Stochastic Models for Epidemics on Networks

by

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Submitted to the Department of Electrical Engineering and Computer Science
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Abstract

In this thesis, I looked at an extension of the Reed-Frost epidemic model which had two-sub-populations. By setting up a Markov chain to model the epidemic and finding the transition probabilities of that chain, MATLAB could be used to solve for the expected number of susceptibles and the expected duration. I simulated the model with more than two sub-populations to find the average number of susceptibles and reviewed previously solved stochastic spatial models to understand how to solve the multiple-population Reed-Frost model on a network.

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Contents

1 Introduction 9

2 Dual Population Reed-Frost with Replacement 13
   2.1 Effect of Parameters on Model ........................................ 16
   2.2 Expected Duration ..................................................... 19

3 Multiple Population Reed-Frost with Replacement 23

4 Discussion of Spatial Modeling 27
   4.1 Background Models ................................................... 27
   4.2 Mean-Field ODE’s Single Attracting Fixed Point .................. 28
   4.3 Mean-Field ODE’s, Two Locally Attracting Fixed Points .......... 29
   4.4 Mean-Field ODE’s, Periodic Orbits ................................. 30
   4.5 Classifying Two-Sub-Population Reed Frost Model ............... 31

5 Conclusion 33

A Reed-Frost Equations 35
List of Figures

2-1 Expected number of susceptibles varying c. .......................... 17
2-2 Expected susceptibles with varied general population incidence rates. 17
2-3 Expected susceptibles with different non-infection rates. ............. 18
2-4 Expected susceptibles in population A given size of population B. . 19
2-5 Expected Duration for Population Size of A ............................ 21

3-1 Layout of Populations ....................................................... 24
3-2 Sample path and average path with $c = 0.85$, $\gamma = 0.8$. ............ 25
3-3 Sample path and average path with $c = 0.98$, $\gamma = 0.8$. ............ 25
3-4 Average path for population A with $c = 0.5$ and $\gamma = 0.2$. .......... 25
Chapter 1

Introduction

There has been much work on creating realistic epidemic models. The most basic models are the deterministic models for simple or general epidemics [2]. Epidemics occur when the members of the population are in one of two groups: susceptible or infected. When the infected individuals can also spread the disease they are also known as infectives. In a simple epidemic, once a person is infected he continues in that state indefinitely. In general epidemics, infected members get removed from the population by either death or immunization. The deterministic models assume knowledge of the pairwise infective rates for the population and from that try to ascertain the general infection rate and the duration of the epidemic. Especially where the epidemic model is deterministic, it should be noted that in many cases the size of the susceptible population goes to zero. Thus, the duration of the epidemic, not the steady state value, is important. There are many extensions to these models that have been explored including spatial variations and probabilistic infection rates.

By adding the probabilistic infection rates, the deterministic models are converted to stochastic models. There are two main discrete stochastic models: the Greenwood model and the Reed-Frost model [2]. In both these models an individual can be either susceptible or infected. Time is broken up into discrete intervals modeling the different generations of the disease. During each interval there is a probability that a susceptible becomes infected. The number of susceptibles in the next time slot is a binomial random variable if we assume that each susceptible is independently
becomes infected with some probability. The probability a susceptible is not infected is called the non-infection rate. The non-infection rate is used as a parameter in the binomial distribution to figure out the number of susceptibles in the next time period.

The difference between the two models is how the disease is spread and thus the probability of non-infection. In the Greenwood model the spreading of the epidemic does not depend on the number of people in the population who are infected. This means that the probability of non-infection is constant. The constant non-infection rate models cases, such as food poisoning, where the epidemic is in the environment rather than the infected individuals. In the Reed-Frost model, infection comes from interaction with infective members so that the non-infection rate depends on the number of people infected. This would include cases such as AIDS, measles, and the flu. For the basic Reed-Frost model it is assumed that a person is equally likely to run into each member of the population. If contact is made with an infective person, there is some probability of the person not catching the disease. Thus the non-infection probability for determining number of susceptibles in the next time step looks like a constant raised to the number of infectives. From these two basic models a number of extensions have been explored, including: pair-wise mixing, which assumes that at each time step each person meets exactly one other person; and extending the states so that an individual can be susceptible, infected or resistant.

