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Problem Chosen

A

2015 Mathematical Contest in Modeling (MCM) Summary Sheet

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Abstract

The mathematical modeling of the spread of disease can be a useful tool in predicting the outcomes of viral epidemics and the efficacy of control methods to combat them. In this paper, we develop a new mathematical model called the SEITHD model, which has similarities to more rudimentary SIR models for detailing the infectious spread of the Ebola Virus in West Africa and how it can be eradicated with a newly developed medication. We consider the factors of total population, number of initially infected individuals, the mathematically determined transmissibility of the virus, and the number of treatment facilities available to administer both a treatment for those infected and a vaccination to prevent the spread of the virus to otherwise healthy individuals. We discuss the efficacy of the vaccination and treatment as function of population factors, and the progressing rate of treatment and immunization as time goes on. We use the concept of herd immunity to develop a strategy to vaccinate a majority of the susceptible population in West Africa in a rapid manner. Once our model has been outlined, we discuss how we can optimize the manufacture, distribution and administration of the treatment and vaccine to eradicate the Ebola virus in the fastest and cheapest manner possible. The result of our model testing showed that we could eradicate the disease in an optimal manner given the current conditions in West Africa and even certain worst-case scenarios. We analyzed the scope in which our model can operate and the threshold at which it begins to lose efficacy and then fail altogether. Finally, we discuss the advantages of using our model and what important information can be gained from our eradication strategy.

Optimizing the Eradication of Ebola Using The SEITHD Model

Team #41161

February 9, 2015

Abstract

The mathematical modeling of the spread of disease can be a useful tool in predicting the outcomes of viral epidemics and the efficacy of control methods to combat them. In this paper, we develop a new mathematical model called the SEITHD model, which has similarities to more rudimentary SIR models for detailing the infectious spread of the Ebola Virus in West Africa and how it can be eradicated with a newly developed medication. We consider the factors of total population, number of initially infected individuals, the mathematically determined transmissibility of the virus, and the number of treatment facilities available to administer both a treatment for those infected and a vaccination to prevent the spread of the virus to otherwise healthy individuals. We discuss the efficacy of the vaccination and treatment as function of population factors, and the progressing rate of treatment and immunization as time goes on. We use the concept of herd immunity to develop a strategy to vaccinate a majority of the susceptible population in West Africa in a rapid manner. Once our model has been outlined, we discuss how we can optimize the manufacture, distribution and administration of the treatment and vaccine to eradicate the Ebola virus in the fastest and cheapest manner possible. The result of our model testing showed that we could eradicate the disease in an optimal manner given the current conditions in West Africa and even certain worst-case scenarios. We analyzed the scope in which our model can operate and the threshold at which it begins to lose efficacy and then fail altogether. Finally, we discuss the advantages of using our model and what important information can be gained from our eradication strategy.

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1 Introduction

In this paper we present a mathematical model for studying the administration of an hypothetical medication that can stop the spread of the Ebola virus. Our model takes the dynamic spread of the Ebola virus as a significant determiner of the efficacy of this medication. Specifically, we utilized various mathematical systems of predicting the spread of the Ebola virus given its current virulence to provide a plan of administration of the drug. Our models give public health organizations the best solution of how to conduct an eradication plan.

1.1 Current Status of The Ebola Virus

The West African Ebola outbreak of 2014 was the largest in history, affecting nine countries and resulting in more than 22,000 confirmed cases. The recent epidemic has prompted the need for rapid development of containment methods which include quarantine, education and the application of experimental treatments. The spread of Ebola in West Africa presents treatment issues due to the inaccessibility of remote African villages, the virulence of the virus and lack of available health-care.

1.2 Problem Statement

The task of this group is to build a model to plan the eradication of Ebola using a new, hypothetical drug developed by the World Medical Association. This model is meant to optimize for factors including the spread of the disease, the quantity of drug needed, the potential delivery systems and the speed of manufacturing the vaccine. The solution must both eradicate Ebola and cure current Ebola patients whose disease is not advanced.

