_{i,j}(t-1))$. Here, the function F can be considered the rule that determines how a particular state configuration of $H_{i,j}$ determines the next state of $C_{i,j}$. For a deterministic cellular automaton, the initial states of each cell and the update rule F completely describes the automaton. During a time step t , a new state $s(t)$ is computed for every cell as described above. An initial state configuration will hence evolve, thus representing a dynamic system.

As shown in the Figure 2 the state of the center cell changes to a state, which is in majority among the cells in the neighborhood. The update rule determines the deterministic or stochastic behavior of a CA. Stochastic behavior is seen by probabilistic update rules in non-deterministic state transitions. For example in a stochastic cellular automata, for every update a cell can choose randomly from a set of update rules or for a particular update rule randomly choose from a set of states to make the transition to. This results in stochastic transition.

Our efforts to design and implement a Cellular Automata based simulator has been necessitated by the need to study the dynamic of spread of a vast number of infectious diseases. This paper focuses on the design and evaluation of EPI-SIM, a global disease outbreak simulator. The following section summarizes some of the research effort in modeling disease epidemic and highlights principle approaches. The design of EPI-SIM is discussed in Section 3. Section 4 discusses the Geo-Spatial model and the approach towards the global model to account for different demographics. Section 5 discusses the modeling of the SCA. Section 6 concludes the paper with a summary.

2 Related Work

Most of the work in modeling infectious disease epidemics is mathematically inspired and based on differential equations and SIR/SEIR model [3]. Differential equation, SIR modeling rely on the assumption of constant population and neglect the spatial effects [5, 6]. They often fail to consider individual contact/interaction process and assume populations are homogeneously mixed and do not include variable susceptibility. Considerable research has been conducted in SIR(Susceptible, Infectious, Recovered) modeling of infectious diseases using a set of differential equations. Both partial and ordinary differential equation models are so deterministic in nature that they neglect the stochastic or probabilistic behavior [8]. Nevertheless, these approaches/models have been shown to be effective in regions of small population [8]. Other approaches for modeling disease epidemics have been using mean field type (MFT) approximations [12]. Even though the MFT models are similar to the differential equations, they add a probabilistic nature by adding different probabilities for the mixing among individuals. According to Boccara [5] mean field approximations tend to neglect spatial dependencies and correlations and assume that the probability of the state of cell being susceptible or infective is proportional to the density of the corresponding population. This approach relies on the quantitative measures to predict local interaction. Boccara and Cheong [5] study the SIS model of spread of infectious disease in a population of moving individuals, thereby introducing non-uniform population density. In every update the cells take up a state of being either susceptible or infectious and randomly choose a cell location to move to.

Ahmed *et al* [2] model variations in population density by allowing cyclic host movement. Other approaches in modeling variable susceptibility of the population, have been done by inducing immunity in the population. Ahmed *et al* [1] introduce incubation and latency time, and suggest that the parameters have an accelerating impact on the spread of a disease epidemic. Nevertheless, the underlying assumption is spontaneous infection of individuals. Boccara and Cheong [6] concentrate on SIR epidemic models and take into consideration the fluctuation in the population by births and deaths, exhibiting a cyclic behavior with primary emphasis on moving individuals. Di Stefano *et al* [8] have developed a lattice gas cellular automata model to analyze the spread of epidemics of infectious diseases. The model is based on individuals, where individuals can change their state independent of others and can move from one cell to other. However, this approach does not consider the infection time-line of latency, incubation period, and recovery which have been shown to be important to model a disease epidemic.

3 EPI SIM - A Disease Outbreak Simulator

In our model the basic unit of cellular automata is a cell, which may represent an individual or a small sub-population. Initially, we have designed the model to use the Moore (8) neighborhood. Each cell can be characterized with its own probability for risk of exposure, probability of contracting the disease and state. Unlike the SIR model, every cell comes in contact with the cells in its defined neighborhood.