There is already in the literature an extension of the Greenwood model to a model of a single sub-population drawing from a general population [2]. In this model the general population has an inexhaustible supply of people with a certain fixed rate of incidence of the disease. At each time period the infected people in the sub-population are replaced with a random group from the general population. They then interact according to the Greenwood model with members of the sub-population for the next time step. The conclusion of the model is that if the rate of infection and the incidence rate of the general population are high enough, the epidemic could be sustained for long periods of time in the sub-population. This was originally used to model intravenous drug users with HIV. As the users died, new ones were recruited from the general population. The general population was much larger than
the sub-population so that the recruiting did not affect the incident rate in it.

Some epidemic models such as the Susceptible-Exposed-Infected-Removed (SEIR) have corresponding deterministic and stochastic models [1]. The SEIR model extends the basic epidemic models by adding allowing an individual to be not only susceptible, infective, or removed, but also exposed. The removed member of the population is immune to the disease. This occurs in such diseases as measles, where if the infected survives they cannot contract the disease again. The exposed individual is not transmitting the disease, but has already been infected with it. Also, the model has extra parameters to account for the birth and death within the population. The stochastic model breaks time down into small pieces, where at any given point in time the susceptibles, infectives, resistives, or exposed can increase or decrease by one.

Models with corresponding deterministic and stochastic parts can be analyzed by looking at an appropriate set of deterministic equations [3]. Converting the stochastic models to a related deterministic set of ODE's, one can draw, conclusions about the stochastic system based on the behavior of the ODE's.

An outline of the rest of this document is as follows. Chapter 2 examines a Reed-Frost model of epidemics with two sub-populations. After setting up the model, the chapter goes on to explore the relationship between the parameters and the expected number of susceptibles. Chapter 3 then looks at an extension of the Reed-Frost model to include an arbitrary number of sub-populations, specifically examining the case of four sub-populations spread out in a line. Finally Chapter 4 reviews previous spatial stochastic models and the results gain by viewing their corresponding ODE's.
Chapter 2

Dual Population Reed-Frost with Replacement

The sub-population Greenwood model discussed in the Introduction can be extended to the case of a sub-population using the Reed-Frost model of infection. Appendix A outlines how this case can be set up as a Markov chain.

To further expand the model, a second sub-population is added. In this extension the general population is denoted by $G$ and the two sub-populations are $A$ and $B$. The number of susceptibles for each sub-population are $X^A$ and $X^B$ respectively; the number of infectives are $Y^A$ and $Y^B$. For simplicity, each sub-population is assumed to have size $N$ so that at each point in time $X_t^A + Y_t^A = N$ and $X_t^B + Y_t^B = N$. In each time period $t$ the infected sub-populations are replaced by a random selection from the general population, which has an incidence rate of $p$ for the disease. Note that the general population is assumed to be infinite so that the incidence rate remains constant over all time. Next the two sub-groups mix, with a non-infection rate $c$ between two people from different sub-groups, and a non-infection rate $\gamma$ within the same sub-group. The non-infection rate encompasses not only the probability of not coming into contact with an infected individual, but also the probability that of not being infected despite contact with an infected person. The number of infectives and susceptibles at the end of this process are then assumed to be the number at the beginning of the next time period.
We can model this with two coupled chains, each of which keeps track of the number of susceptibles in one sub-population; the overall model constitutes a Markov chain although we avoid connection the full transition matrix explicitly. Note that for each sub-population we need only keep track of either the susceptibles or the infectives, because the size each of the groups is \( N \). It is not necessary that the two sub-populations have equal size to create the Markov chain, only that the size remain constant. Each time period is broken down into two updates. The first is when the infectives are replaced from the general population. The new number of infectives and susceptibles are respectively denoted \( Y_t^{A'} \), \( Y_t^{B'} \) and \( X_t^{A'}, X_t^{B'} \). Using \( \text{bin}(n, p) \) to denote the binomial distribution where the number of independent trials is \( n \) and the probability of a success is \( p \), the first update equations are:

\[
Y_t^{A'} = \text{bin}(Y_t^A, p) \\
Y_t^{B'} = \text{bin}(Y_t^B, p)
\]

