Cellular Automata SIR Model

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I. INTRODUCTION

In the course of our history, humanity has faced countless viral outbreaks. The contagions responsible spread from person to person among the susceptible population. Apart from viruses whose rapid mutations prevent effective long-term immunization against them, people eventually either die or become resistant to these microbial attackers, preventing further illness. When someone becomes infected with a virus in a population susceptible to it, many people become affected by it in a cascading effect, and the results can be terrific. The Black Death is the most historic example of this, when the bubonic plague swept through Europe leaving a devastation of expired bodies in its wake.

To combat horrific events such as this, and to better the human condition, we have developed various treatments for disease. We have developed medications to manage symptoms and reduce contagiousness, and even cure some diseases. However, some viruses are beginning to grow resistant to our medicines, notably penicillin, one of our most effective treatments. However, we have also discovered vaccinations: a process of administering a weakened strain of a virus to someone susceptible to it so that their immune system can eradicate that virus and develop a lasting immunity to it in the process. This has proven highly effective, although there is still some misguided reticence about it, chiefly a baseless but widespread rumor that it can cause autism.

The purpose of this paper is to recreate the work of White, del Rey, and Sánchez in their article, *Modeling epidemics using cellular automata*. We have written our own implementation of their algorithm to simulate the spread of a contagion in a susceptible population, perhaps the outbreak of a new virus, and have focused our exploration on how vaccination affects the spread of contagion (2007, p.193).

II. MODEL

At any given time, everyone belongs to one of three classes: susceptible, infected, or recovered. These three classes give this type of model its name, SIR. Susceptible people are those who are not infected, but have no immunity to the contagion and so are vulnerable. Infected people are those who have contracted the contagion and are contagious to susceptible people. Recovered people are those who have developed immunity to the contagion, and are not contagious. Our model is discrete; there is a finite number of people, and we consider changes in their state (i.e. which SIR class they belong in) to occur between unit time intervals. We refer to these intervals as "days."

We model a region with a vulnerable population as a 50 by 50 grid of cells, each representing a local population. We use the Von Neumann neighborhood of each cell to determine what cells are adjacent to each other, and each day we allow the local populations of adjacent cells to mingle and potentially infect one another. We assume the local populations are static, and everyone returns to their original cell each day after mingling. The Von Neumann neighborhood of a cell with coordinates (i, j) in a square grid is the set of up to four neighboring sites given by the coordinates $(i+1, j)$, $(i-1, j)$, $(i, j+1)$, $(i, j-1)$, except those at coordinates that are not on the defined grid. Thus, cells on the edge will only have 3 Von Neumann neighbors, and cells in the corners will only have 2. All internal cells will have 4.

There are a few ways people can shift between classes. Each day, susceptible people within a cell become infected from contact with infected people from that same cell and from neighboring cells, a fraction of infected people recover naturally, and a fraction of susceptible people are moved directly to the recovered class through vaccination. These shifts are reflected in three equations that govern the simultaneous updating of each cell each day.

$$S_{ij}^t = S_{ij}^{t-1} - \omega \cdot S_{ij}^{t-1} - v \cdot S_{ij}^{t-1} \cdot I_{ij}^{t-1} - v \cdot S_{ij}^{t-1} \cdot \sum_{(\alpha,\beta)\in V^*} \frac{N_{i+\alpha,j+\beta}}{N_{ij}} \cdot \mu_{\alpha\beta}^{(i,j)} \cdot I_{i+\alpha,j+\beta}^{t-1}, \tag{1}$$

$$I_{ij}^t = (1-\epsilon) \cdot I_{ij}^{t-1} + v \cdot S_{ij}^{t-1} \cdot I_{ij}^{t-1} + v \cdot S_{ij}^{t-1} \cdot \sum_{(\alpha,\beta)\in V^*} \frac{N_{i+\alpha,j+\beta}}{N_{ij}} \cdot \mu_{\alpha\beta}^{(i,j)} \cdot I_{i+\alpha,j+\beta}^{t-1}, \tag{2}$$

$$R_{ij}^t = R_{ij}^{t-1} + \epsilon \cdot I_{ij}^{t-1} + \omega \cdot S_{ij}^{t-1}. \tag{3}$$