The time-line for infection that we consider is shown in Figure 3. The Moore neighborhood leads to a saturation of neighbors and limits the speed of spread of a disease. This model is also restricted in modeling population demographics and travel patterns. Section 4 and 5 discusses a modification to this model which leads to a global neighborhood eliminating this limitation. The following sections discuss the definitions, features and rules of the model and simulator. The time-line for infection that we consider is shown in Figure 3.

3.1 Definitions

The following section describes the different states a cell can attain and parameters used in simulation.

States of a cell

State S for Susceptible is defined as the state in which, the cell is capable of contracting a disease from its neighbors. In the infectious state, I the cell is capable of passing on the infection to its neighbors. In the recovery state, R the cell is neither capable of passing on the infection nor capable of contracting the infection.

Parameters for the simulator

Infectivity ψ , at any given time is defined as the probability of a susceptible individual to become infectious, if one of cells in the neighborhood is infectious. Latency λ is defined as the time period between the cell becoming infected and it becoming infectious. Infectious period θ is the period during which the infected cell is capable of spreading the disease to other cells. Recovery period ρ is defined as the time period the cell takes to recover, wherein it is neither capable of passing on the infection nor capable of contracting the infection.

3.2 Rules for spread of disease

The rules described below determine the state transitions of individual cells in the CA.

1. A cell's state changes from susceptible $S \rightarrow L$ Latent when it comes in contact with an infected cell in its defined neighborhood. The cell acquires the disease from the infected neighbor based on the probability of given by the parameter of infectivity ψ . The cell remains in the latent state for the number of time steps (updates) as defined by the parameter latency λ .
2. The state of the cell changes from latent $L \rightarrow I$ Infectious after being in state L for a given λ . In this model we assume for simplicity, that every cell exposed to the pathogen, will become infectious. In state I , the cells are capable of passing on the infection to neighborhood cells. For example for a disease \mathcal{D} , with $\lambda = 2$ units the cell will enter the infectious state I after two time steps.

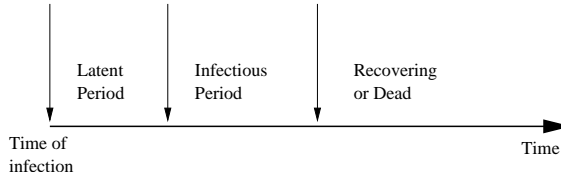


Figure 3: Infection Time-line

3. After a time period, defined by the infectious period θ , the state of the cell changes from infectious I to recovered or removed R . Once the cell enters the state R , the cell is no more capable of passing on the infection.
4. From the state R , the cell's state changes back to either susceptible S or it remains in state R , signifying complete immunity. The 'healing mode' turned on determines the transition from state R to state S and vice versa.

3.3 Simulating different disease characteristics

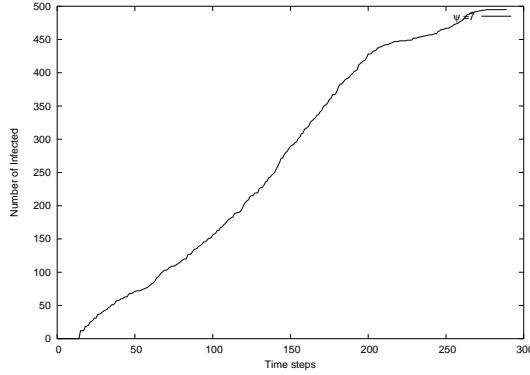
While modeling an epidemic, the parameters that are considered important include neighborhood radius, contact between individuals, infection probability (variable susceptibility), immunity, latency, infectious period and recovery period. The simulator is highly parameterized to let the user change and modify the above parameters. The neighborhood of every cell can be changed from a size 8 to a size 4 depending on the region being simulated and the contacts among the individuals of the region. As mentioned above, the infection probability represented as infectivity ψ is a significant parameter for the spread of a disease. In our model, ψ is based on the virulence of the disease and contact rate among individuals. For some diseases, individuals attain lifetime immunity after being infected, while for disease like the common cold, individuals attain temporary immunity. Thus, to take this fact into consideration, the simulator has a feature of healing mode. With the healing mode enabled the simulation is executed in a mode that forces cells to turn into susceptible after the recovery state and with healing turned off, the cells attain complete lifetime immunity.

