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# MODELLING POPULATION HETEROGENEITY IN EPIDEMICS USING CELLULAR AUTOMATA

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**Abstract.** Compartmental models are very popular in epidemiology. One of the reason for such popularity is the excellent results obtained when the populations satisfy the building hypotheses (large populations, individual uniformity and appropriate structure), while the complexity of the resulting model is low. Besides, the ease of analysis, the wide variety of analysis tools available and the "*intuitive reasonableness*" make this kind of models very attractive. However, in many situations they ignore important factors inherent to the problem, such as the nature of contacts between individuals and the heterogeneity of the population. Cellular models are adequate to describe natural systems consisting of a massive collection of simple objects. They are a special case of models based on individuals, which represent the global system behavior from the description of the behavior of individuals within it. In this paper we study the time evolution of a heterogeneous population through the various stages of disease resulting from the individuals interactions (epidemic). The objectives of this work are *i*) the development of a model that includes the effects of heterogeneity and individual contacts in the evolution of the epidemic, *ii* the implementation of the proposed model through a cellular automaton and *iii* its validation with data from the 1918 influenza pandemic in the Geneva.

## 1 INTRODUCTION

Disease dynamics are often analysed using compartmental models based on the solution of systems of ordinary differential equations where homogeneous mixing between different classes (i.e. the mean-field approximation) is assumed (Kermack and McKendrick, 1927; Bailey, 1975; Anderson and May, 1991). In this type models, the reproductive number $R_0$ is the number created by a typical infected individual introduced into an otherwise fully susceptible population. This number is determined by the intrinsic infectivity of a single case, and the environment in which a case exists, for instance, the number of susceptibles that it comes into contact with. It is usually assumed that individuals are identical with respect to their infectivity.

This models also incorporate the homogeneous mixing assumption, sometimes called *the law of mass action* (Anderson and May, 1991; Diekmann and Heesterbeek, 2000), whereby the rate of increase in epidemic incidence is proportional to the product of the number of infectious and susceptible individuals. This assumption has been relaxed (den Driessche and Watmough, 2002; Lloyd and May, 1996; Finkenstadt and Grenfell, 1998; Grenfell et al., 2001; Watts et al., 2005), but not eliminated from models. The mass-action assumption is robust in the sense that it is consistent with several scenarios for the individual–to–individual transmission of disease. In particular, it is equivalent to a model in which all individuals in a population make contact at an identical rate and have identical probabilities of disease transmission to those contacts per unit of time. Although this assumption is unrealistic, it facilitates mathematical analysis and, in some cases, offers a reasonable approximation.

However, populations can be quite heterogeneous with respect to susceptibility, infectiousness, contact rates or number of partners, and simple homogeneous mixing models do not allow for extreme variation in host parameters. Heterogeneity in susceptibility and infectivity is an important feature of many infectious diseases and has been considered to improve the accuracy of epidemiological models. In the analysis of these models, focus has been placed on the impact of heterogeneity in the final size of epidemics (Miller, 2007; Rodrigues et al., 2009) as well as on its consequences to disease control (Andersson and Britton, 1998), and data interpretation (Anderson and May, 1991). In the context of epidemic models, it has been shown that the final size of the epidemic is reduced when the risk of infection is heterogeneously distributed in the population, both for the deterministic and the stochastic formulations (Andersson and Britton, 1998). More recently, results were extended to the investigation of epidemic spread in a random network (Miller, 2007).

New models capture some, but not all, of these features (Callaway et al., 2000; Strogatz, 2001; Newman, 2002; Newman et al., 2002; Eames and Keeling, 2002; Meyers et al., 2005, 2006). Ideally, an epidemic model would incorporate the following issues:

- A given individual has contact with only a finite number of other individuals in the population at any one time, and contacts that can result in disease transmission are usually short and repeated events.
- The number and frequency of contacts between individuals can be very heterogeneous.
- The numbers and identities of an individual's contacts will change as time goes by.
- The individuals have potential to transmit the pathogen and susceptibility.