These are the SIR equations (2007, p.201). S_{ij}, I_{ij}, and R_{ij} are respectively the proportion of people in cell (i, j) who are susceptible, infected, and recovered. t represents the time index, or day, ϵ is the recovery rate, v is the virulence of the contagion, ω is the vaccination rate, $\mu_{\alpha\beta}^{(i,j)}$ is the level of contact between cells (i, j) and $(i + \alpha, j + \beta)$, V^* is the set of relative coordinates in the Von Neumann neighborhood of the cell at (i, j), and N_{ij} is the size of the local population in cell (i, j). To be precise, the recovery rate ϵ is the proportion of infected people who recover naturally each day, the virulence v is the proportion of susceptible people who have come into contact with the contagion that day that becomes infected, and the vaccination rate ω is the proportion of susceptible people who become vaccinated each day and develop an immunity without ever actually becoming infected, thus going directly to the recovered class. These equations are applied to all (i, j) coordinates in the grid between days.

$\mu_{\alpha\beta}^{(i,j)}$ is actually the product of two factors, $c_{\alpha\beta}^{(i,j)}$ and $m_{\alpha\beta}^{(i,j)}$. $c_{\alpha\beta}^{(i,j)}$ is the connectivity between cells (i, j) and $(i+\alpha, j+\beta)$. It can take values of 0, 0.3, 0.6, and 1, depending on the number of modes of transportation between two particular (adjacent) cells. If there are no modes of transportation, then $c_{\alpha\beta}^{(i,j)} = 0$. If there is only one mode of transportation, $c_{\alpha\beta}^{(i,j)} = 0.3$. If there are two, $c_{\alpha\beta}^{(i,j)} = 0.6$, and if there are three then $c_{\alpha\beta}^{(i,j)} = 1$. Modes of transportation considered include by plane, by train, and by car or bus. $m_{\alpha\beta}^{(i,j)}$ is the movement factor between cells (i, j) and $(i + \alpha, j + \beta)$; it is a value between 0 and 1 and represents the probability that an infected person will travel to cell (i, j) from its neighboring cell, $(i+\alpha, j+\beta)$. It depends on the disease itself, and peoples' response to it. If we were to account for quarantining effects, we would do it by reducing this factor, thus reducing the intermingling of infected people with people in the neighboring communities.

Each term in the SIR equations represents a shift in a finite number of people between SIR classes. Each day, each person is entirely within one of the three SIR classes and neither of the other two; we do not allow fractional recovery or infection of people. We address this in our implementation of our simulation by rounding each term to represent a whole number of people. However, this creates a numerical issue towards the end of our simulation: as infected people continue to recover, eventually the number of people to recover may be less than 1 despite there being infected people remaining. This could lead to a small portion of the population staying infected forever and never recovering. To address this issue, we check each cell for this situation. If it does have too few infected people for anyone to recover, we allow one person to recover in that cell with probability e. In doing so, we have broken the determinism of this algorithm by introducing a probabilistic step, but this is not concerning because the effects of this fix are only experienced towards the end of the simulation, and it does not affect the behavior of the model. Plus, with 2500 cells, there will be very little overall variation between simulations from introducing this non-determinism.

The parameter space for this simulation is very large. For this reason it has the potential to simulate a rich variety of situations and effects, from different spreading patterns depending on the connective infrastructure and population distribution in a region to the effects of quarantine protocols to herd immunity, to name a few. However, we are investigating the effects and effectiveness of vaccinating against a new pathogen once it has been introduced to an entirely susceptible population. To that end, we limit the scope of our investigation to a small slice of the potential parameter space. First, we consider all cells in the region to be essentially the same. All cells have a local population of 100 people, and each pair of neighboring cells have three modes of transportation between them ($c = 1$) and a movement factor of 0.5. We run several simulations with different vaccination rates, but use the same values for the recovery rate and virulence of the contagion. The values we have chosen are $\epsilon = 0.4$ and $v = 0.6$. All cells begin with $S = 1$, $I = 0$, and $R = 0$, except the central cell at $(25, 25)$ which begins with 30 infected people.