In the second update the populations mix with themselves and each other. The probability that two people from the same population meet and are not infected are in the non-infection rate. Because the mixing is assumed to be homogenous, each person meets every other person with the same probability, and the meetings are independent. Using that the probability of non-infection within a sub-population is the same for every infective leads to the probability that any susceptible is not infected by a person of the same group is \( \gamma_t^{A'} \). Thus, the second update equations are:

\[
X_{t+1}^A = \text{bin}(X_t^A, c_t Y_t^{B'} \gamma_t^{A'}) \\
X_{t+1}^B = \text{bin}(X_t^B, c_t Y_t^{A'} \gamma_t^{B'})
\]

Using these two pairs of equations we can calculate a transition matrix \( P^A \) and \( P^B \).
for each population respectively to propagate the probability distributions of $X_t^A$ and $X_t^B$ forward in time. If both populations have a size of $N$, then $P(X_t^A|X_t^A)$ and $P(X_t^B|X_t^B)$ are shown by the first pair of equations to be given by:

$$R1 = \begin{pmatrix}
    p^N & (\binom{N}{N+1})pq^{N-1} & \cdots & (\binom{N}{1})pq^{N-1} & q^N \\
    \vdots & \ddots & \ddots & \ddots & \ddots \\
    \vdots & \cdots & \ddots & \ddots & \ddots \\
    \vdots & \cdots & \ddots & \ddots & \ddots \\
    \vdots & \cdots & \ddots & \ddots & 1
\end{pmatrix}$$

From the second pair of equations we can generate a probability matrix, $R2^A$, for $P(X_{t+1}^A|X_t^A, Y_{t}^B)$:

$$R2^A = \begin{pmatrix}
    1 \\
    (1 - \gamma^{N-1}c_{Y_{t+1}^B}) & \gamma^{N-1}c_{Y_{t+1}^B} & \cdots & \cdots & \cdots \\
    \vdots & \ddots & \ddots & \ddots & \ddots \\
    \vdots & \cdots & \ddots & \ddots & \ddots \\
    \vdots & \cdots & \ddots & \ddots & \ddots \\
    (1 - \gamma^{N-N}c_{Y_{t+1}^A}) & \gamma^{N-N}c_{Y_{t+1}^A} & \cdots & \cdots & \cdots & (\gamma^{N-N}c_{Y_{t+1}^A})^{N-1}
\end{pmatrix}$$

There is a similar matrix $R2^B$ which calculates $P(X_{t+1}^B|Y_{t}^A, X_t^B)$:

$$R2^B = \begin{pmatrix}
    1 \\
    (1 - \gamma^{N-N}c_{Y_{t+1}^A}) & \gamma^{N-N}c_{Y_{t+1}^A} & \cdots & \cdots & \cdots \\
    \vdots & \ddots & \ddots & \ddots & \ddots \\
    \vdots & \cdots & \ddots & \ddots & \ddots \\
    \vdots & \cdots & \ddots & \ddots & \ddots \\
    (1 - \gamma^{N-N}c_{Y_{t+1}^B}) & \gamma^{N-N}c_{Y_{t+1}^B} & \cdots & \cdots & \cdots & (\gamma^{N-N}c_{Y_{t+1}^B})^{N-1}
\end{pmatrix}$$

Using $P(X_{t}^A|X_{t}^A)$ as given above and $X_{t}^A + Y_{t}^A = N$, we know $P(Y_{t}^A|X_{t}^A)$. Then by summing over the conditioned probabilities we can find $P(Y_{t}^A)$. The same technique can be used to find $P(Y_{t}^B)$. Finally using the conditioned probabilities we get:

$$P^A = R1(\sum_{i=0}^{N} R2^A(Y_{t}^B = i)P(Y_{t}^B = i))$$
\[ P^B = R1 \left( \sum_{i=0}^{n} R2^B(Y_t^{A'} = i)P(Y_t^{A'} = i) \right) \]

