A Space-Heterogeneous Stochastic SIR Model
Math 596 Project Summary – Spring 2016
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1 Overview

In general terms, an SIR model is one that describes the way a disease spreads through a population by measuring the number of people in the population who are Susceptible (S) to the disease, the number of people who are Infected (I), and the number of people who have Recovered (R). Typically the term SIR is used when the model allows for individuals in the population to move from susceptible to infected to recovered, and no other transitions are possible. There are several variations of this that are appropriate to model different situations; for example, one can find literature on SI, SIS, or SIRS models, in which case the corresponding transitions between states are allowed. One resource we’ve found particularly useful is a SIAM Review paper by Hethcote; see Primary Resources section for the citation.

The prototypical SIR model is one formulated in terms of systems of ODEs. In these models, one considers a system of differential equations for the susceptible population $s(t)$, infected population $i(t)$, and recovered population $r(t)$, all defined depending on a continuous time parameter $t \in [0, \infty)$. The ODEs are formulated depending on parameters that describe the severity of the disease (in terms of infectiousness and recovery time), interaction between individuals, and resilience of the population. Much can be done with the models given only an introductory ODE course, but there is also much more in depth analysis that is more challenging. The major drawback of these models is that a “well-mixing” assumption is necessary on the population in order to impose the ODE structure on $(s(t), i(t), r(t))$. (This “well-mixing” assumption is the same as the typical tank problems in ODE textbooks.) Roughly speaking, this assumption for the SIR model means that every member of the population is in contact with every other member, and any infected person has equal likelihood of infecting any susceptible person in the population; that the population is uniform in some sense. For obvious reasons, this assumption does not accurately model human interaction. One way to reformulate these SIR models to address spacial relations is to use PDE models related to the ODE ones. We take a slightly different direction here that does not require the mathematical background for PDEs (or even ODEs).

There are other versions of SIR models that are formulated in terms of stochastic models. The typical formulation of this model is to define random variables $S(t)$, $I(t)$, and $R(t)$, depending on either a discrete time parameter $t = 0, 1, 2, 3, \ldots$ or a continuous time parameter $t \in [0, \infty)$, where $S(t)$, $I(t)$, and $R(t)$ take values in a discrete state space $\{0, 1, 2, 3, \ldots, N\}$ (here $N$ is the size of the population, and often one imposes a fixed population condition of the form $S(t) + I(t) + R(t) = N$ for all $t$). A good resource for introducing such models is Allen’s book on stochastic processes in biology cited in the Primary Resources section. However, this formulation of the model suffers from roughly the same drawback as the ODE model described in the preceding paragraph. The model simply counts the number of people in the population that fall into each of the three states ($S$, $I$, and $R$), but lends no consideration for distinguishing how individuals interact with each other. Again, this amounts to a “well-mixing” type of assumption, which imposes unrealistic assumptions on the population and limits the interpretation of results to an averaged sense of how the disease spreads through the population.

The goal of this project is to build a discrete-time stochastic SIR model that accounts for varying interpersonal interactions in the population. We formulate our model in the following way. Suppose there are $N$ members in a population, each of which can occupy exactly one of three states: susceptible, infected, or recovered. We track the spread of the disease by computing the probability that each individual occupies each state at time $t = 1, 2, 3, \ldots$, given an initial infection profile of the population at time $t = 0$. In more technical terms, we model the state of each individual $i$ in the population at each time $t$ by a random variable $X_i(t)$ taking values of susceptible, infected, or recovered for $t = 0, 1, 2, \ldots$. We impose a Markov chain type structure on the model by requiring that the probabilities at
time \( t \) depend only on the state of the population at time \( t - 1 \); that is, the probability of an individual being susceptible/infected/recovered at time \( t \) depends on the state of the entire population at time \( t - 1 \), not on any previous states \( t = 0, 1, ..., t - 2 \). In this way, the model assigns a random variable to each member of the population, and evolves one time step at a time depending on only the more recent state of the population as a whole. The dependence of each random variable on the state of entire population in the preceding time step allows us to remove the “well-mixing” type assumption imposed in the previously mentioned models, and allows us to account for different probabilities of interaction between individuals in the population. We also impose an assumption that each individual interacts with at most one other individual in the population during a single time step. To model this, we introduce an interaction random variable for each individual describing their interaction at each time state. Hence the transition probabilities from state \( t - 1 \) to state \( t \) depend on these random variables as well. As long as the time step used is small enough, this is a reasonable assumption to impose on the model.