Cellular automata (*CA*) can overcome these issues and have been used by several researches as an efficient alternative method to simulate epidemic spreading. Specifically, a two-dimensional

*CA* is formed by a two–dimensional array of identical objects called *cells*, which are endowed with a state that changes in discrete steps of time according to specific rules. As the *CA* evolves, the updated function (whose variables are the states of the neighbors cells) determines how local interactions, can influence the global behaviour of the system see (Ahmed and Agiza, 1998; Fuentes and Kuperman, 1999; Beauchemin et al., 2005; White et al., 2007), besides other studies in life sciences and computing journals). It is of special interest the *CA*-epidemic proposals modeling the motion of individuals (Mansilla and Gutierrez, 2000; Ahmed and Elgazzar, 2001).

In order to address these issues, and develop a realistic model for an epidemic situation, we introduced an individual–based–model built upon cellular automata that include all the features described in previous paragraphs. This modelling approach (*CA*) allows us to capture the individual heterogenity as well as a realistic model of individual contacts, modeling individuals explicitly exposed. Each individual will be characterised by its own intrinsic infectivity, expressed as the expected reproductive number $R_i$, which will be achieved by varying the infectiousness (i.e., the rate of transmission, given an unlimited supply of susceptibles) between individuals. Different assumptions with which we construct the classical model are applied directly to each individual, and the differential equations of the classical model are incorporated implicitly through rules. The resulting model is validated using the data from the 1918 influenza pandemic in Geneva (Chowell et al., 2005).

## 2 GENERAL REVIEW

Martín del Rey (White et al., 2007) offers a SEIR model implemented via a cellular automaton where each cell represents a particular population such as rural or urban core. The neighbors of each cell are those between which there is a communication channel that allows the flow of population from one to the other. The assumptions that underpin this model can be summarized as follows:

1. The total number of individuals in the cellular space and in each particular node remains constant over time. That is not taken into account migration processes or births or deaths. In turn, each cell has a maximum capacity of the population

2. The mode of transmission of disease is directly by physical contact between an infected and a susceptible individual (principle of mass action).

3. Finally, individuals can move between nodes and then return to the node to which they belong over time.

This model implements a classic SEIR at each node or cell. Assumes a homogeneous distribution of the population in each node, and heterogeneous between nodes. Implements a system of difference equations at each node, which is identical to the classic with the addition of a term of infection among neighboring cells. There is a neighborhood parameter and a parameter $\mu_{ab}^{(i,j)}$ is a factor that takes into account the rate of infection, migration and connection strength between cells.

Liu et al (nli, 2006) implements a classic SEIR model based on ordinary differential equations. The paper explores the spatial behavior of the epidemic diseases that are seasonal. For this is considered a variable rate of infection, according to a sinusoidal function. However, the rate is the same in all cells at a given instant of time.

To simulate the spatial-temporal movement associated with the different waves of epidemic they implemented a model called "dependent on the neighborhood," which is a modification of

the classical model, so that the equations do not evolve only with time t but they also depend on space $r$. Is, in short, a system of partial differential equations discretized in time and space. Unlike the first example, is not modeled infection between neighboring cells, so the population in each cell is variable. It also incorporates a stochastic migration.

Defining $c(r,t)$ as the vector of individual densities for a given position $r$ and time t given $L(c(r,t))$ as the local kinetic vector (corresponding to the ordinary differential equation model) and $D$ as the matrix of diffusion coefficients (which affects the rate of passage between cells for each type of individual), the system is expressed in compact form as:

$$\frac{\partial c(r,t)}{\partial t} = L(c(r,t)) + D \bigtriangledown^2 c(r,t). \tag{1}$$

The first step in each iteration corresponds to determine the local dynamics and the status of each cell independently of the states of neighboring cells. The second step corresponds to the spatial movement between neighbors, where each automaton calculates the sum of migration to and from every cell in the neighborhood, defined as Moore.

These two examples show a way of modeling is useful for visualizing the distribution patterns of the epidemic in the spatial dimensions, but does not capture or explain the patterns and fluctuations observed in temporal dimension. An approach close to that proposed in this paper is implemented by Quan-Xing Liu et al (Liu et al., 2008). In the implementation of an SIR model, defines a set of attributes for each cell:

- Ability to carry.
- Total population
- Susceptible population.
- Infected population.
- Population recovered.

The number of individuals per cell is variable. The main motivation for this is that you can include multiple individuals per cell, reducing the total number of cells required and therefore the computation time required for simulation. Second, it provides generality to the model.