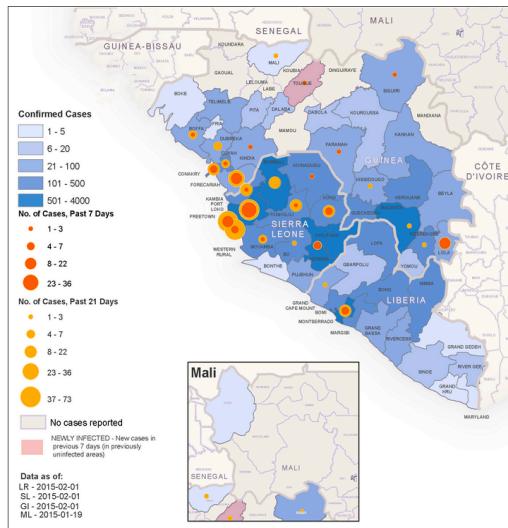


Figure 1: Map of West African Ebola Outbreaks February 2015¹⁴

1.3 Definitions

- **Eradication:** The Center for Disease Control defines eradication as "*control of an infection to the point at which transmission ceased within a specified area.*"⁴
- **Neutralizing Antibody:** Overbaugh and Morris identify neutralizing antibodies as "*antibodies [that] play a key role in controlling viral infections and contribute to the protective effect of many successful vaccines.*"¹⁵
- **Herd Immunity:** The U.S. Department of Health and Human Services defines herd immunity as "*when a critical portion of a community is immunized against a contagious disease, most members of the community are protected against that disease because there is little opportunity for an outbreak.*" The efficacy of modern vaccination programs hinge on achieving herd immunity, which generally requires a large majority of a population.¹²

1.4 Assumptions

Due to the high degree of variability of predicting the spread and behavior of a virulent virus, particularly one as unfamiliar as Ebola, our group made the following assumptions when constructing our model:

- We assume that individuals with the virus are only contagious when they display symptoms. During the incubation period of the disease, where the patient does not display symptoms, the individual is not contagious.
- We did not design our model to account for communicability from deceased individuals, non-human virus reservoirs or non-symptomatic individuals. Our model only accounts for the spread of the disease through human to human interaction during the symptomatic portion of infection.
- The initial conditions for our models come from the most recent data to this group. This means our model has a strong degree of topicality to the current status of the Ebola virus, but is limited in that its conclusions only apply to the current state of affairs.

2 Eradication Strategy

By the definitions set forth, this group has established two end goal criteria in order to qualitatively determine that Ebola has indeed been eradicated:

1. **All existing infected Ebola patients have been treated.**
2. **Herd immunity for the given population has been reached.**

In order to create a comprehensive treatment plan that achieves the above criteria, the following will be accomplished:

- Outline the drug that will be used, including its properties, manufacture, quality, administration method, cost, and efficacy, in order to minimize manufacturing time and amount manufactured.

- Determine the ideal method and plan of delivery to the affected areas in order to maximize treatment speed and minimize cost and time. The plans for remote and densely populated areas will differ slightly in approach.
- Model the transmission of the disease and the dissemination of our treatment and vaccine to determine the efficacy of the treatment and determine how to minimize the spread of the virus.

3 Virus Treatment

In order to eradicate the Ebola virus, this group will use a combination of treatment of infected patients and vaccination of the remaining susceptible population. Our group will be utilizing a mixture of neutralizing antibodies and attenuated live cultures of the Ebola virus as the mechanism to destroy the Ebola virus within host patients and ultimately eradicate the disease. This medicine will allow us to vaccinate healthy individuals with the same treatment that is used to destroy the Ebola virus in infected individuals. These neutralizing antibodies will eliminate the virulence of the live cultures and serve to safely inoculate healthy human hosts.¹⁶