This model can be applied in several situations, and it provides a higher level of adaptability than the other models mentioned above. One way to visualize this model is to view each member of the population as a node on a graph, where weighted edges are connected between members of the population that have positive probability of interacting. The weights here represent the probability that the corresponding nodes come into contact inside a single given time interval. Then each node on the graph is given an initial state of susceptible, infected, or recovered, and the model evolves over time to compute the probability that each individual in the population is in each state. In application, this will account for the effects of a “highly connected” individual versus a relatively “secluded” one in terms of the spread of infection (for instance a toll booth attendant is “highly connected,” and a person who works from home is more “secluded”). Another way to apply this model is to designate each node in the graph to be a city or region, and compute probabilities based on proximity and population of the region relative to others. In this way, the model is easily adapted to model infections spreading through geographic regions that accounts for the distribution of people in the population.

### 2 Mathematical and Programming Content

To complete this project, a background in the following topics is recommended.

- **Systems of ODEs**: As a background to SIR models, the early stages of this project involves studying simple ODE versions of SIR models. This is a good introduction to the way SIR models work, and what relevant information SIR models provide. It also provides a nice motivation for the purpose of the model; to remove the well-mixing assumptions and add a stochastic transmission element to the model. Strictly speaking, this background could be omitted, but it does help to understand the project and put it into context.

- **Linear algebra**: Some familiarity with matrix operations and dot products is necessary for this project. Although, it does not require a rigorous understanding linear independence, linear combinations, bases, subspaces, diagonalization, etc.

- **Discrete probability**: A solid background of discrete probability is necessary for this project. In particular, it relies heavily on discrete random variables, probability laws, independence of random variables, and conditional probabilities.

- **Markov chains**: A background in discrete Markov chains is helpful as well. This can be limited to understand transition probabilities, evolving a Markov chain with the transition matrix, and how to use the Markov property in computing conditional probabilities. There is no need to cover things like long term behavior, stationary probability vectors, transient/absorbing states, or ergodicity of Markov chains, unless extensions involving these things are added to the model described above.
- Graph theory: A very rudimentary understanding of graph theory is helpful to understand the structure imposed on the population and interaction between individuals. This background can be limited to the basic familiarity with weighted graphs, connected graphs, and adjacency matrices.

- Programming: This project involves a fair amount of programming ability. A thorough understanding of working with vectors/matrices/arrays, decision statements, and loops are essential to implement simulations. For some of the applications a understanding of computer graphics is also helpful.

3 Primary Resources

For much of the mathematical content listed above, typical text books in the pertinent area are sufficient. Some additional resources on SIR models and specifically stochastic SIR models are the following (this is by no means a complete list).


4 Mathematical Description of the Project

Define \( S = (1, 0, 0), \ I = (0, 1, 0), \ R = (0, 0, 1), \) and \( S = \{S, I, R\} \). Assume that \( X_i(t) \) are random variables taking values in the state space \( S \) for \( i \in [N] = \{1, 2, \ldots, N\} \). Also let \( Y_i(t) \) be random variables on the state space \( [N] \) for \( i \in [N] \) and \( t \in \mathbb{N} \). The random variable \( X_i(t) \) is the state of person \( i \in [N] \) at time step \( t \in \mathbb{N}_0 \), and \( Y_i(t) \) is the person by whom person \( i \in [N] \) is affected at time step \( t \in \mathbb{N} \). We make the following assumptions on \( X_i(t) \) and \( Y_i(t) \):