As mentioned above, the infection time-line is also an important factor in modeling a disease epidemic. Thus latency λ , infectious θ , and recovery ρ are all expressed as uniform time units, for example, latency of two days, can be represented as $\lambda=2$. The simulator allows the user step through the simulation, or execute it continuously.

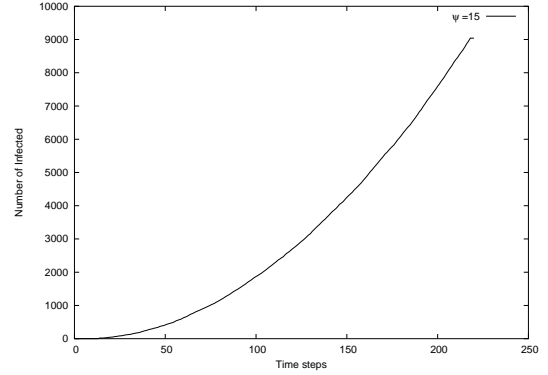
3.4 Experiments and Results

In the following we discuss the experiments conducted with the model described above. Experiments were conducted on 140 by 140 grid cellular automaton with different values for infectivity, latency, infectious period.

The results in this section represent the mean over multiple random experiments. The analysis of results in this section have been conducted with reference to



(a) Number of infected individuals over time for $\psi = 7$.



(b) Number of infected individuals over time for $\psi = 15$.

Figure 4: Variation in spread for different ψ 's

the above definitions.

Analysis of variation in infectivity ψ

As mentioned earlier, ψ is an important factor in the analysis of spread of a disease. Figures 4(a) and 4(b) show the results of executing the simulation of a disease \mathcal{D} with ψ of 7 and 15 respectively. In Figure 4(a) the number of infected reached around 500 in 300 time steps, whereas in Figure 4(b) with ψ of 15 the number of infected reached around 10000 in 300 updates. Figure 5, shows the comparison of the different values of $\psi = (7, 10, 12, 15)$. The graph for ψ of 15 is significantly steeper as compared to other values of ψ . This experiment was conducted with the $\lambda = 2$ units, $\theta = 3$ units, $\rho = 2$ units and healing option was turned off. This experiment exemplifies the sensitivity of the parameter ψ .

Effects of Vaccination

Vaccination has contributed significantly towards the eradication and reduction of effect of many infectious diseases [7]. The following experiments were conducted on the simulator by vaccinating about 5% of the population at random and infecting few cells. Figure 6, shows the growth of infected individuals in a vaccinated and non vaccinated population. Figure 6 indicates the increase in number of infected individuals in a population with only 5% of the population being vaccinated, is considerably less as compared to a non-vaccinated population.

We study the effects of spatial distribution of population, by vaccinating a part of the population using the spread and ring vaccination. Every time a new vaccine is discovered, the question arises as to how should the vaccine be distributed to minimize the spread of a disease and maximize the effect of vaccination. In the following experiment we compare the random vaccination, which is also known as uniform strategy [9], and ring vaccination. The doses of vaccine available is often

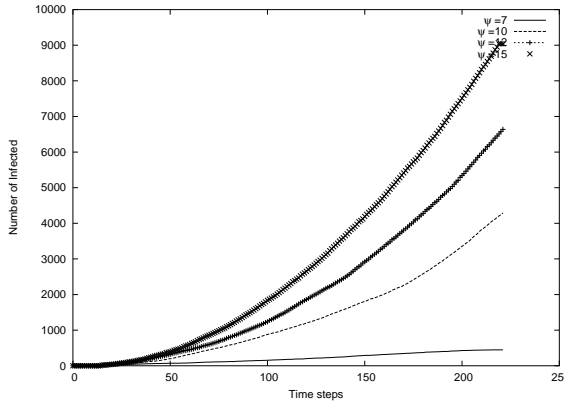


Figure 5: Comparison of growth rate for ψ' s of 7,10,12,15 is shown.