3.1 Infection Cycle

The Ebola virus has a four step life cycle inside of its host. After exposure, there is an incubation period where no symptoms are expressed which lasts between 2-21 days with an average of 6 to 10 days.⁶ In the next stage, the host begins to show flu-like symptoms such as fever, chills, and fatigue. It is very difficult to diagnose someone with Ebola during this stage due to these symptoms being common with many other infectious diseases. It is during this initial period of symptoms that treatment will most commonly be applied to patients. This period will last between 1 and 34 days, with a mode of five days. After this period of common symptoms, hosts enter the advanced symptoms stage of the infection which is characterized by vomiting, diarrhea, and bleeding from orifices. Once this stage has been entered there is a low chance of survival. The treatment is no longer effective at this stage, making it imperative that treatment is applied prior to reaching the advanced stages of the infection.

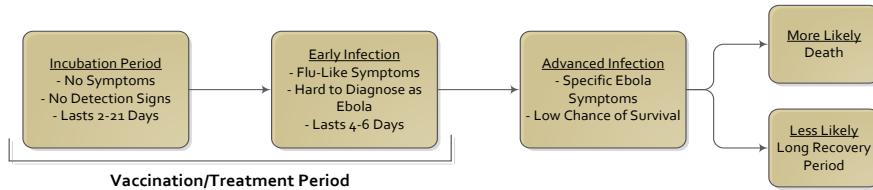


Figure 2: *Ebola Infection Cycle*

3.2 Treatment

Treatment of individuals will occur at available *Ebola Treatment Centers (ETCs)*, centers that are evaluated by the World Health Organization to be equipped to treat Ebola patients. Our ability to treat patients is limited to the number of available beds in these ETCs. There are three steps in the treatment of individuals: quarantine of patients at ETCs, treatment of individuals for twelve days, and release of recovered individuals. In areas without ETCs, our model will not consider the number of people treated for Ebola, only the number vaccinated.

Since the medicine developed by the World Medical Association is only able to cure patients whose disease is not advanced, treatment and administration of the drug needs to occur prior to the third infection stage. However, we are unable to see symptoms of Ebola until we reach the second stage of the cycle. This leaves us with a small window of time (avg. 5 days) to treat patients before the virus enters the untreatable stage. Individuals that exhibit flu-like symptoms in high risk areas for Ebola outbreak must be treated immediately, if possible, in order to minimize death and spread of infection to the susceptible population.

3.3 Immunization

While treatment of the infected population is prioritized, it is also important to vaccinate the susceptible and exposed populations in order to achieve herd immunity. Vaccinating a percentage of susceptible populations over time will ensure that the entire population approaches an "infection free" state. The percentage of the population we need to vaccinate depends upon the *basic reproduction number* (R_0), which is the expected number of secondary cases that are produced from a single case in a completely susceptible population.⁸ Luckily, Ebola has a relatively low reproduction number compared to other common viruses so the vaccination rate does not need to be extremely fast and makes the "infection free" state realistically within reach for developing countries.

3.4 Efficacy

There are two efficacies to consider, that of the vaccination and that of the treatment. The Ebola treatment is only effective in the first symptomatic stage of the infection. The success of the treatment diminishes as time goes on, and as the victim nears the second, advanced symptomatic stage of the infection. The efficacy of the treatment during this period is modeled after the efficacy of the live attenuated vaccine for smallpox, which can be used to treat smallpox within a seven day period following viral infection (Table 1). The utility of the post exposure treatment drops significantly after three days of infection.

The efficacy of the vaccine is difficult to judge, as pre-exposure vaccination rates generally tend to vary based on the type of vaccination, patient immunity considerations such as age and health and present herd immunity rates. Most vaccinations are able to reduce cases of disease by 95% or more in subsequent years following their widespread implementation.¹¹ For this reason, the vaccination efficacy for the Ebola inoculation was chosen to be 100%. This rate is considered appropriate considering the lack of true Ebola vaccination data, and the trend of high rates of effectiveness of most vaccinations.