- The random variables \( Y_i(t) \) and \( X_j(s) \) are independent for all \( i \neq j, \ t = 1, 2, 3, \ldots, \) and \( s = 1, 2, \ldots, t - 1, \) and there are constants \( \lambda_{i,j} \in [0, 1] \) for \( i, j \in [N] \) such that \( P(Y_i(t) = j) = \lambda_{i,j} \) for all \( i, j \in [N] \) and \( t \in \mathbb{N} \). For notation purposes, we collect these \( \lambda_{i,j} \)'s into a \( N \times N \) matrix \( \Lambda = (\lambda_{i,j}) \). This assumption imposes that the probability that person \( i \) interacts with person \( j \) at time \( t \) is independent of the state of any other person in previous time states. The \( \lambda_{i,j} \) parameters give the probability that person \( i \) interacts with person \( j \) in a given time state (when \( i = j \) this is interpreted as person \( i \) not having any interaction during the given time state). The matrix \( \Lambda \) is closely related to an adjacency matrix of the \( N \)-node graph representing the individuals of the population.
• There exist $\beta, \gamma > 0$ and $0 < r < 1$ such that for any $i, j \in [N]$ and $\mu, \nu \in \mathbb{S}$

$$P(X_i(t) = S | X_i(t - 1) = \mu, X_j(t - 1) = \nu, Y_i(t - 1) = j) = \begin{cases} 
\left( \nu(1) + \frac{\beta}{\beta + \gamma} \nu(2) + \nu(3) \right) \mu(1) & \text{if } i \neq j \\
\mu(1) & \text{if } i = j
\end{cases}$$

$$P(X_i(t) = I | X_i(t - 1) = \mu, X_j(t - 1) = \nu, Y_i(t - 1) = j) = \begin{cases} 
\frac{\gamma}{\beta + \gamma} \nu(2) \mu(1) + (1 - r)\mu(2) & \text{if } i \neq j \\
(1 - r)\mu(2) & \text{if } i = j
\end{cases}$$

$$P(X_i(t) = R | \vec{X}(t - 1) = \vec{\mu}) = r\mu(2) + \mu(3),$$

where $\mu = (\mu(1), \mu(2), \mu(3)), \nu = (\nu(1), \nu(2), \nu(3)) \in \mathbb{S}$. The parameters $\beta, \gamma > 0$ are such that $\frac{\gamma}{\beta + \gamma}$ is the probability that a susceptible person is infected during a single time interval, given an interaction with an infected person. Hence $\frac{\gamma}{\beta + \gamma}$ is the probability that a susceptible person is not infected during a single time interval, given an interaction with an infected person. The $r$ parameter is the probability that an infected individual recovers in a single time interval.

This model satisfies (by the assumptions imposed on $X_i(t)$ and $Y_i(t)$) the following Markov type property. For each $i \in [N]$, $t = 1, 2, 3, \ldots, \tau \in \mathbb{S}$, and $\vec{\mu}_0, \ldots, \vec{\mu}_{t - 1} \in \mathbb{S}^N$, we have

$$P(X_i(t) = \tau | \vec{X}(t - 1) = \vec{\mu}_{t - 1}, \ldots, \vec{X}(0) = \vec{\mu}_0) = P(X_i(t) = \tau | \vec{X}(t - 1) = \vec{\mu}).$$

The purpose of the model now is the simulate the evolution of the disease through the population by tracking the probabilities in the $3 \times N$ matrix

$$\vec{P}(t) = \begin{pmatrix} 
P(X_1(t) = S) & P(X_2(t) = S) & \cdots & P(X_N(t) = S) \\
P(X_1(t) = I) & P(X_2(t) = I) & \cdots & P(X_N(t) = I) \\
P(X_1(t) = R) & P(X_2(t) = R) & \cdots & P(X_N(t) = R)
\end{pmatrix} = \begin{pmatrix} 
p_{1,1}^t & p_{1,2}^t & \cdots & p_{1,N}^t \\
p_{2,1}^t & p_{2,2}^t & \cdots & p_{2,N}^t \\
p_{3,1}^t & p_{3,2}^t & \cdots & p_{3,N}^t
\end{pmatrix}$$