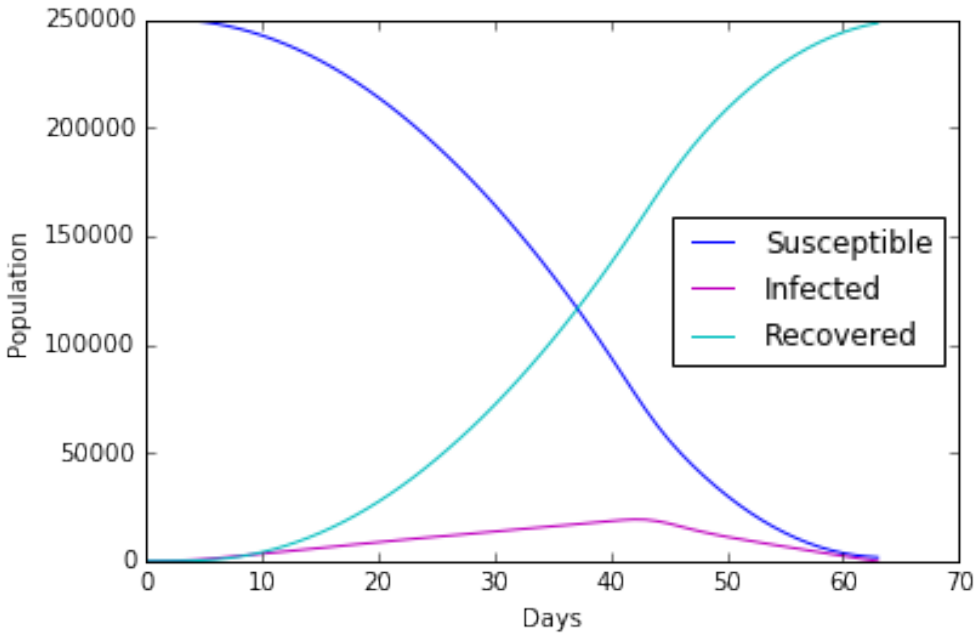

FIG. 1. Susceptible, Infected, and Recovered populations as functions of time with no vaccinations

III. RESULTS

We begin by presenting the evolution of the system without vaccinations, i.e. with $\omega = 0$. Figure 1 is a plot of the total number of people in each of the three SIR categories as a function of time.
As you might expect, the initial susceptible population is the total number of people in our simulation, except 30 who are infected in the center cell. As time goes on, the infected population rises as more and more people become exposed. However, the recovered population grows even more quickly as people who were infected recover.

A more direct way to examine the system's behavior is to look at the actual spread of infection through the region. Figure 2 is a series of 6 snapshots of the I-values in the region, where a darker pink indicates a greater value of I (the proportion of people in that cell who are infected). They begin at day 5 and increment by 10 days between images, up to day 55.

As you can see, the contagion spreads outward unhindered, expanding linearly with time until eventually reaching the borders of our space where there are no more people to infect. It spreads in a single roughly circular wave; its shape is slightly non-circular due to the non-circular character of the Von Neumann neighborhood we have used. This wave of infection does not grow thicker because people continuously recover at a rate proportional to the number of people infected, so you might think of a recovered wave as "chasing" after the infected wave.

The behavior changes notably when we include vaccinations. The two sets of 6 images in Figure 3 and Figure 4 are snapshots of the I-values in the region just like the above set of 6 figures. The timing of these images is the same as above: every 10 days from day 5 to day 55. However, in the first set we use a vaccination rate of $\omega = 0.01$, and in the second we use $\omega = 0.02$. As we increase ω we find that the spread of the infection is slowed, since the infected wave front does not extend as far after a given number of days, and the magnitude of the infection at the wave front decays and eventually vanishes.