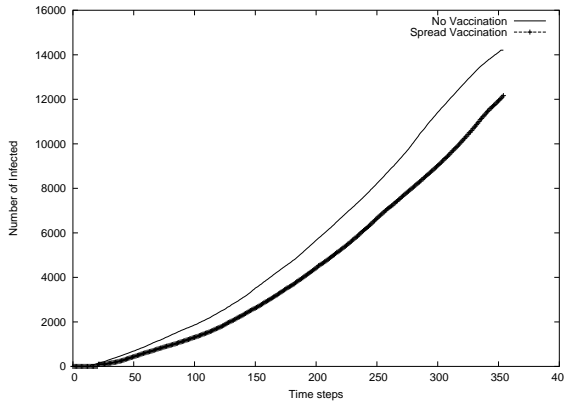


Figure 6: Comparison of Random Vaccination (5% of population was vaccinated) and no Vaccination

limited. Thus for the purpose of the experiment we consider N doses of vaccine at our disposal, where N is about 5% of the population. For random vaccination, the N doses of vaccine, are randomly distributed to individuals in a population, independently. For the ring vaccination, individuals are vaccinated in a ring surrounding an area containing the index case of the disease epidemic. The width and circumference of the ring clearly depend on N . Many more individuals are infected when using random vaccination as compared to using ring vaccination as indicated by Figure 7. This experiment validates the result shown by Fuk \acute{s} and Lawniczak in [9]

3.5 Conclusion

The classical simulation methodology described suffers from saturation of a limited neighborhood. A neighborhood of 8 cells, quickly saturates and thus reduces the number of susceptibles. In such a situation the increase of infectivity parameter plays no role and has the same effect on the spread of the disease. Also, the need

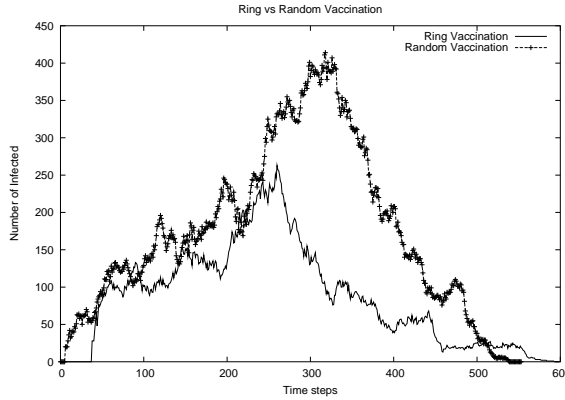


Figure 7: Comparison of Random and Ring Vaccination with N doses of vaccination available for both strategies

to simulate a disease, where an infective can spread the disease to an extended neighborhood in one time step, can not be simulated. The movement of people, migration, or travel is not considered. Some of the models discussed in the literature, deal with movement of individuals from one cell to another in the defined neighborhood. Clearly as discussed above they are deemed to be hampered by early saturation.

4 Geo-spatial Model - Extending the neighborhood

The Geo-spatial model is designed for simulating a global outbreak of a disease in an environment with global interaction. Even for this model, the basic unit of CA is a cell, may represent an individual. The neighborhood defined for this model is global i.e. each cell has $n-1$ neighbors in a $n \times n$ grid.

For the model, the definitions for the states of cell and parameters for the simulation are same as for the SIR model discussed earlier. This model has an additional parameter of contact rate defined below.