for each $t \in \mathbb{N}_0$. Using the assumptions above, one can show that

$$p_{1,i}^t = p_{1,i}^{t-1} \cdot \left( 1 - \frac{\gamma}{\beta + \gamma} \sum_{j \neq i} \lambda_{i,j} \cdot p_{2,j}^{t-1} \right),$$

$$p_{2,i}^t = p_{1,i}^{t-1} \cdot \frac{\gamma}{\beta + \gamma} \sum_{j \neq i} \lambda_{i,j} \cdot p_{2,j}^{t-1} + p_{2,i}^{t-1} \cdot (1 - r),$$

and

$$p_{3,i}^t = p_{2,i}^{t-1} \cdot r + p_{3,i}^{t-1}.$$

Hence, if we define

$$B_i^t = \sum_{j \neq i} \lambda_{i,j} \cdot p_{2,j}^t,$$

then we can compute $\vec{P}(t)$ from the $\vec{P}(t - 1)$ with the formula

$$\vec{P}(t) = \begin{pmatrix} 
p_{1,1}^t \cdot \left( 1 - \frac{\gamma}{\beta + \gamma} \cdot B_{1,1}^{t-1} \right) & \cdots & p_{1,N}^t \cdot \left( 1 - \frac{\gamma}{\beta + \gamma} \cdot B_{1,N}^{t-1} \right) \\
p_{1,1}^t \cdot \frac{\gamma}{\beta + \gamma} \cdot B_{1,1}^{t-1} + p_{2,1}^{t-1} \cdot (1 - r) & \cdots & p_{1,N}^t \cdot \frac{\gamma}{\beta + \gamma} \cdot B_{1,N}^{t-1} + p_{2,N}^{t-1} \cdot (1 - r) \\
p_{2,1}^t \cdot r + p_{3,1}^t & \cdots & p_{2,N}^t \cdot r + p_{3,N}^t
\end{pmatrix}.$$
In practice, we provide an initial distribution $\bar{P}(0)$, and use the equation for $\bar{P}(t)$ to evolve the distribution of $X_i(t)$ for $i \in [N]$ and $t \in \mathbb{N}$.

With similar techniques, we can formulate a stochastic heterogeneous SIS model. Assume that

$$P(X_i(t) = S|X_i(t-1) = \mu, X_j(t-1) = \nu, Y_i(t-1) = j) = \left\{ \begin{array}{ll} \left( \frac{\beta}{\beta + \gamma} \nu(2) + \mu(1) + r\mu(2) \right) & i \neq j \\ \mu(1) + r\mu(2) & i = j \end{array} \right.$$ \hspace{0.5cm}

$$P(X_i(t) = I|X_i(t-1) = \mu, X_j(t-1) = \nu, Y_i(t-1) = j) = \left\{ \begin{array}{ll} \frac{\gamma}{\beta + \gamma} \nu(2) + (1-r)\mu(2) & i \neq j \\ (1-r)\mu(2) & i = j \end{array} \right.$$ \hspace{0.5cm}

where $\mu = (\mu(1), \mu(2)), \nu = (\nu(1), \nu(2)) \in \mathbb{S}$, and it follows that the evolution of the SIS model is given by

$$\bar{P}(t) = \left( \begin{array}{ccc} p_{1,1}^{-1} \cdot \left( 1 - \frac{r}{\beta + \gamma} \cdot B_{1,1}^{-1} \right) + p_{2,1}^{-1} \cdot r & \cdots & p_{1,N}^{-1} \cdot \left( 1 - \frac{r}{\beta + \gamma} \cdot B_{1,N}^{-1} \right) + p_{2,N}^{-1} \cdot r \\ p_{1,1}^{-1} \cdot \frac{\gamma}{\beta + \gamma} \cdot B_{2,1}^{-1} + p_{2,1}^{-1} \cdot (1-r) & \cdots & p_{1,N}^{-1} \cdot \frac{\gamma}{\beta + \gamma} \cdot B_{2,N}^{-1} + p_{2,N}^{-1} \cdot (1-r) \end{array} \right).$$