These effects are the result of an increasingly immunized population as the infection spreads. As each day goes by, a portion of susceptible people become immunized and go into the recovered category. This impedes the spread of the infection because it is less likely that an infected person wandering into an uninfected cell will come into contact with and expose a susceptible person. Or, to put it deterministically, there are simply fewer susceptible people to infect, and so fewer people in the uninfected cell contract the virus. Eventually, the infected wave front thins, and most everyone is immunized against the contagion, and so it dies out. For a given vaccination rate, we could determine the maximum radius out to which the contagion would spread

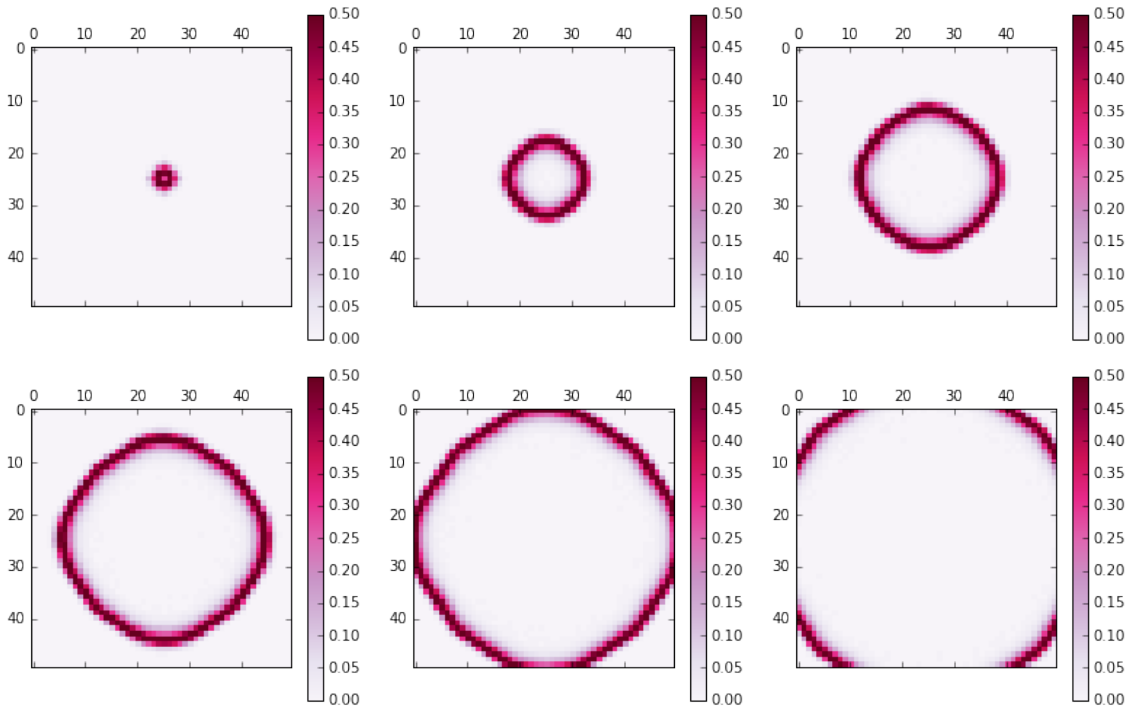

FIG. 2. Level of infection I in the entire region every 10 days, starting at day 5, with no vaccinations

before dying entirely, even without a definite border.

The infection rate is proportional to the number of people exposed, which is roughly proportional to the circumference of the infected wave, or equivalently its radius. The recovery rate is proportional to the number of people infected, which is also roughly proportional to the circumference of the infected wave, so the actual slope of the overall infected population is a result of the competition between these two equally scaling rates, and is thus essentially linear. To gauge the accuracy of this assertion that the overall infected population scales linearly with time (without vaccinations), we have plotted the number of days it took the contagion to reach the edge of the grid for several grid edge lengths in Figure 5. This is discernably linear, and therefore the radius of the contagion increases linearly with time, and our assertion is validated.

To better view the infection curve, we have plotted it alone for several vaccination rates in Figure 6. Again, this is the total number of infected people as a function of time.

As you can see, the infection curve for the case with no vaccinations ($\omega = 0$) increases approximately linearly after a brief "burn-in" time of about 5 days. During this burn-in time, the infection wave gets set up; the infection spreads slightly and those inside the wave front recover. The infection wave shown in Figure 2 reaches the borders of the region just before day 45. This is where the infection curve falls out of its linearly increasing regime and quickly drops, as there is no one left to infect and those who are still infected recover.