The **Contact rate** parameter defines the number of contacts made by an individual per time unit. Instead of having the same contact rate parameter for every cell in the lattice, for simulation purposes this parameter CR is Poisson distributed over time.

1. In a time step a cell chooses k cells at random from the pool of $n-1$ neighbors, where k is the contact rate defined for that cell. Thus the cell has now established contacts with k cells.
2. Once a contact has been established between cell a and cell b , depending on the virulence of the disease defined by the infectivity parameter, cell a can pass the infection to cell b if cell b is in a susceptible state S . If cell a is not infected currently and cell b is infected then cell a can acquire the infection from b . Thus the infection can be transmitted bi-directionally.

4.1 Towards a Global model : Accounting for different Demographics

The traditional CA model described above may be used for simulating diseases over small regions with local interaction and global interaction respectively. As mentioned before, the model does not take into account the demographics of the region and may not be accurate for simulating disease spread over large geographic regions because of the neighborhood constriction posed by them. Hence we have designed a global stochastic cellular automata with demographics will facilitate the understanding of effects of different demographics, the population density, socio-economics of a region and culture. It can also be used effectively for investigating different vaccination strategies and understanding the effects of travel.

For simulating the spread of disease in such an environment, contacts need to be established between cells. In this model, every cell has a chance of interacting with every other cell in the environment, but the probability of contact varies based on what is defined to be the interaction coefficient. The interaction coefficient reflects the factors which are important when considering contact between two cells. Such as distance, population and other demographics and socio-economic factors. In the present model the interaction coefficient is based on the distance between cells. In the future models, population and other factors will also be considered.

The **neighborhood** for a global SCA is defined using a fuzzy set:

Definition 1 : Fuzzy Neighborhood

The set $F \subset S$ where S is a set of all the cells

$F: \{ \langle s, p \rangle | s \in S, 0 \leq p \leq 1 \}$

$\langle s, 1 \rangle$: Total/Complete membership

$\langle s, 0 \rangle$: No membership

Interaction Coefficient i for a particular cell is defined as the strength or likelihood of interaction between two cells. As mentioned above the interaction coefficient depends on various factors which can affect contact between two subjects, such as individuals or cells. However, for this model we presently consider the distance between cells as the factor influencing the interaction coefficient. It is calculated as the reciprocal of the Euclidean distance between the cells. Equation 1 shows the calculation for interaction coefficient based on distance. Experiments were conducted on calculating the coefficient based on distance and population as derived from the GIS gravity model. Equation 2 shows the calculation of interaction coefficient based on distance and population.

$$i_{C_{i,j}, C_{k,l}} = \frac{1}{\sqrt{\langle i - k \rangle^2 + \langle j - l \rangle^2}} \quad (1)$$

$$i_{C_{i,j}, C_{k,l}} = \frac{P_{C_{i,j}} \times P_{C_{k,l}}}{\sqrt{\langle i - k \rangle^2 + \langle j - l \rangle^2}} \quad (2)$$

State of infection δ is defined for every cell as a number between 0 and 1, indicating the level of infection present in the cell. 0 indicates not infected, 1 indicates