The carrying capacity is the maximum number of individuals per cell, which defines a maximum population population density. The grid is rectangular and the movement of individuals occurs only between adjacent cells, the environment in itself represents a natural barrier to migratory movement. The parameters that explain the behavior of the epidemic are handled in a probabilistic except the effective radius of infection. The latter determines the maximum distance that the virus can move out of a particular host. On the other hand, the radius of movement determines the maximum distance that a particular host can be moved and is associated with a probability of movement. This probability determines the frequency with which this event can occur within a cell or between neighboring cells. Assuming a population distributed evenly in every cell. The cell to which the migration process occurs is chosen at random among since the neighbors of the current. An immigration parameter allows this model to simulate a world open or closed depending on its value. This parameter defines the probability of each cell to receive immigrants; this event is also affected by the load capacity. The model also incorporates a birth rate and natural death which relate to the incorporation of a new individual cell by natural reproduction in the case of the first parameter and the disappearance of one in the second case,

the latter affects alli ndividuals in the population. These events can occur with a probability associated with each one at each time step.

The morbidity of the virus is a measure of how fast the virus kills the host, only affects those who are in the state of infection. This model also incorporated other probabilistic parameters such as the likelihood of infection by contact with vectors, the probability of re-infection and the likelihood of spontaneous infection. The model of Shih Ching Fu incorporates hundreds of individuals per cell. In ascale of work this may be valid, but has the unintended effect of "softening" dynamics similarly to the previous examples. In our model we simulate an individual per cell maximum.

## 3 METHODS

A first definition of our automata network is:

- Each cell represents an individual in one of the possible states, or the state of empty cell. No distinction is made between the state of the deceased and the empty cell. Births involve passing empty cell to cell in a state susceptible.
- The transition between states is probabilistic. The transition probabilities correspond to the parameters of the classical model. These parameters are deterministic in the classical model, but as a result of an aggregation of individual probabilities under the assumption of a large population. Therefore, the probability is the transition behind the classical model and not vice versa. Applied directly to each controller, there is evidence of decreasing variability with increasing population, to coincide with the deterministic evolution of the classical model.
- The initial distribution is random, provided the assumption of homogeneous distribution for large population sizes.
- It simulates a random motion of the automaton through a reciprocal change in state neighboring cells, ie, a cell goes from $E1$ to $E2$ status as a neighbor state changes from $E2$ to $E1$. This movement seeks to emulate the approximate movement of real people (who really do not follow random movements), which contributes to the homogeneous distribution and contacts between infectious and susceptible.
- Potentially infectious contact is made between infectious individuals (symptomatic and asymptomatic) and is susceptible within the neighborhood defined as a zone of influence of infectious individuals. Infection is also probabilistic and directly related to the corresponding parameter in the classical model.
- For simplicity, the grid type used is rectangular, and the Moore neighborhood is kind, with a size not defined a priori.
- The boundary condition is fixed, with a contour consisting of non interacting empty cells, compatible with the situation in a city, an area of high population density area surrounded by a much lower density.

The automata are then defined as a stochastic Moore machine (White et al., 2007) by $A = (E, X, Y, \delta, \beta, \rho)$, where:

- $E$, the set of possible states comprises 7 states: $S$ (susceptible), $E$ (exposed), $I$ (infectious), $A$ (asymptomatic), $J$ (infectious reported), $R$ (recovered) and $D$ (dead or empty).
- $X$ is the set of input (real numbers). An automaton recives input only when state $S$, issued by another in state $I$ or $A$, when the automaton is in the vicinity of the issuer. Transitions that do not involve contact with infectious individuals are made in probabilistic form independently of a possible entry (transitions on empty entry $\varepsilon$).
- $Y$ is the output set (equal to X) issued in state $I$ or $A$, corresponding to the input received in state $S$. The output corresponds to the probability of infection from contact that has the automata in stage $I$ or $A$, obtained from distributing the beta value for that automaton in the neighborhood under consideration.
- $\delta$ is the state transition function, which applied to the active state at iteration $k$, the state decides probabilistically active at iteration $k+1$. The function is applied in two steps, one for the state change and recovery from infection and the other corresponding to the movement. To decide the status changes to define two probability matrices: one for empty transition $\varepsilon$ and a blank entry for the transition from contact with infectious
- Since each element of the matrix pij the probability of moving from state i to j in each time step, and placing the states $S$, $E$, $I$, $A$, $J$, $R$ and $D$ in increasing order from row or column 1 to 7, the transition matrix for empty entry is defined in table 1