### 5 Summary of Results

We ran several simulations to demonstrate the capability of this model. The first few examples are demonstrations that the model is working appropriately and how the spacial inhomogeneity can be recognized in the model. Later, we apply the model to more complicated models based on diseases spreading through the continental United States and through the world. In each simulation, we must specify several model parameters. In the simulations presented below, we choose these parameters to yield demonstrative and interesting results; they are not based on parameters for actual diseases. It should be noted that the definition of $\Lambda$ contains all of the information about the space heterogeneity. For trivial matrices $\Lambda$, the SIR model reduces to some of the known space homogeneous SIR models. Hence, this is one of the most important elements of the simulations below.

The first simulation we present is quick check that the model is working correctly in the space homogeneous setting. For this demonstration, fix parameters $N = 100, \beta = \gamma = 1, r = .1, \text{ and } T = 30$. The initial probability distribution $\bar{P}(0)$ was chosen so that approximately 90% of the population was susceptible and approximately 10% of the population was infected. The locations of the infected individuals was chosen at random. To collapse our simulation to the space homogeneous model, we define $\Lambda = (\lambda_{i,j})$ via $\lambda_{i,j} = \frac{1}{N-1}$ for $i \neq j$ and $\lambda_{i,i} = 0$. Below in Figure 1 is the matrix $\Lambda$ represented by pixel intensity (white represents larger values and black represents smaller values). In this case, everything off the diagonal in $\Lambda$ is constant. The plots to the right below show the plots of $\bar{P}(t)$, where $t$ is the horizontal axis, the probability of each person being susceptible, infected, and recovered are the vertical axis in the top, middle, and bottom graphs respectively. There is a line for each individual in the population in each of the three plots. It is a bit difficult to notice this because most of the probabilities coincide in the space homogeneous situation. In fact, given the uniform transitions given by $\Lambda$, this is the type of results that should be expected.

In the next two examples, we generate simulations is a similar fashion but with different ways to define $\Lambda$. For this demonstration, fix parameters $N = 500, \beta = \gamma = 1, r = .1, \text{ and } T = 30$. We define $\Lambda$ by making $\lambda_{i,i} = 1$ for all $i$ and randomly choosing $\lambda_{i,j}, \lambda_{j,i} > 0$ for approximately 75% of all possible connections. Then we modify $\Lambda$ so that the non-zero values in each row form a uniform distribution. In this way, our simulation represents an approximation of a the uniform population, but has several connections between individuals missing. The intuition here should be that since 75% of connection are present, the simulation should look similar to the uniform $\Lambda$ simulation from the previous paragraph. On the other hand, there is some variation introduced, and so one should expect the simulation to reflect that variation. This choice of $\Lambda$ and a SIR plot for this simulation are shown below in Figure 2. Note that the plots look almost identical to those in the simulation where $\Lambda$ was selected uniformly. However, it is now clear to see the different lines representing different individuals in the model.
We (essentially) repeat the last simulation, but choosing only 20% of the possible connections between individuals, rather than 75%. We also run this simulation with $N = 400$ instead of 500, but this change is irrelevant. The rest of the parameters are chosen to be exactly the same. The feature to note here is that there is even more variation in the SIR plots below in Figure 3. Since the simulated population is “less connected,” the spread of the disease through the population is less uniform. Hence the individual plots below are more differentiated.

In the next few simulations, we consider some SIR models on graphs that contain more spacial information. We consider a population of $N = 50$ individuals, numbered from 1 to 50. We impose a interaction construct on the population by constructing $\Lambda$ in the following way. Let each individual be connected to any other individual within 3 units of it, which can also be described by $\lambda_{i,j} > 0$ for $j = i - 3, i - 2, i - 1, i, i + 1, i + 2, i + 3$ as long as $1 \leq j \leq 50$. This interaction is pictured in Figure 4 below. Then $\Lambda$ is again normalized so that the non-zero entries of each row form a uniform distribution. The matrix $\Lambda$ constructed in this way is pictured below in Figure 5. This construction can be represented as the connected graph given below. We used this $\Lambda$, along with parameters $N = 50$, $\beta = \gamma = 1$, $r = .1$, and $T = 200$. Also, we set the initial distribution $\vec{P}(0)$ so that the first individual was infected, and others were susceptible. The results of the simulation are shown below. This simulation demonstrates the ability of this SIR model to represent spacial information about a population and it effect on the spread of a disease.