As we turn on and increase the vaccination rate ω, the duration of the epidemic is increased for $\omega = 1\%$, but decreases for subsequent values. The increase for small ω is due to the decreased rate at which the infection spreads; essentially, the epidemic lasts longer because the contagion takes longer to reach the confines of the region. However, for greater vaccination rates, the reduced infection rate overtakes this effect and the contagion dies off before even reaching the borders.

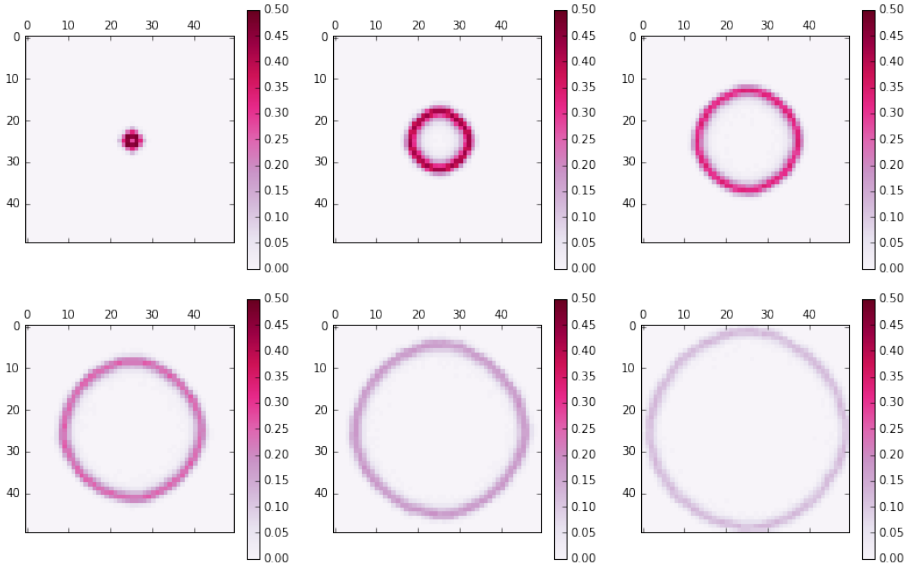

FIG. 3. Level of infection I in the entire region every 10 days, starting at day 5, with $\omega = 1\%$

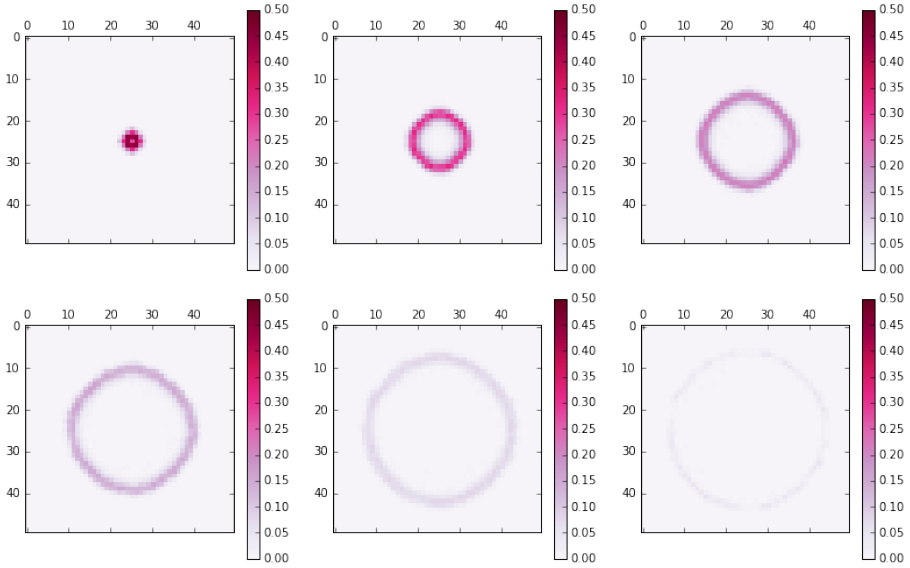

FIG. 4. Level of infection I in the entire region every 10 days, starting at day 5, with $\omega = 2\%$

Another effect of turning on and increasing ω that we can see from Figure 6 is that the peak of the infection curve quickly drops. Increasing ω from 0 to just 1% more than halves the maximum height of the infection curve. This is more clearly illustrated in Figure 7, which plots in purple the maximum heights of these curves for each of several vaccination rates. These are the largest number of people infected at the same time over the course of the epidemic for each of these ω values.