2.1 Effect of Parameters on Model

By running the Markov model in Matlab, we can see the effects of different variables on the system. Using the transition matrices given above and solving for the steady state values, we can find the expected number of susceptibles and infectives for each population. For this example, using 50 people in each sub-population and varying the non-infection rates between the populations, we can see how different \( \gamma \) and \( c \) affect the expected number of susceptibles. In this example the non-infection rates were taken to be the same for the two sub-population groups, with \( \gamma \) set to 0.9, 0.5, and 0.1, and with \( c \) varying as shown in Figure 2-1. The expected number of susceptibles increased exponentially with \( c \). This is as expected because the probability of infection decreases as \( c' \). As the non-infection rate \( \gamma \) within the group increased, so did the expected number of susceptibles, because they were less likely to be infected by a member of their own population. What is interesting is that the expected number of susceptibles for each value of \( \gamma \) is essentially the same and constantly close to 0 for values of \( c \) less than 0.85. Because the total non-infection rate for each population is the product of \( \gamma \) and \( c \), when \( c \) is small and raised to a high power the effect of it can dominate that of a higher \( \gamma \) raised to a lower power.

One would expect that as the incidence in the general population increased the expected number of susceptibles in the sub-populations would decrease. This is not always the case, as seen in Figure 2-2. In this example, the two sub-populations had a size equal to 20, and \( \gamma = c \). Notice that at each level of \( p \) the higher the non-infection rate, the more susceptibles there were. For each \( \gamma \) there is a critical level of \( p \) where the expected number of susceptibles declines. However, up until that point the increasing \( p \) actually leads to a higher number of susceptibles. This is because it is more likely that the person will get infected from their interactions with the other 39 people than be infected from the general population.
Figure 2-1: Expected number of susceptibles varying c.

Figure 2-2: Expected susceptibles with varied general population incidence rates.
Another example looks at varying $c$ between the two sub-populations. In Figure 2-3 are the expected number of susceptibles in steady state for each population. In this case the value of $c$ for the sub-population $B$ was 0.1, 0.5, and 0.9 in the three examples, while the value of $c$ for population $A$ was varied. The non-infection rate within a sub-population group, $\gamma$, was held constant at 0.1. With an incidence rate of $p = 0.1$ in the general population, the two expected populations cross at the point where the both $c$’s are equal. Whichever sub-population has the higher rate of non-infection has more susceptibles. In some way the healthy sub-population is pulling up the other sub-population when the non-infective rate is higher than $\gamma$, the self-mixing rate.

**Unequally sized populations**

Consider the case where the two different sub-populations have different sizes. Let the larger population, in this case population $A$, have a non-infection rate of 0.8 for within the sub-population and 0.95 for interacting outside the sub-population. Let population $B$ will have a non-infection rate of 0.1 within and 0.25 outside. We now solve for the expected number of susceptibles with the population of $A$ being 150 and the size of population $B$ varying as shown in Figure 2-4. Notice that the expected
The number of susceptibles in population $A$ decreases as the population of $B$ increases. This decrease would be expected as population $B$ interacts with a higher number of infective people each time. The effect of population $B$ on the expected number of susceptibles in $A$ is not linear. The reason for the non-linearity is that the distribution of susceptibles in population $A$ is a binomial whose probability depend on the number of infectives in population $B$ and $A$. Because the number of infectives in $B$ increase as the population size of $B$ increases, the larger size of $B$ decreases the non-infection rate, which in turn increases the number of infectives in $A$. Both these increases allow the effect to be more than simply linear.

### 2.2 Expected Duration

The expected numbers of infectives and susceptibles are important for determining the size of the epidemic. In the long run the epidemic will die out when both sub-populations are completely made up of susceptibles. When this happens there are no infectives to replace from the general population and thus no way to spread the infection. The question to be asked then is how long will it be before the epidemic dies out?

It is possible to find the expected duration of the two-sub-population model using the full Markov chain, which we avoided explicitly constructing in the previous section.
The two chains representing the susceptibles in each group need to be explicitly combined into a single Markov chain. Each state in the new chain is \((X^A, X^B)\) for all values of \(X^A\) and \(X^B\). Assuming that population \(A\) has size \(N^A\) and population \(B\) has size \(N^B\), the total number of states in the Markov chain is \(N^A N^B\). In terms of the Markov chain the duration of the epidemic is the number of steps taken before absorption in the final state of \((N^A, N^B)\). The solution to the expected duration is given by the value of \(v\) where \(v = r + [P]v\). Hence \([P]\) is the transition matrix for the Markov chain and \(r\) is the column vector of all ones, except for an entry of 0 corresponding to the state \((N^A, N^B)\), then the \(i\)th component \(v\) is the expected number of steps to absorption, assuming we start in state \(i\) [4].