Table 1: Chances To Prevent Smallpox by Time After First Symptoms¹¹

Time After First Symptoms (days)	Natural Immune Effectiveness
1	0.350
2	0.350
3	0.359
4	0.389
5	0.438
6	0.519
7	0.583
8	0.667
9	0.778
10	0.875
11	0.933
12	0.933
13	1

3.5 Administration Method

Live attenuated vaccinations may be administered intranasally, orally, or through injection. There are three primary considerations when deciding on an administration method: cost of the medicine, storage of the medicine, and safety.

Given these criteria, we classify 10-dose intradermal injection vaccination as the optimal method of administration of the vaccination, and single-dose intradermal injection as the optimal method of administration of the treatment. Multi-dose injections are already a commonly-utilized method of vaccination, and can be applied to a wide range of patient ages. In 2000, approximately 80% of vaccinations administered globally were supplied in multi-dose vials. Multi-dose vaccinations describe common glass vials that are available in varying sizes from two to up to fifty doses. Most childhood immunizations come in 10 or 20-dose vials, and can be applied to 10 or 20 individuals respectively.⁵ Our group assumes that one dose of the drug is sufficient to cure and/or vaccinate a patient. The high degree of vaccination demand warrants the use of high-dose injection vials.

Due to the higher volume of manufacture, multi-dose injection viles are cheaper than intranasal, oral vaccines, and single-dose injections and can be used for a wider age group than intranasal and oral vaccinations.

For the treatment of infected patients, single-dose intradermal injection is preferred for two reasons. The first is that viral contamination is less likely to occur when a single needle is used for a single victim of infection. The second is that the number of infected victims cannot always be accurately predicted on a day-to-day basis. As such, using single-dose vials for infected patients will reduce vaccine wastage compared to multi-dose vials.

The storage of the treatment is an important consideration as most vaccinations require refrigeration. The 10-dose vials will require much less space than the single-dose vials, and will be easier to transport. Additionally, multi-dose vaccines

that have been stored properly can be used past their labeled expiration dates, making them more ideal candidates for long-term immunization.⁵

3.6 Composition and Safety

The medicine being used by our group is idealized to perform two tasks: treatment of the disease in infected populations and vaccination of the healthy population. It is partially composed of attenuated live cultures of the Ebola strain to act as the vaccine, which brings the possibility of post-vaccination side effects and development of the disease in the healthy host. In order to prevent this, the concentration of neutralizing antibodies in the medicine is designed to be greater than that of the virus. This over saturation will cause all of the virus cultures to be neutralized to render them non-volatile and then have extra antibodies leftover to introduce to the host.

Involving the use of live Ebola cultures in the treatment of the virus seems counter-intuitive, but the combination with antibodies makes the medication safe. According to the ACIP, "Subsequent exposure to infection usually does not lead to viremia but to a rapid anamnestic antibody response," meaning the exposure to the attenuated cultures is not problematic, and produces the desired result of inoculation.²

4 Distributing The Medication

The Ebola virus outbreak is concentrated in West Africa, particularly in the nations of Liberia, Guinea, and Sierra Leone. While there have been Ebola infection cases in the nations of Mali, Nigeria, Senegal, Spain, The United States and the United Kingdom, there have been no new cases in these countries and the infection is effectively contained in these areas. In order to achieve the highest level of treatment and immunization of the susceptible population, our first model (SEITHD) considers more densely populated areas first to predict the spread of Ebola. We then use a second mathematical model (SEITD) to predict the spread in remote areas.

4.1 Densely Populated Areas

The Ebola treatment and vaccine will primarily be disseminated through 44 available Ebola Treatment Centers, seen in Figure 3. These are almost exclusively located in densely populated areas that can more easily be reached by conventional travel. It is assumed that the basic reproduction rate of the Ebola virus will be much faster within densely populated areas. It is also known that the most densely populated areas have the highest amount of currently infected patients. Concentrating on densely populated areas will minimize the amount of time it takes to reach herd immunity, and minimize the amount of time it takes to end the viral epidemic. Finally, reaching herd immunity will ensure that Ebola will have far more limited impacts in future outbreaks, if it is to occur again.