This graph also includes the total number of people infected over the course of the epidemic, in red. We used a logarithmic y-axis to show the separation between points for larger ω values because the magnitudes of the differences decrease substantially. As a quick sanity check, notice that the total number of people infected without vaccination is right around 250,000, the total number of people in the entire region. This is what we would expect, so this affirms the physical accuracy of our model.

These are both informative measures of the severity of an epidemic. If a large number of people are infected simultaneously, then there could be issues accommodating them all if they require treatment. Alternatively,

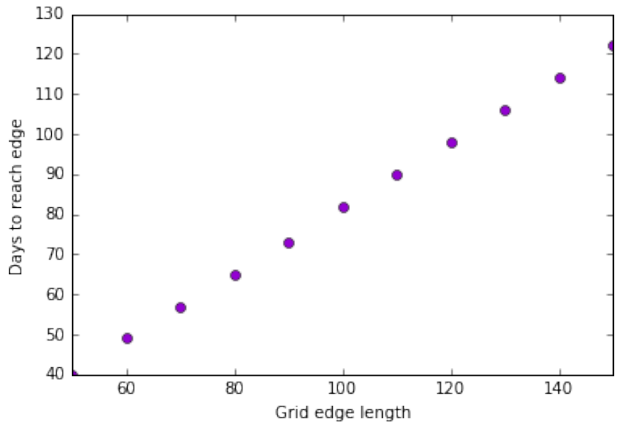

FIG. 5. Days to reach the border of the grid as a function of grid size

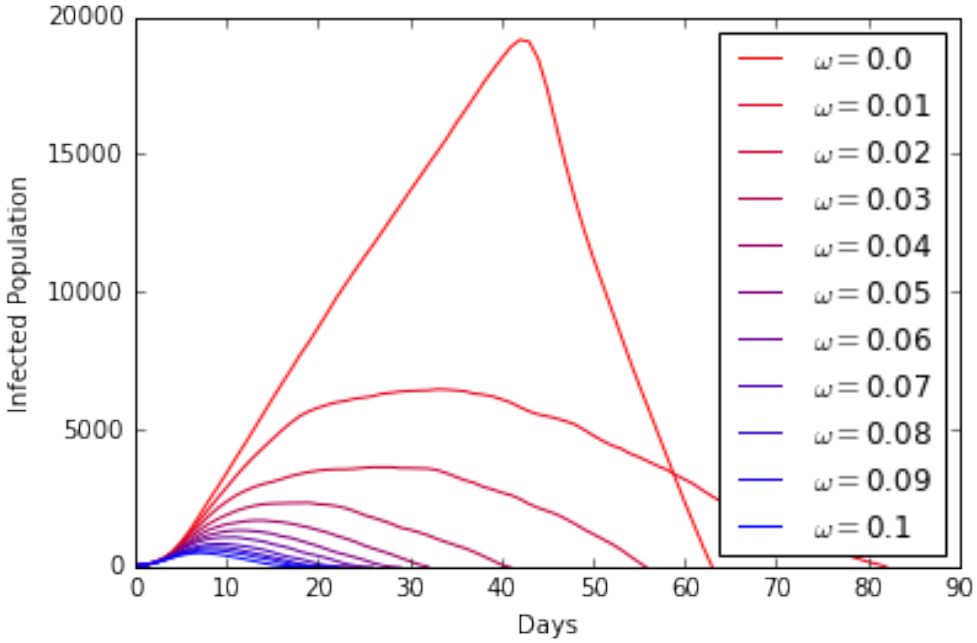

FIG. 6. Total infected population as a function of time for various vaccination rates ω

if the virus exhibits symptoms that keep its hosts from work, then this may impact how well, for example, a city is able to function. On the other hand, if a large number of people are infected over the course of the epidemic, this implies it has spread very far and is more likely to expand into more distant regions not modeled by our algorithm, perhaps a different continent. Additionally, if the contagion leads to lasting injury or death, then a large number of people infected overall would imply a crippled population or devastating loss of life as was seen with the Black Death.