\([P]\) can be created using a similar method to the method used in the preceding section employing conditional densities. Given \(Y^A_t\) and \(Y^B_t\) then:

\[
P(Y^A_{t'}, Y^B_{t'}|Y^A_t, Y^B_t) = P(Y^A_{t'})P(Y^B_{t'}|Y^B_t)
\]

Similarly, given \(Y^A_{t'}\) and \(Y^B_{t'}\)

\[
P(X^A_{t+1}, X^B_{t+1}|Y^A_{t'}, Y^B_{t'}) = \text{bin}(X^A_{t'}, \gamma^A_{t'} c^B_{t'}) \text{bin}(X^B_{t'}, \gamma^B_{t'} c^A_{t'})
\]

The first of the above equations is a consequence of the fact that in the replacement stage of the epidemic the two populations are independently drawing from the general population. Independent drawing ensures that \(Y^A_{t'}\) and \(Y^B_{t'}\) are independent. When conditioning on the new number of infectives, solving for the number of susceptibles at the next time epoch is also independent. Solving for susceptibles after the interaction with infectives is shown in the second of the two equations above. By weighting the probabilities in the second equation by the probabilities in the first equations we come up with the following expression for \(P(X^A_{t+1}, X^B_{t+1}|Y^A_t, Y^B_t)\).

\[
P(X^A_{t+1}, X^B_{t+1}|Y^A_t, Y^B_t) = \\
\sum_{(i,j)} P(X^A_{t+1}, X^B_{t+1}|Y^A_{t'} = i, Y^B_{t'} = j) P(Y^A_{t'} = i, Y^B_{t'} = j|Y^A_t, Y^B_t)
\]
Note that conditioning on $Y_t^A, Y_t^B$ is equivalent to conditioning on $X_t^A, X_t^B$ since $X_t^A + Y_t^A = N$, $X_t^B + Y_t^B = N$. Using MATLAB to solve for $u$ defined earlier, Figure 2-5 shows how the original size of the population $A$ affects the expected duration. Solving using $\gamma = 0.8$ and $\gamma = 0.1$ for the non-infection rates for populations $A$ and $B$ respectively, with $c = 0.95$ for population $A$ and $c = 0.25$ for population $B$, the size of the population $B$ was held constant at 5 while the population of $A$ was varied as shown. When $A$ increased, the duration of the epidemic increased almost linearly (at least for small values), as shown in Figure 2-5. Notice that using the same non-infection rates the expected number of susceptibles dropped off closer to exponentially.
Chapter 3

Multiple Population Reed-Frost with Replacement

One way to enhance the model is to add more sub-populations and adjust the non-infection rates among them to reflect a network structure. The higher two non-infection rate between the sub-populations, the less coupled they are. Any two sub-populations having a non-infection rate of 1 with each other can be seen as having no direct path between them. As stated in the last chapter, the calculation of expected duration becomes more complicated. This is because the Markov chain would have to keep track of all the states of each population: the state of the Markov chain becomes an $L$-tuple, $(X^A, X^B, \ldots, X^L)$. It is clear that if each set has a population size $n$ and there are $L$ sub-populations we would need $O(n^L)$ states. Although computing the expected duration from the Markov chain is computationally intensive, simulating large populations is not. Each simulation provides a sample path for the number of susceptibles and infectives. By running the simulation multiple times and averaging over all of them, we can empirically deduce the average behavior.

We have carried out one such set of simulations for four populations strung out in a line, as in Figure 3-1, with each population having a total size of 100. Each population replaces its infectives with a random selection from the general population whose incidence rate for the disease is $p = 0.1$. We set $\gamma = 0.8$ and $c = 0.85$ for all populations in the simulation shown in Figure 3-2, while $\gamma = 0.8$ and $c = 0.98$ for all
populations in the simulation shown in Figure 3-3. For both simulations the population A starts out completely infected and the other populations start out completely susceptible. As expected, the epidemic spreads out over time. The less coupled the populations are, the longer it takes for the infection in A to reach population D. This can be seen in Figure 3-2 and Figure 3-3 by the time at which the average number of susceptibles of population D goes below 100. Also, notice that the less coupled the populations are the higher number of average susceptibles in the long run. We would expect this because the probability for infection decreases as c increases. Thus, isolation works to curtail the epidemic.