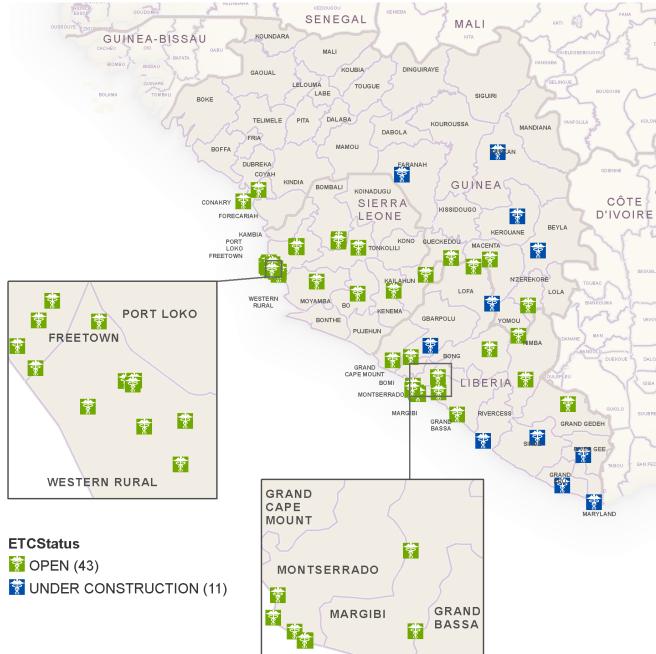


Figure 3: Ebola Treatment Facilities in Western Africa¹⁴

4.2 Remote Areas

Vaccination and treatment of remote areas of Liberia, Guinea, and Sierra Leone will utilize a different strategy as the spread of Ebola is slower and access to Ebola Treatment Centers is more limited. Areas within the interior of the continent will be reached with the limited available network of main roads, secondary roads, waterways and airports, seen in Figure 4.

We will consider Sierra Leone as an example to demonstrate this strategy. Currently, there are 24 operational Ebola treatment centers in Sierra-Leone. The bulk of these treatment centers are located in the densely populated Western Rural and Western Urban districts. The districts of Bonthe, Pujehun, Kambia, Kono, and Koinadugu which form 22.245% of Sierra-Leone's total population, lack access to treatment facilities.³ In the cases of Bonthe, Kono, and Koinadugu, we will send the medicine to Sherbro International Airport, Yengema Airport, and Kabala Airport respectively. The most ideal initial treatment site in the district of Kambia is the town of Kambia. Medicine can be sent to Kambia quickly on the Trans-West African Coastal Highway. The case of Pujehun is problematic since there are no initial points which can be reached quickly. Therefore we must consider distributing the vaccine throughout Pujehun from adjacent districts of Bo, Kenema, and Bonthe.

Our strategy will not consider the treatment of affected individuals in areas lacking ETCs, because we cannot accurately determine the number of individuals that can be removed from the pool of infected to be quarantined and treated.



Figure 4: Sierra-Leone Map of Infrastructure²¹

The number of people being treated per day in our SEIRHD model is based solely on the space available in the local ETC. For this reason, we cannot ensure the number of people being treated or the number of people quarantined on a given day. Thus we implement an alternative model (SEIRHD) that does not consider the number of people being treated at a given time.

5 Modeling Methodology

5.1 Modeling Treatment Efficacy

Because efficacy of the Ebola treatment is directly related to the time at which the treatment is applied following the initial symptoms, it is important to apply this factor in the spread and treatment model. The efficacy of the treatment, based on time after the initial symptoms and base immunity of the host is modeled by the following equation:

$$c_{tot} = c_1 + (1 - c_1) \cdot c_2 \quad (1)$$

Where c_{tot} is representative of the total chance of survival after the combination of c_1 , the chance of treatment success, and c_2 , the chance of a natural immune response taking out the virus. This efficacy value is then applied to the considered number of patients treated to determine the success of their treatment.