We present one more simulation in this section similar to the last one, but with a different initial probability distribution to demonstrate the impact of the initial distribution on this model. For this model, we take $N = 200$, $\beta = \gamma = 1$, $r = .1$, and $T = 140$. The initial distribution $\vec{P}(0)$ is set by infecting 4 people in the initial population. We set persons 10, 40, 90, and 140 to be infected, and leave all other individuals susceptible. The results of this simulation are shown below in Figure 6. It is not hard to recognize that the general shape the plots is very similar to the ones in the simulation with only the first individual infected, but there are places in the plots that appear to have higher concentrations of plots. This is caused by the spacial relationship between the initially infected individuals. In fact, it may be reasonable to expect to be able to recover the origins of the infection based on when increased/decreasing infections or recovery occur. In particular, there are 3 peaks in the infection plots that rise above the rest of the plot (occurring at about 30, 60, and 95). The times of these peaks can be an indication of the number of initial infected regions and the distance between them. If we think of each of the 4 initial infected individuals as a source of an “infectious wave,” these three peaks represent the times at which these waves collide. Hence 3 collisions would lead one to believe there are initially 4 infectious breakouts, as is the case here. There are videos available online at the [author’s webpage] demonstrating the spacial attributes of this simulation.
Our next applications of this model are to the spread of a disease through the continental United States population and world population. One of the challenges of this model is the task of constructing a graph theoretic model of the appropriate region and a matrix $\Lambda$ to represent the probability of transmission between regions. We use population density heat maps to construct these model parameters, which we describe now.

We obtained a population heat density map of the United States in 2010 from the United States Census Bureau at [census.gov](http://census.gov). With some preprocessing, formatting, and downsampling we modified this image to the one we used to construct our adjacency matrix $\Lambda$. The preprocessing here is superficial. We removed the text, changed the background color, and cropped off non-essential features. No population density values or relative locations were changed during preprocessing. Figures 7 and 8 below show the original population density heat map and the map after preprocessing. In order to identify “individuals” in our population, we make a search of the population density map in Figure 8 for the highest populated areas. Hence the individuals in our population are geographic regions, which we approximate to be no more than 10,000 square miles in area (compared to the 3+ million square miles in the continental United States, this appears to be a reasonable size for regions).

We identify $N = 500$ individuals (regions) for our model from the map in Figure 8 and now we must construct a matrix $\Lambda$ that identifies a spatial relationship between the regions. For these purposes, we borrow some intuition from Newtonian physics and gravitational attraction. For each region, we compute a population $p_i$ (by summing over the population densities in the region), and compute the paired distances $d_{i,j}$ between regions $i$ and $j$ by computing the distance between the center pixels of the two regions. Then we define our $\Lambda$ matrix by $\lambda_{i,j} = 0$ for all $i = 1, 2, ..., N$ and for $i \neq j$

$$\lambda_{i,j} = \frac{p_i \cdot p_j}{\tau_j d_{i,j}^2}, \quad \text{where} \quad \tau_j = \sum_{i \in [N], i \neq j} \frac{p_i \cdot p_j}{d_{i,j}^2}.$$