Another situation to look at is where one population is isolated but very susceptible to the disease, as in the simulation shown in Figure 3-4. In this case for population A we set $\gamma = 0.2$ and $c = 0.5$. All the other populations have $\gamma = 0.95$ and $c = 0.98$. Again population A started out completely infected and all other populations were completely susceptible. Even with such extreme values the epidemic in A completely spreads to population D. Notice that the average size of susceptibles of population D does not drop below 100 before four steps in time whereas in the previous simulation it was less than three steps. Thus, the spread of the epidemic takes longer with the isolated population. Notice also that the population A is almost always entirely infected and this affects the other three populations. As we would expect the population B is most affected by its interactions with population A and thus has a lower number of susceptibles than C and D. Similarly since population C is closer to A it has a lower number of susceptibles than population D.
Figure 3-2: Sample path and average path with $c = 0.85$, $\gamma = 0.8$.

Figure 3-3: Sample path and average path with $c = 0.98$, $\gamma = 0.8$.

Figure 3-4: Average path for population A with $c = 0.5$ and $\gamma = 0.2$. 
Chapter 4

Discussion of Spatial Modeling

Looking at the two sub-population Reed-Frost models discussed in Chapter 3, we would like to be able to predict the long term behavior of the model by looking at previous results in stochastic spatial modeling. In Durrett’s paper, “Stochastic Spatial Models” [3], he first reviews several stochastic models that have been solved: the Ising model, simple exclusion process, and the stepping stone model. Then he looks at the stochastic models by finding their corresponding deterministic mean field equations. By looking at the solutions to the mean-field ODE’s, a statement can be made about the steady state solution to the stochastic model. Mean-field ODE’s are the deterministic equations assuming that all neighboring sites are independent. Durrett comes up with three cases: attracting fixed point, two locally attracting fixed points, and periodic orbits.

4.1 Background Models

The first of the background models is the Ising model, which was originally used to study magnetism. It contains a lattice where each node can be in one of two states. The change in state is determined by the percentage of neighbors with the same state. Unlike the biological systems, this model is time reversible so that looking backwards and forwards are the same.

The next model was the simple exclusion process which, similar to the Ising model,
is a two state system where each node is either occupied or empty. The model simulates the movement of particles in a grid where each particle may move to another node only if the new node is empty. Because the particle has an equal chance of moving each direction, its path can be modeled by a random walk. By assuming each node acts like it is independent of all others, then there exists a stationary distribution for each node being occupied. Like the Ising model the exclusion process is also time reversible.

The final model reviewed is the stepping stone model, again situated on a lattice with nodes either being occupied or empty. For each node that is occupied there is assumed to be a single individual who can be one of \( k \) types. The individuals dies off with rate one. When the node becomes empty it is filled with the same type of individual as a surrounding state. That is to say, if node \( x \) becomes empty and then the probability that it is replaced by the state of node \( y \) is \( p(y - x) \) where \( p \) is a function of distance. Using the probability to be \( p(y - x) = \frac{1}{2^d} \) with \( y \) and \( x \) nearest neighbors and \( d \) is the dimension of the grid, it was found that the model converged for \( d \leq 2 \).

4.2 Mean-Field ODE’s Single Attracting Fixed Point

When the mean-field ODE’s have a single attracting fixed point, then there is a stationary distribution for the corresponding stochastic model where all the states have a strictly positive density. If we consider each state to be a single type of species then this condition is equivalent to coexistence, since each state has a positive probability of not being zero. For instance if we have a ecological system like a field, where location corresponds to nodes in the spatial model and species such as grass, bushes, or trees are the states. Now at any point in time there is in each location either grass, bushes, or trees. Each species dies or gives birth with some probability. If the node \( x \) gives birth, then it sends its offspring to a nearest neighbor \( y \), with probability \( p(y - x) \) and will take over the node if the species is dominant. In the field model, trees dominate all others and bushes dominate grass. Now coexistence
in the probabilistic sense means that for any node the steady state probability that a node will be grass, bushes, or trees is positive. Because each node is independent and has the same distribution, if the steady state probability of grass at one node is 0 then the steady state probability of grass at all nodes is 0. Thus, in the long run there will be no grass and so there is no coexistence.