IV. ALGORITHMIC CONSIDERATIONS

In this section we defend our choice to relax the determinism of our algorithm to prevent small numbers of infected people from persisting without ever recovering, as was discussed in the Model section. On the left in Figure 8 is the infection curve for $\omega = 0$ with a purely deterministic version of our model. In the center is

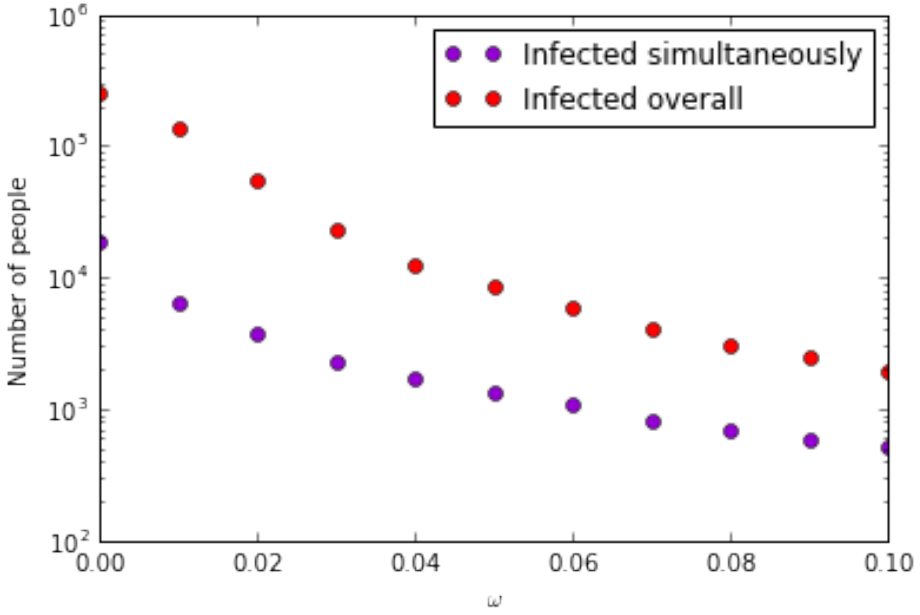

FIG. 7. The greatest number of infected at a given time, and the total number of people infected, over the course of the outbreak as a function of the vaccination rate ω

10 overplotted infection curves for the slightly non-deterministic model that we have used throughout this paper. On the right we have plotted the values of those curves at day 60, ordered for clarity. We have calculated the average of these points to be 2370, with a standard deviation of only 22. Day 60 is towards the end of these runs so any differences between these curves should be maximized around this point, but as you can see they are very small. Additionally, by examination, the 10 non-deterministic curves in the center are all very similar to the deterministic curve on the left. Therefore, we conclude that our choice of using a probability of ϵ to force recovery does not disturb the model.

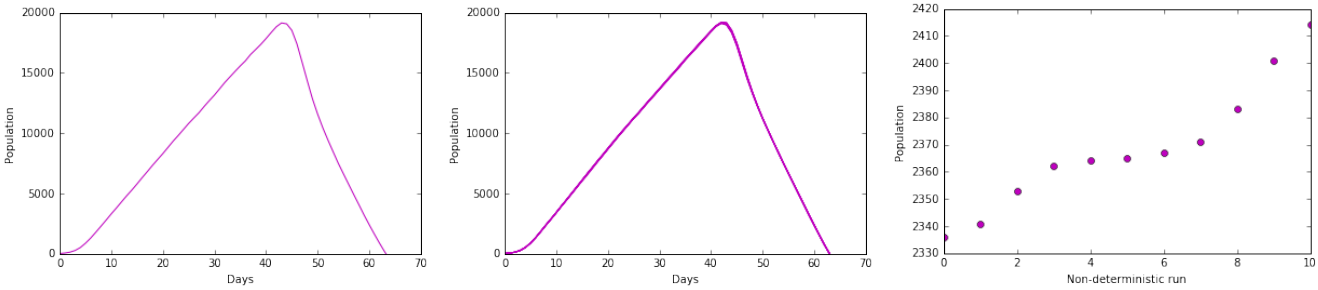

FIG. 8. (left) Infection profile with no vaccinations for a purely deterministic model,
(center) Infection profile with no vaccinations for a slightly non-deterministic model,
(right) Ordered total infected population values at day 60 for each of 10 non-deterministic runs of our algorithm

For the most part, the difference between these curves is small, and only slightly noticeable since they are qualitatively the same in all but one respect: the curve on the right terminates at 5000 people. To use this deterministic version of the algorithm we would have to arbitrate some cutoff point for an effectively completed simulation, but that would skew the numbers over the whole simulation because no cell would be able to recover completely.