This is the “gravitational” force between two regions if one interprets the population of a region as its mass, and if one takes gravity such that the total gravitational force acting on a region as 1. Of course, these analogies give no rigorous way to analyze the spread of disease or the movement of people, but it seemed to us to be a reasonable way to simulate these things. Note also that our construction imposes $\lambda_{i,i} = 0$, which means that every region interacts with another region in each time interval. We also acknowledge a shortfall of our model in terms of computing the distances between regions. We do not account for the distortion of distances due to the Mercator projection used to make the map. Distances are computed as Euclidean distances in the plan as they appear on the maps. Figure 9
shows results of our simulation of the spread of a disease through the continental United States with \( N = 500 \), \( \beta = 1 \), \( \gamma = 5 \), \( r = .2 \), \( T = 40 \), \( \Lambda \) constructed as above, and 3 randomly selected infected regions for the initial distribution. Below in Figure 10 is a second simulation with different parameters, \( N = 500 \), \( \beta = 2 \), \( \gamma = 3 \), \( r = .2 \), and \( T = 40 \). The matrix \( \Lambda \) is constructed in the same way, and several regions in the northeast of the United States were infected initially. The main difference we want to highlight here is the long term behavior of the SIR plots in this simulation. Note that as \( t \) gets large, the susceptible plot does not tent to 0 and the recovered plot does not tend to 1. This is different behavior than all of the previous simulations, and different from the possible behavior of any of the space homogeneous models. Based on the spacial relationship between the individual regions of this model, it is possible for the probability of a region to remain susceptible to remain bounded away from 0 in the long run. In this situation, it appears that the recovery rate \( r = .2 \) is large enough that regions can recover fast enough as to reduce the probability of spreading the disease to neighboring regions.

We also ran SIS simulation for the United States maps above and SIR/SIS simulations for a world population constructed from the world population density heat map in Figure 11. More plots, pictures, and videos related to these simulations are available at the **author’s webpage**.

## 6 Possible Extensions

SIR models have been used in many situations, and they have been varied and extended in many different ways (e.g. SI, SIS, or SIRS models or models that incorporate birth/death rates, incubation periods, and/or quarantines). They have also been used to assess attributes of given populations like herd immunity, susceptibility to
epidemics/pandemics, and likelihood of endemic diseases versus eradication. Almost all of these directions of study appear to be viable for this SIR model. Now that we have introduced a model with an inhomogeneous population, one can also consider a graded population, where some individuals are more susceptible to the disease than others or quicker to recover than others (this may be useful in modeling the spread of the flu through populations with different ages, for instance; it is generally accepted that individuals over 65 are significantly more susceptible to the flu). These modifications can be made by allowing the parameters $\beta$, $\gamma$, and $r$ to vary person to person; that is, one can adapt the model to assign a particular $\beta_i$, $\gamma_i$, and $r_i$ for $i = 1, 2, ..., N$ to describe each individual's susceptibility to and ability to recover from the disease.

7 Note From the Author

This is a student project from the Math and Biomedical Research course, taught by the current author Jarod Hart, offered at the University of Kansas in the Spring of 2016. Some modification and additions were made to the original project for this summary. The course is supported by the Initiative for Maximizing Student Development (IMSD) through an NIH grant NIH-NIGMS 5R25GM062232. The PIs of this IMSD grant are Professors Estela Gavosto (Mathematics Department) and James Orr (Biology Department). We are happy to share these project ideas, and welcome those who are interested to use them. We'd love to hear about your results and extensions related to these projects, and in some cases, will provide some support for the projects. Please contact Jarod Hart at jvhart@ku.edu with any typos, errors, questions, or comments about this project summary.
Figure 6: left - picture of $\Lambda$, defined by connecting nearest three neighbors. right top - susceptible population, right middle - infected population, right bottom - recovered population.

Figure 7: Population density map of the Unitized States from the United States Census Bureau, available at census.gov.

Figure 8: Processed, formatted, and subsampled image of US population map used to construct the adjacency matrix $\Lambda$. 
Figure 9: left - picture of Λ, constructed using the continental United States population density map. right top - susceptible population, right middle - infected population, right bottom - recovered population.

Figure 10: left - picture of Λ, constructed using the continental United States population density map. right top - susceptible population, right middle - infected population, right bottom - recovered population.

Figure 11: Preprocessed world population density map from Wikipedia.org, original available at [this link](https://www.wikipedia.org).