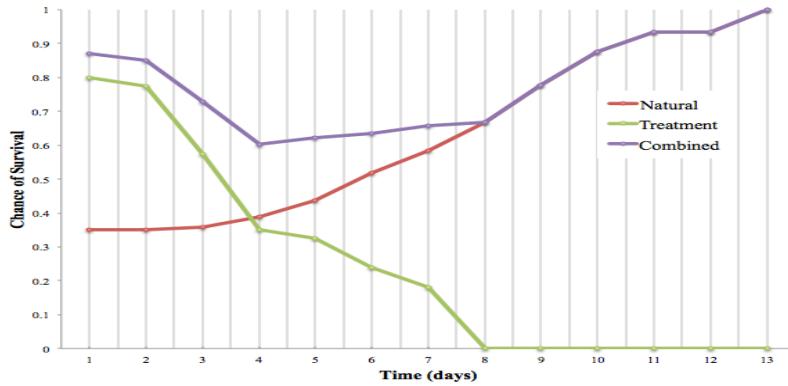
Figure 5: *Chance of Survival By Time After First Symptoms*

Table 2: Chances of Survival by Time After First Symptoms

Time After First Symptoms (days)	Natural Immune Effectiveness	Treatment Effectiveness	Combined Effectiveness
1	0.350	0.800	0.870
2	0.350	0.775	0.851
3	0.359	0.575	0.728
4	0.389	0.350	0.603
5	0.438	0.325	0.621
6	0.519	0.24	0.634
7	0.583	0.18	0.658
8	0.667	0	0.667
9	0.778	0	0.778
10	0.875	0	0.875
11	0.933	0	0.933
12	0.933	0	0.933
13	1	0	1

5.2 Modeling The Infection Cycle

The duration of the incubation period and the duration of the infection varies from host to host. However, data from victims of the Ebola virus can tell us what the distributions of these times are. It can be seen that the mode period of incubation is 5 days (Figure 6). The distribution of the number of days after first symptoms of the 1995 Ebola outbreak in the Democratic Republic of Congo are seen in Figure 5.¹⁷ These distributions were used in the SIETHD model to determine how quickly populations could move from incubating to symptomatic stages and how quickly death or recovery would occur following the onset of the symptomatic stage.

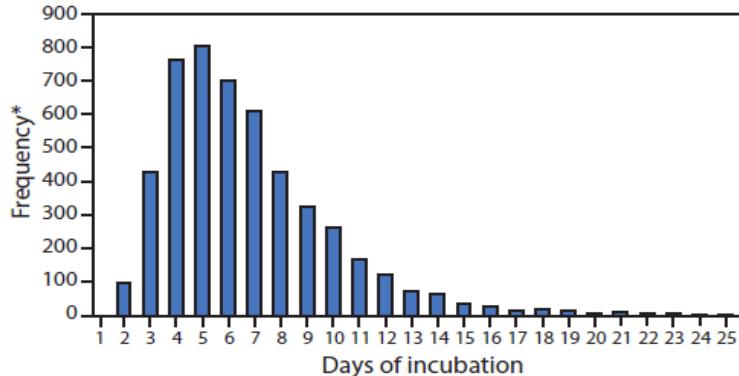
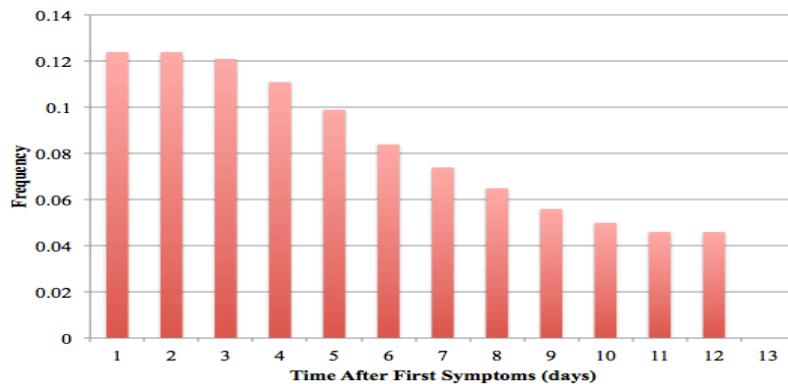
Figure 6: Distribution of Incubation Times Following Exposure¹⁰