The infection curve on the right for the deterministic version of the algorithm has a slightly smaller slope than the one we used on the left, and has a slightly lower highest point. However, these minute differences do not affect the overall behavior of our model, as can be seen quite clearly from the juxtaposition of the

plots of all three SIR populations for the two versions of the algorithm, shown in Figure 9.

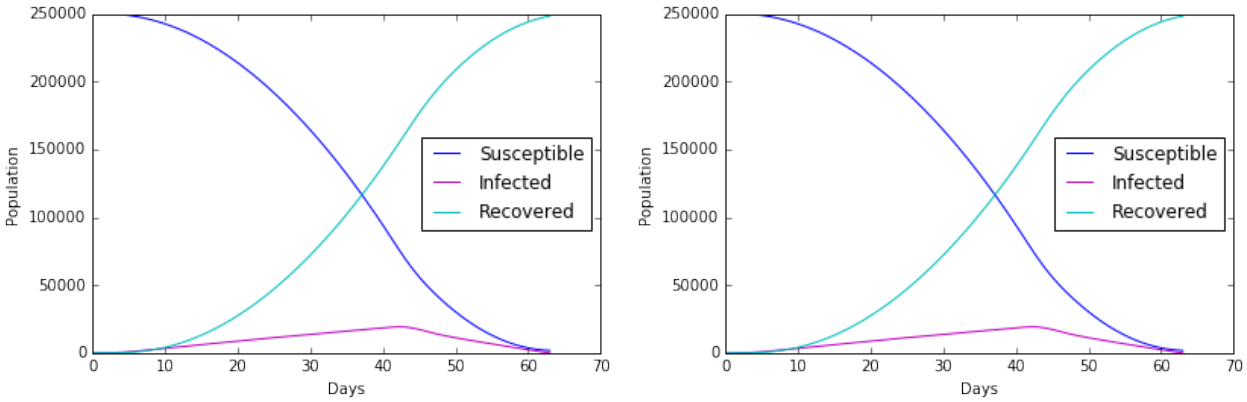

FIG. 9. Susceptible, Infected, and Recovered profiles with no vaccinations for (left) a purely deterministic model (right) a slightly non-deterministic model

V. CONCLUSION

In this paper we have created a robust SIR model for the spread of a contagion. That is, we categorize people into three classes: susceptible, infected, and recovered (SIR). We used a grid of cells with local populations and allowed the number of susceptible, infected, and recovered people in each cell to evolve according to equations 1-3. This model has a very rich parameter space that allows it to simulate epidemics under a variety of circumstances and to probe several types of effects.

To focus on the effects of vaccination, we varied ω and held other parameters constant. Over the course of our investigation we found several benefits of vaccination. We found that vaccinating those susceptible to infection reduces the rate at which the infection spreads, and eventually eliminates the contagion, even in an unbounded region. Increased vaccination rates also reduce the number of people infected simultaneously, as well as the total number of people infected over the course of the epidemic.

For the most part, our results are intuitive. However, this algorithmic treatment does serve to bolster the repute of vaccinations, in terms of their effectiveness and their importance. Recall that without vaccination our model describes an unhindered wave of infection that spreads until it reaches the confines of the entire region. If this contagion has severe symptoms, this could be catastrophic, as history vindicates. Our model also serves to provide the machinery for a variety of other simulations for further study, to probe effects of interest and simulate particular situations should it be required.

VI. REFERENCES

White, S. H., del Rey, A. M., Sánchez, G. R. (2007). Modeling epidemics using cellular automata. *Applied Mathematics and Computation, 186,* 193–202.