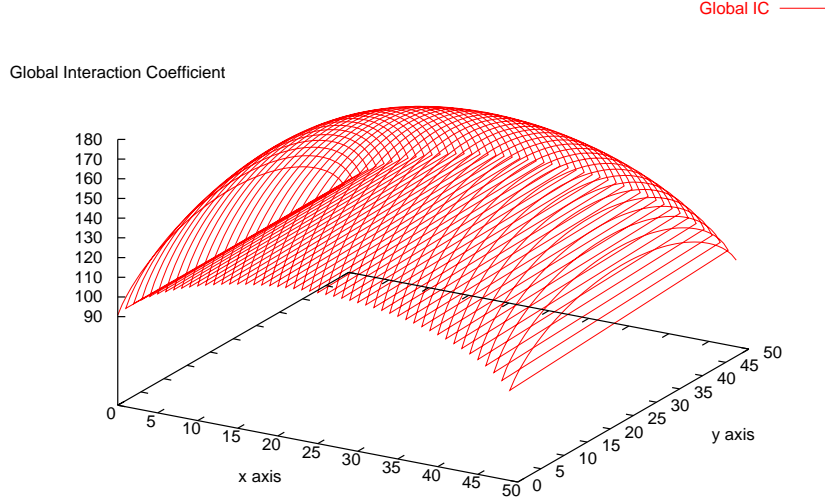


Figure 8: Global Interaction Coefficient based on distance

fully infected. This parameter is used in order to determine whether the subject or group is capable of transmitting the infection or not.

Global interaction coefficient Γ of cell $C_{i,j}$ is the sum of all the individual (n-1) interaction coefficients of the cell. This coefficient represents the overall interaction of the particular cell. It is different for every cell based on their position. Figure 8 shows the Global interaction coefficient based on distance for every cell on a 50×50 grid. Naturally, the center cell has the maximum interaction coefficient. Figure 9 show the Global interaction coefficient based on distance and population for every cell on a 50×50 grid. Experiment was conducted with two cities of high population. The Figure depicts that population dominates over distance. This however may not hold true if the interaction coefficient incorporates measures of population and other demographic values.

The global interaction coefficient and the interaction coefficients are calculated based on the distance. As the distance between the cells reduces, the interaction coefficients increases, which indicates greater chances of interaction between them. Equations 3 and 4 show the calculation of the Γ based on distance and distance and population respectively.

$$\Gamma_{C_{i,j}} = \sum_{\forall C_{k,l} \neq C_{i,j}} \frac{1}{\sqrt{\langle i-k \rangle^2 + \langle j-l \rangle^2}} \quad (3)$$

$$\Gamma_{C_{i,j}} = \sum_{\forall C_{k,l} \neq C_{i,j}} \frac{1}{\sqrt{\langle i-k \rangle^2 + \langle j-l \rangle^2}} \times P_{C_{i,j}} \times P_{C_{i,j}} \quad (4)$$

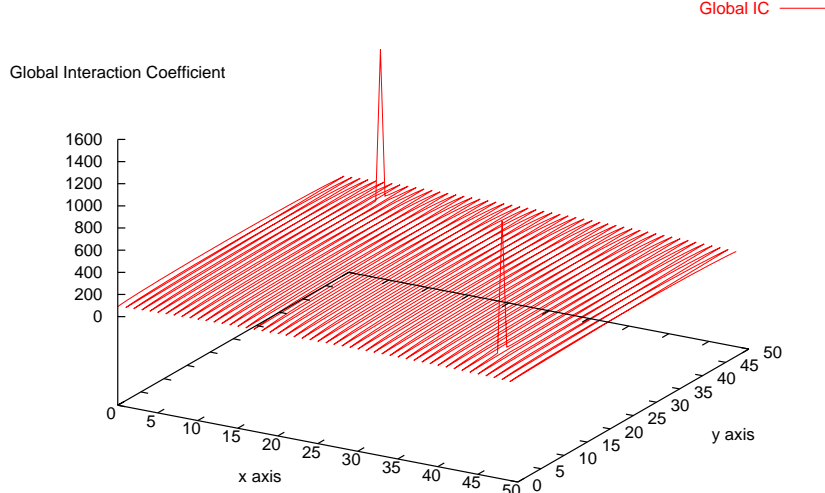


Figure 9: Global Interaction Coefficient based on distance and population

The **infection factor I** is calculated as a fraction of the interaction coefficient to the global interaction coefficient Γ , for every cell to cell interaction. It is also based on the virulence of the disease and the state of infection of the infecting agent. Referring back to definition 1 the parameter p is the ratio of interaction coefficient to the global interaction coefficient.

$$I_{C_{i,j}} = \sum_{\forall C_{k,l} \neq C_{i,j}} \frac{i_{C_{i,j},C_{k,l}}}{\Gamma_{C_{i,j}}} \times \delta_{C_{k,l}} \times \psi \quad (5)$$

5 Modeling the global stochastic model

In the global SCA model, the population is considered to be uniformly distributed over the grid. Each cell is considered as an individual, with certain structural properties. As defined in earlier models, contact rate for each cell is drawn from a Poisson distribution of parameter contact rate CR based on an average contact rate provided by the user. To model the interactions between cells, we present two different algorithms for selection of contacts.