Figure 7: Frequency of Patients In a Population By Days After First Symptoms

6 SIR Model

The first complete deterministic mathematical model for the spread of infectious disease developed was the SIR model.¹ An SIR model consists of three equations representing the rates of change of three sections of the target population: The Susceptible, the Infected, and the Recovered. The basic versions of these differential equations are modeled as follows:

$$\begin{cases} \frac{dS}{dt} = -\beta SI & (2.1) \\ \frac{dI}{dt} = \beta SI - \gamma I & (2.2) \\ \frac{dR}{dt} = \gamma I & (2.3) \end{cases}$$

Where S and I represent the populations of susceptible and infected individuals at a given time, respectively, β is the transmission rate of the disease, and γ is the average recovery rate from the disease. For example, if it usually takes 10 days to recover from the disease, then γ would be $1/10$.

If initial conditions for the sub-populations, β , and γ are given, then the differential equations can be used to model population changes over time. Solving these equations analytically can pose a challenge, thus numerical simulations are run to find the system behaviors.

The concept of modeling the changes over time is useful, but the SIR model is limited in its approach. The SIR model does not take into account any other stages the population could be in, so while it may be good at representing a system with only susceptible, infected, and removed, the SIR model cannot account for how factors outside of the given system effect spread over time. Also, the population rates of change only depend upon simple terms that are characteristic of all disease transmissions, but there are many other factors that play a role in movement between sub-populations. Vaccinations, births/deaths, and immune responses are some examples of such factors.

The SIR model has useful qualities and concepts involved in its modeling process, but overall, it is limited in its ability to model the transmission of Ebola.

7 SEITHD Model

In order to model the spread of Ebola and simulate the implementation of control methods, a more holistic mathematical model is necessary. The SEITHD model is capable of considering the following:

- variable incubation periods.
- variable symptomatic periods.
- variable efficacy of treatment.
- vaccination and treatment of separate section of the population.
- dynamic changes to the transmissibility of the virus depending on population characteristics.

During the incubation period, the virus is undetectable since the host shows no symptoms. This means that the population in the incubation stage cannot be treated as susceptible, but also are not in the same stage as the truly infected. In our assumptions, we stated that hosts are not contagious unless they are showing symptoms. Our eradication model also assumes that during quarantine, patients in ETCs being treated are unable to pass on their infection to the susceptible.

A section of the infected population is passing through treatment, while there is also a section of the healthy population that is getting vaccinated. These two combine to create a sub-population of total immune individuals. There is the chance of unsuccessful treatment or not receiving treatment in time, resulting in death. This leaves six sub-populations: Susceptible (S), Exposed (E), Infected (I), in Treatment (T), Immune (H), and Dead (D). We adapted the SIR model to show how the population moves through these sub-populations over time, and we call it the **SEITHD Model**:

$$\begin{cases} \frac{dS}{dt} = -\beta(t)SI/N - \alpha S & (3.1) \\ \frac{dE}{dt} = \beta(t)SI/N - \mu E - \alpha E & (3.2) \\ \frac{dI}{dt} = \mu E - \gamma I - \gamma(1-v)(I - \gamma I) & (3.3) \\ \frac{dT}{dt} = \gamma I - \gamma c T - \gamma(1-c)T & (3.4) \\ \frac{dH}{dt} = \gamma c T + \alpha S + \alpha E & (3.5) \\ \frac{dD}{dt} = \gamma(1-v)I + \gamma(1-c)T & (3.6) \end{cases}$$

The overall change in population is equal to zero. When one term leaves a stage, it is added to another, meaning there is no change in the total population. In the following sections, each rate of change will be broken down by term.