| | S | E | I | A | J | R | D |
|---|---|---|---|---|---|---|---|
| S | $1-\mu$ | 0 | 0 | 0 | 0 | 0 | $\mu$ |
| E | 0 | $1-(\varepsilon+\mu)$ | $\varepsilon\rho$ | $\varepsilon(1-\rho)$ | 0 | 0 | $\mu$ |
| I | 0 | 0 | $1-(\alpha+\gamma 1+\mu)$ | 0 | $\alpha$ | $\gamma 1$ | $\mu$ |
| A | 0 | 0 | 0 | $1-(\gamma 1+\mu)$ | 0 | $\gamma 1$ | $\mu$ |
| J | 0 | 0 | 0 | 0 | $1-(\gamma 2+\mu+\delta)$ | $\gamma\, 2$ | $\mu+\delta$ |
| R | 0 | 0 | 0 | 0 | 0 | $1-\mu$ | $\mu$ |
| D | $\mu$ | 0 | 0 | 0 | 0 | 0 | $1-\mu$ |

Table 1: Transition matrix for empty entry

The symbols in table 1 correspond to the parameters used in the classical model (Chowell et al., 2005). Defining the size of the neighborhood as $\nu$ and the input value as $\lambda$ ($\beta$ may correspond to or comes from a q$\beta$ as symptomatic or asymptomatic infection, respectively), the contact transition matrix is defined in table 2

| | S | E |
|---|---|---|
| S | $1-(\lambda/\nu)$ | $\lambda/\nu$ |
| E | 0 | 1 |

Table 2: contact transition matrix

Finally, the movement is equally likely from a cell centered in an area of predefined size to any other, including itself, including in the area. The cells are swapped positions, which can be interpreted as changes of state.

- $\beta$ is the output function, which gives the value of infection rate if the automaton is instate I or A.

- P(o), the initial state vector is composed of the probabilities for each initial state be given an automaton. $Gt$ defined as the total number of cells in the grid and as $Si$, $Ei$, $Ii$, $Ai$, $Ji$, $Ri$, $Di$ the initial number of individuals in each state in the grid (being the sum equal to Gt and the population total, and that God includes the empty cells), the vector is defined as $P(o) = [Si/G, Ei/G, Ii/G, Ai/G, Ji/G, Ri/G, Di/G]$

The cellular automata are defined by $R = G(T, C)$ where:

- The topology $T$ is square. The neighborhood is kind Moore, and is only seen for cells in stage $I$ or $A$. The boundary conditions are fixed, with a contour consisting of empty cells not interacting.

- The connection $C$ is unidirectional from cells in state $I$ or $A$ to the cells in a state $S$ that are within the vicinity. The connection is isotropic and equal anywhere in the neighborhood, and provides an entry for each cell in $S$ consisting of the value of $\lambda$ that has the cell $I$ or $A$, used to make the transition to the state probabilistic $E$. The cells in a state $S$ which are included in several neighborhoods in a given time step will have many opportunities to change state as the number of neighborhoods in which they are included.

The pseudocode for the proposed controller system can be described as follows:

```
for day=1 to N do
  for each grid cell do

    {Infection}

    if cell state = I then
      if neighboring cell = S then
        Z ~ U[0,1]
        if Z<β/ν then
          move to state E
        end if
      end if
    else
      if cell state = A then
        Z ~ U[0,1]
        if Z<qβ/ν then
          move to state E
        end if
      end if
    end if

    {Exposed to infectious}

    if cell state = E then
      Z ~ U[0,1]
      if Z<εr then
        cell state = I
      if εr<Z<εr then
        cell state = A
      end if
    end if
  end if

  {Step-by reported}

  if cell state = I then
    Z ~ U[0,1]
    if Z<α then
      cell state = J
    end if
  end if

  {Recovery}

  if cell state = I or A then
    Z ~ U[0,1]
    if Z<γ1 then
      cell state = R
    end if
  end if
  if cell state = J then
    Z ~ U[0,1]
    if Z<γ2 then
      cell state = R
    end if
  end if

  {Death from disease}

  if cell state = I or J then
    Z ~ U[0,1]
    if Z<δ then
      cell state = D
```

```
    end if
  end if

  {Births and natural deaths}

  if cell state ≠ D then
    Z1 ∼ U[0, 1]
    Z2 ∼ U[0, 1]
    if Z1<μ then
      cell state = D
    end if
    if Z2<μ then
      cell state = S
    end if
  end if

  {Movement of individuals}

    Z1, Z2 ∼ U[−r, r]
    aux = cell state(i,j)
    cell state(i,j)=cell state(i + Z1,j + Z2)
    cell state(i + Z1,j + Z2)= aux
  end for
end for
```