Demonstrating that if the mean-field ODE’s have a single attracting fixed point, then there are solutions for coexistence is the contact process, which is similar to the stepping stone model above. In a contact process, though, the deaths occur at rate $\delta$ and each node produces an individual at rate $\beta$. The individual then goes to another node $y$ with probability $p(y - x)$ and if $y$ is unoccupied then the state of $y$ changes to the state of $x$, otherwise nothing happens. Durrett shows that the probability distribution predicts the same coexistence conditions as the mean-field ODE in the case when the number of nodes in the contact processes goes to infinity. Also, coexistence conditions hold for both ODE’s and probabilistic models when rapid mixing is added. In rapid mixing two sites have a probability of swapping states with a probability related to their distance apart. The well-known predator-prey is model is a contact process with rapid mixing where the probability of a predator state switching to a prey state is zero. But there is a positive probability of a prey state being switched to a predator state. The rapid movement comes into the equation when prey and predator, through migration, are likely to switch locations.

4.3 Mean-Field ODE’s, Two Locally Attracting Fixed Points

The second case is where there are two locally attracting fixed points. In this case there is in the stochastic system a single equilibrium whereas in the ODE’s there are two equilibria. Because one of the equilibria is more likely, it is able to dominate in the long term. As long as the system starts with a positive density such that each population is represented, the system will go to equilibrium. The ODE’s can be used
to figure out which one that is. Durrett then uses this result in the quadratic contact process; the ‘quadratic’ refers to fact that a birth happens only when there exist two occupied neighbors that are diagonal. He then uses the ODE’s to solve for the critical values to decide the coexistence on both finite and infinite sets. There is also a sketched proof of the rates when there is rapid mixing.

The same type of mean-field solution exists when modeling two bacterial populations in which one can produce colicin to kill the other. Solving the ODE’s for equilibria gives three, of which two are sinks and one is a saddle point. This suggests that whichever population is dominating at the beginning will be dominant in the end. But it is interesting to note that the critical points for the deterministic model are not the same as for the probabilistic model. Then the model evolves to a three-species colicin model such that two species produce a colicin and are immune to it and the third species is not. Unlike the two-species colicin model for which the ODE’s and spatial model agree as to what coefficients led to coexistence, the three species model does not.

4.4 Mean-Field ODE’s, Periodic Orbits

For the last case, Durrett showed that when the ODE’s had periodic orbits, the spatial model had different behaviors, depending on the scaling. With small scaling there were large swings in the densities; with moderate scaling there were moderate swings in the densities; and with large scaling the densities were nearly constant. His first example for this case was the multitype biased voter model. This model is similar to the Ising model given above, except now each node can be one of many types. If one of the species has a greater birth and dispersion rate than every other species, then in the long run the first species will dominate. The probability of this can be shown to go to 1 when time goes to infinity.

Also having the same mean-field solutions is the cyclic voter model. The cyclic voter model is an extension of the contact process. When an individual gives birth and sends its offspring to a new site, instead of dying if the site is occupied the
offspring may take over the site. There is a hierarchy to the states such that state $i$ can take over state $j$ if $i \geq j$. In a cyclic voter model if we have states $1, \cdots, k$ then $k \leq 1$ in addition to the previous rules leading to cycle. Durret conjectures that for all birth rates greater than zero there will be coexistence of all the species.

Moving to an example from game theory shows that the hawks and dove model has many of the same properties. The hawk and dove game works by having an interaction matrix which then yields the net birth rates of both species. The two species are then dropped on a grid where they can move by one space at a given rate. Using the ODE's we can predict what sort of interaction matrices lead to coexistence in the system.

The next model in the paper is the epidemic with regrowth of susceptibles where individuals can be either infected, susceptible, or resistant. Durrett states that if the epidemic without regrowth does not die out, then the epidemic with regrowth continues indefinitely. Finally the last model he looks at is WATOR, which is a continuous-time predator-prey model with stirring. What's interesting here is that he uses the spatial model to figure out the directional arrows on the ODE's equilibrium solutions.