As mentioned earlier and as derived by the gravity model, the probability of interacting with cells closer is greater than the probability of interacting with cells farther. Considering a $n \times n$ grid with n^2 cells, each cell has $n - 1$ possible interactions. The simulator selects k interactions from the possible $n - 1$ as controlled by the exponential decay function, which yields an inefficient computational solution. When n is considerably large as to 10^3 cells then the amount of data can approach a terabyte range. Thus to solve the n^2 bottleneck problem we propose the following algorithms. We assume that the probability decays exponentially with distance. See

Equation 6. The parameter λ is a multiplicative factor used to scale the distance parameter to match the distances on the grid.

$$P = \lambda \times e^{-\lambda d} \quad (6)$$

In order to calculate the exponential random variant, we draw a random number $R \in U[0,1]$ and transform equation 6 to obtain d . Once the distance is found two different ways are used to choose contact cells.

5.1 Threshold Based Algorithm

For the threshold method, cells are chosen at random and a rejection method is used. The cells with a distance less than the threshold (calculated) distance are chosen, otherwise rejected. The drawback of the method is the number of rejections that might occur, until a cell at a distance less than the threshold is chosen. If a new distance is calculated after every rejection then the number of calls to the random number generator increases drastically, which may hamper the performance of the system. Figure 10 demonstrates that a large portion of cells chosen are at smaller distances. However, for this algorithm the number of calls to the random number generator is large, because of uniformly selecting from $n-1$ cells until a cell is found at a distance less than d .

The following pseudo code describes the threshold based algorithm for a cell x

```

1 choose distance d from the exponential decay function
2 do
3   choose a cell y at random uniformly
4   calculate the distance of y from x
5 repeat until cell distance leq d
6 establish contact with y
7 repeat above steps until expected number of contacts are established.
```

Clearly, this method is based on trial and error. Consequently we propose the bounding box algorithm, where in instead of choosing from $n-1$ cells the choice is restricted by the bounding box, thus reducing the number of calls to random number generator. The bounding box based algorithm is described in the next section.

5.2 The Bounding Box Algorithm

With the cell as the center a virtual bounding box is drawn around the cell at a distance d . Cells within this bounding box are chosen randomly for contact. For every new contact a new d is calculated. Experiments were conducted by generating 5000 contacts. Figure 11 shows the number of contacts generated for different intervals of distances, indicating that more contacts are generated with cells closer to the subject cell.