## 4 ADJUSTMENT AND RESULTS

When attempting to adjust the classical model with the parameters minimized, there are several obstacles and alternatives to use. First, there is the problem of the scale used. Obviously the larger the grid and employed population, the closer we are to large population of the classical model (as well as the higher the computational cost of the simulation). As the extension of the validity of this hypothesis is just something that is challenging to apply the model of automata, it is neither necessary nor desirable to use too large grid sizes when it comes to analyzing the temporal behavior of the epidemic, but what is the purpose of validating the model automatically using as parameter the classical model.

Another choice that shows necessary and important effects on the overall performance is the size and shape of the neighborhood. The use of alternative forms shows no significant change, so Moore is chosen for simplicity of programming and computation. The size of the neighborhood, however, determines the degree of global influence of the heterogeneity. The larger the neighborhood size used, the closer the results to the assumption of spatial homogeneity of the classical model. This is because a very large neighborhood can influence the infectious even in low density areas of infectious, "softening" the effect of heterogeneity. If we used a grid size neighborhood-wide, the spatial distribution of individuals would not matter, and the result would be equivalent to a perfectly homogeneous distribution because each infectious influence would spread throughout the grid, regardless of location..

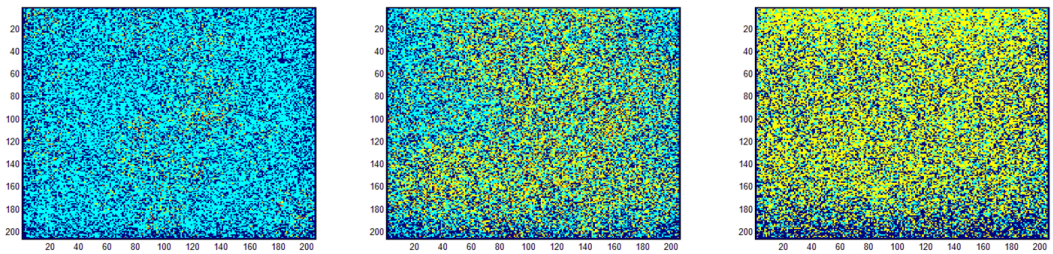


Figure 1: Evolution in time of the model, can see the hetorogeneity distribution along time,neighborhood size (ratio) is $r = 12$

A third problem arises at the time of transfer the parameters of the classical model to the automata (see table 3 ). How to apply different sets of parameters obtained from the minimizations is not straightforward. In the classical model, these parameters represent daily rates per person, but can not go directly to a daily variation by multiplying the number of individuals,

because they are instantaneous rates that depend on the number of individuals in each moment, which does not vary during the time between one step and another in the discrete case but it does in the continuous case for the classical model.

| $\beta$ | $\rho$ | $\gamma_1$ | $\gamma_2$ | $\alpha$ | q | Ne | Ni |
|---|---|---|---|---|---|---|---|
| 8,3 | 0,087 | 0,246 | 0,97 | 0,465 | 0,0008 | 207 | 136 |

Table 3: Minimized parameters from classical model

The linear terms generally respond to exponential decay rate $F(t) = F(0)e(-k_1 t)$, that must correspond to the discrete version:$f(n) = f(n-1)k_2$, with $0 < k_2 < 1$ Using a time step of one day, $F(0)$ must correspond to $f(1)$ and $F(1) = F(0)e(-k_1)$ must do so $f(2) = f(1)k_2$, then $k_2 = e(-k_1)$.