4.5 Classifying Two-Sub-Population Reed Frost Model

The discussion on stochastic spatial modeling may be able to be used in solving questions about the multiple populations using the Greenwood replacement model discussed in Chapter 3. However an essential difference from the epidemic models discussed is that the nodes in the spatial model would be the entire populations instead of an individual. Assuming that all populations had size $N$, we could model the state of each node by the number of susceptibles such that each site could have states 0 through $N$. This only works assuming that the infectives die out and are replaced each turn. Part of the difficulty of this is that for each time step there are two transitions, one to replace infectives and one to circulate the epidemic.
Chapter 5

Conclusion

We started by looking at a two-sub-population Reed-Frost model that had replacement from a general population. From the model we derived a Markov chain and used MATLAB to solve for the transition matrices. Using these matrices to solve for the expected number of susceptibles in steady state and the expected to duration, we could see the effects of the parameters on the model. One surprising result was that as the incidence rate of the general population increased so did the expected number of susceptibles.

Next, we extended the model to an arbitrary number of sub-populations. Solving for the expected number of susceptibles and expected duration was computationally hard, although simulating the model was not. Using MATLAB we simulated four sub-populations laid out in a line. An interesting example for further study would be a spreading the sub-populations out on a grid, especially since it should then be easier to use the spatial models discussed in Chapter 5.

Suggestions for further study include finding out whether in a multiple-sub-population Reed-Frost model each node can be viewed as independent. If that were true then we could create a corresponding mean-field ODE. Then using the techniques suggested in Chapter 5 we could then find values of \( \gamma \) and \( c \) that lead to complete infection in the long run.
Appendix A

Reed-Frost Equations

Single sub-population using Reed-Frost infection:

This model has two populations: a general population and a sub-population. The sub-population has size $N$, whereas the general population is assumed to be infinite. The time step is denoted by $t$. Those of the sub-population who are infected, $Y_t$ in number, are replaced by a random selection of people from the general population. The infected population in this new group is denoted $Y'_t$. The number of susceptibles is now denoted $X'_t$, which is the total of the incoming susceptibles and the original susceptibles, $X_t$. These two groups now intermix such that the susceptibles have a chance of getting infected and thus being infectives at time $t + 1$.

General population incidence rate: $p$ (and we denote $1 - p$ by $q$)

The non-infection probability is $\gamma$

$N$ is size of sub-population

$X_t$ is the number of susceptibles in population at time $t$

$Y_t$ is the number of infectives in population at time $t$

$N = X_t + Y_t$

$Y'_t$ is the number of incoming infectives

$Y'_t = \text{bin}(Y_t, p)$
\[ X'_t = N - Y'_t \]

Then after mixing, the number of new susceptibles for the next time period number
\[ X_{t+1} = \text{bin}(X'_t, \gamma Y'_t) \]

We can set this model up as a Markov chain that tracks the probability distribution of the number of susceptibles. Accordingly, \( P(X'_t|X_t) \) can be written as matrix \( R1 \):

\[
R1 = \begin{pmatrix}
  p^N & \binom{N}{p}p^{N-1}q & \cdots & \binom{N}{1}pq^{N-1} & q^N \\
  \vdots & \vdots & \ddots & \vdots & \vdots \\
  \vdots & \vdots & \ddots & p & q \\
  \vdots & \vdots & \ddots & 1 \\
\end{pmatrix}
\]

Then \( P(X_{t+1}|X'_t) \) can be written as matrix \( R2 \):

\[
R2 = \begin{pmatrix}
  1 & \cdots & \cdots & \cdots & \cdots \\
  (1 - \gamma N^{-1}) & \gamma N^{-1} & \cdots & \cdots & \cdots \\
  \vdots & \vdots & \ddots & \vdots & \vdots \\
  (1 - \gamma)^{N^{-1}} & \binom{N^{-1}}{1} & (1 - \gamma)^{N-2} \gamma & \cdots & \gamma^{N-1} \\
  0 & 0 & \cdots & 0 & 1 \\
\end{pmatrix}
\]

Thus, the entire transition matrix of the Markov chain is \( P = R1 \times R2 \).
Bibliography