The following pseudo code describes the algorithm for a cell x

```

1 choose distance d from the exponential decay function
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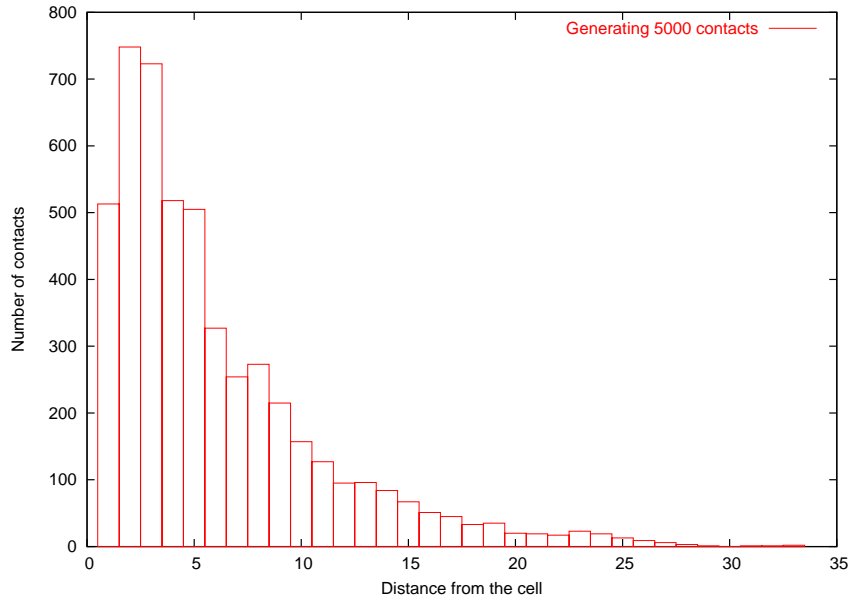


Figure 10: Frequency of distances based on threshold

- 2 draw a bounding box of distance d from x on all four sides
- 3 calculate the boundaries of the box
- 4 choose a cell y at random within the box boundaries
- 5 establish contact with y
- 6 repeat above steps until expected number of contacts are established.

6 Summary

This paper describes a disease outbreak simulator using the stochastic cellular automata paradigm. Three different models, have been discussed. The classical CA model, poses a limitation due to saturation of the cell neighborhood. The results show the variation in the spread of the disease for different parameters of infectivity ψ . The simulator also facilitates the study of different vaccination strategies. The global model supports the simulation of disease spread in an environment with global interaction including travel and migration over large geographic regions. However, the global model poses the problem of choosing contacts from a global neighborhood in an efficient way, which is computationally feasible. Although the global model is capable of simulating disease spread over large geographic regions, new computational methods need to be developed to incorporate demographics into the model. The experiments conducted on the global simulator validates the algorithm for effectively choosing contacts from a global neighborhood based on distance. While computational tools to facilitate surveillance, monitoring, prevention and control of dynamics of different diseases are being developed, the current simulator has proven as valuable tool to study the dynamics of different diseases.

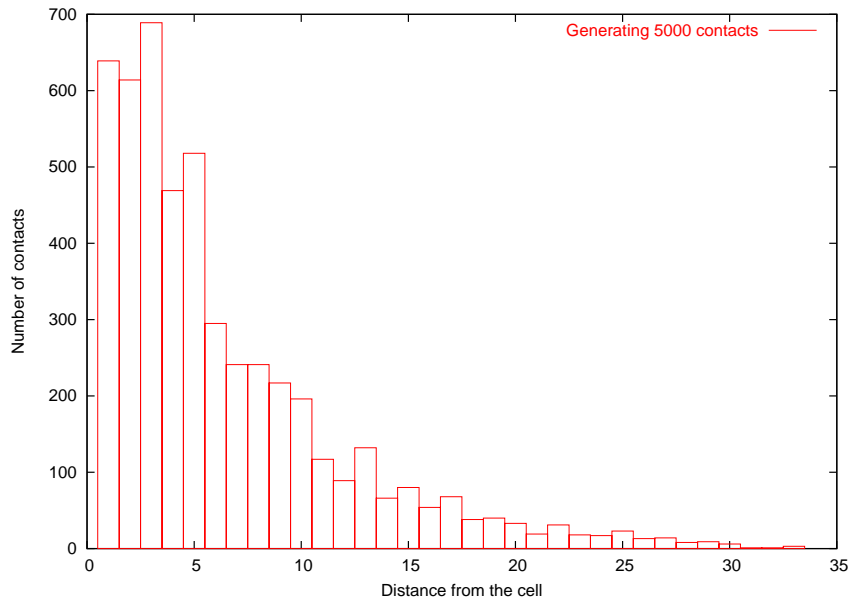


Figure 11: Frequency of distances based on bounding box

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