The exponential decay can be expressed as discrete $f(n) = f(n-1) - f(n-1)(1k_2)$. The second term on the right side represents how much function decreases at each step. In our case, corresponds to how many individuals go out of state, so that $(1 - k_2) = (1 - e(-k_1))$ is the parameter used to decide in each stochastic iteration step to the next state. In the case of having a non-linear term, a good approximation was not achieved, so that it was a good approximation.

Finally, it is possible to make a modification of the model in the compartment $J$. This compartment is included in the classical model of necessity, since the data reported correspond to individuals and not the total number of patients. However, the rate of infection is considered equal to the compartment $I$. For all purposes of the model, the behavior varies with respect to symptomatic infectious individuals. It is then possible to remove the magazine and obtain $J$ at the end of each iteration from the rate reported. This results in an initial error, as in the classical model there is no initial individuals $J$. However, this modification can be used to obtain an additional advantage.

With all these considerations, we first performed a simulation that meets the assumptions of the classical model as much as possible. To do this, we used a total population size of ten times the population of Geneva in 1918, evenly distributed on the grid. The neighborhood size was enlarged to the maximum, so that each infectious interact evenly with everyone. $J$ compartment was included explicitly. To approach more realistic conditions, simulations were performed after the actual population size, and small neighborhoods. The initial distribution of individuals is kept uniform.

The problem is that the adjustment of parameters in the classical model for any set of values that minimizes the error, regardless of whether they have physical significance or is likely to occur and, of course, say nothing of their behavior under other conditions, in this case, a discrete population, where the total homogeneity is not possible.

The parameter set used in this case, the parameter $q$ that affects the infection rate in passing the asymptomatic state is zero, implying that these individuals are not infected at all. In addition to the low proportion of exposed who become infectious symptoms (less than a tenth), the effect on the automata model is a rapid decline in the effective rate of infection, because each infectious individual is, after a short time, surrounded by a large proportion of asymptomatic individuals who can not infect anyone and which can not spread.

We tried to solve the problem by adjusting the rate of infection, but no improvements are obtained because the effect of reduction of infectivity persists to stay $q$. In contrast, for a small adjustment of $q$, the behavior reverts to the expected one when evaluating different possible probability distributions on the rate of infection, it is necessary to make an adjustment of statistical parameters that characterize each distribution: mean and standard deviation. In the scale

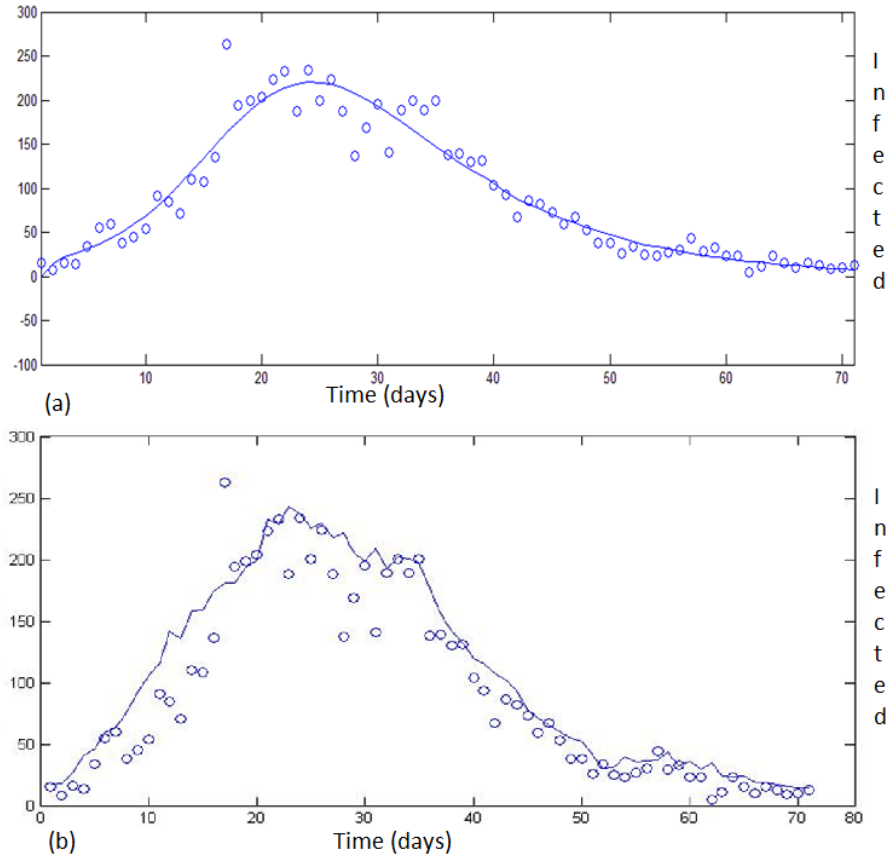


Figure 2: Comparison between classical model (a) and cellular automata (b), may see the efect of heterogeneity in the kinetics of disease

used, this is impractical because the computational cost would sue. If we reduce the scale, the variability between different realizations and each realization in particular is very large, far from the observed. This is where it is useful to remove the compartment $J$.

Clearing the way to $J$ in the simulation and applies it to deterministically the evolution of infectious obtained, we get: You can see that passing in deterministically to $J$, the effect is "softened" by reducing the expected variability. This is equivalent to a simulation with $J$ explicit, but on a larger scale. To achieve a variable equivalent to that obtained with $J$ explicit, should then be reduced scale. If you shrink the scale by a factor similar to the rate of step $J$ and perform the simulation with $J$ removed from the grid, you get a similar trend to that expected in the original scale, both overall and behavioral variability. This will reduce the computational cost achieved by the adjustment of parameters.

In the simulation, the value of the infection rate is the same for all individuals, considered as the average of individual rates. Classical population models applied to the study of epidemics using these quantifiers averaged to describe the system, being that it is naturally heterogeneous(Lloyd-Smith et al., 2005; Li et al., 2004). This type of estimate does not take into account the role of individual variation in the infection process. This variation can be of great influence when the spatial distribution is not uniform and when there is a significant presence of "superspreaders"(Shen et al., 2004).

Superspreaders are those individuals within the population that are capable of infecting a greater number of susceptible individuals than the average(Galvani and May, 2005), in other words, if the reproductive number $R0$ of the epidemic, a superspreader has a reproductive num-

ber larger than the average. Thus we can define the individual reproductive number, $\upsilon$ as the expected number of secondary cases infected by a particular. This parameter can be described using probability density functions with one or more continuous half as if the function used. With a continuously distributed degree of infectivity is achieved by adding more heterogeneity to the model.

These changes in the reproductive number may be due to factors such as:

- Individuals infected and undiagnosed.
- High rates of contact between individuals.
- High viral load in infected individuals.

In our case, the variation of $R0$ is achieved only through the variation in infection rate. In one case using a normal probability distribution to generate a grid of individuals infected with variable values, this probability distribution has $\beta$ and variance $\sigma$ half. Another uses a bimodal probability distribution to generate a grid of infected individuals, this distribution has two modes: $\beta_i$ and $\beta_s$ for the probability of infection through contact with infected individuals and superspreaders respectively.

While not specifically stated, in both cases there are also present asymptomatic individuals, who have a very low infectivity but influence the dynamics of the epidemic as they represent a significant percentage of the population. In this case the rate of infectivity of asymptomatic individuals remains at a fixed value, but could also be considered as a third heterogeneous population modeling together as a trimodal distribution, with modes will be $Pa$, $\beta_i$ and $\beta_s$, for asymptomatic individuals, infective and superspreaders, respectively. For both the normal distribution as for the bimodal parameters were adjusted average (or fads) and variance. In this context, the patterns that appear as epidemiological ripples within a short time period are interpreted as regrowth due to passage of a certain amount of superspreaders to infection status (see figures 3 and 4).

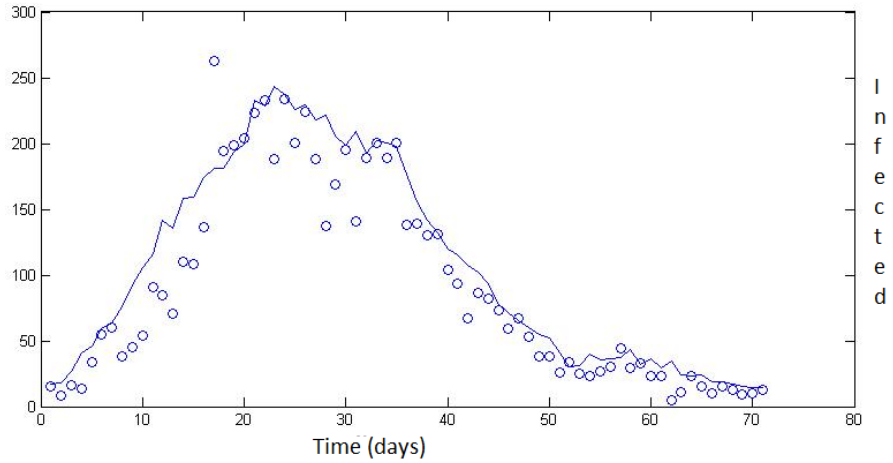


Figure 3: Simulation results Vs real data using normal distribution

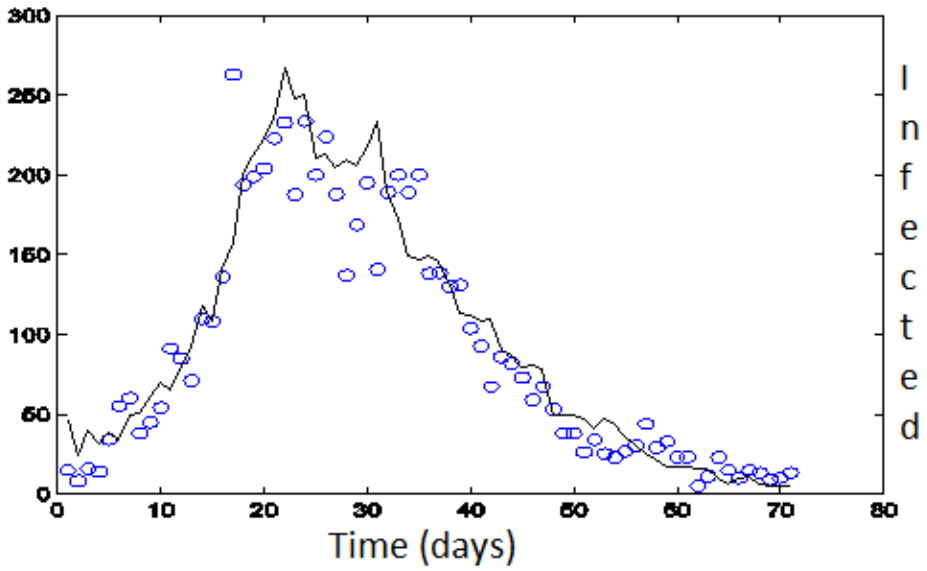


Figure 4: Simulation results Vs real data using bimodal distribution

## 5 CONCLUSIONS

As noted earlier, individual-based models are especially useful when trying to model a system as a massive collection of entities, all of which influence the global behavior. Modeling by a set of rules results in a natural stochasticity and therefore implies a heterogeneity. The implementation of different probability distributions for the population of infectious individuals results in greater heterogeneity and a dynamic that is more like the real thing. The simulation of random motion through the reciprocal exchange of states between adjacent cells contributes the homogeneous distribution of the population, which increases the probability of contact between individuals susceptible and infectious and therefore the spread of the epidemic.

The size of the neighborhood you choose determines the level of overall system heterogeneity, thus when working with neighborhoods too small to reach the epidemic could spread not by the lack of contact between healthy individuals and infection, on the other hand, too large a neighborhood could generate a distribution too homogeneous resembling the behavior to a common compartment model and ignoring the effect of each individual if that is what you want to study.

The inclusion of superspreaders in the population of infectious individuals results in greater stochasticity in the system. It can be seen that the number of infectious individuals at time t does not follow a pattern of growth and normal disminution, Prodicus small sprouts along the epidemic.

Finally, it can be noted that the importance of this type of modeling lies in the fact that it allows to see a system as a set of individuals or smaller systems that contribute to overall system performance. Heterogeneity is a property of all populations and understand their role in population phenomena such as an epidemic is of vital importance because it allows us to better understand this phenomenon.

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