7.1 Assumptions

- This model assumes that the chain of transmission, i.e. the original source of a patient's illness, cannot be known. This is because our model cannot predict the ability for healthcare officials to track and terminate a chain of transmission. Ideally, the source of a victim's illness would be assessed upon their quarantine.
- We assume that if a patient receives successful Ebola treatment, then they are now immune and do not reenter the pool of susceptible individuals. While we do model certain aspects of general immune responses, we cannot holistically model aspects of an individual's immune response. The ACIP reports that, "Vaccines that stimulate both cell-mediated immunity and neutralizing antibodies (e.g., live-attenuated virus vaccines) usually can induce prolonged immunity..." which gives us good reason that this assumption is acceptable.²

7.2 Model Rates

7.2.1 Susceptible Rate

The rate of change of the population of susceptible individuals can be modeled with the following equation:

$$\frac{dS}{dt} = -\beta(t)SI/N - \alpha S \quad (3.1)$$

Where $\beta(t)$ is the transmission rate, S is the susceptible population, I is the infected population, N is the total population, and α is the vaccination rate. The rate of change of the susceptible population is always decreasing due to two negative terms. The first term accounts for susceptible humans becoming infected. $\beta(t)$ represents the probability of contact thus moving them to E . Dividing I by N allows the model to be dependent upon the percentage of infected individuals in the population and not just the raw number.

$\beta(t)$ can be determined from the following equation:

$$\beta(t) = R(t)\gamma \quad (4)$$

Where $R(t)$ is the reproduction number and γ is the average recovery rate. The average recovery rate was discussed earlier as being the reciprocal of the average number of days to recover, but for our model it will be the reciprocal of the treatment time (12 days). This is because it is the amount of time it takes to be deemed either recovered or dead or past the time period in which treatment is effective.

$R(t)$ can be determined with this equation:

$$R(t) = R_0 \left(1 + \frac{H}{N}\right)^{-1} \quad (5)$$

Where R_0 is the basic reproduction number of Ebola in the target area and the fraction in the parentheses represents the portion of the total population that is immunized. Since $R(t)$ is inversely related to this term, the reproduction number decreases as more people are vaccinated and treated.

The second term that affects the rate of change of the susceptible population is the vaccination rate. An important thing to know about this term is that α is equal to zero until the time the vaccination of the at-risk population begins. Our model is based on the idea that the distributors of the vaccination need a certain amount of time after hearing about the outbreak before they begin vaccinating the population. Once this time has passed, α is modeled by the equation:

$$\alpha = \frac{1}{365} \left(1 - \frac{1}{R_0}\right) \quad (6)$$

Where R_0 is the basic reproduction rate in the target area. The quantity inside the parentheses represents the *critical vaccination proportion*, the least amount of people that must be vaccinated in one year in order to reach herd immunity.¹⁸ Since our model is going to progress day by day, we divide this number by 365 to get the amount that must be vaccinated per day. Each day after vaccination begins, the amount α multiplied by the susceptible population moves to the immune population.

7.2.2 Exposed Rate

The rate of change of the exposed population can be modeled with the following equation:

$$\frac{dE}{dt} = \beta(t)SI/N - \mu E - \alpha E \quad (3.2)$$

Where μ is the average rate of movement from exposed to infected and E is the exposed population. The exposed rate has terms that both increase and decrease its population. The positive term has already been discussed earlier as the transmission rate of the virus, moving some of the susceptible population to the exposed population. The first negative term in the rate models how quickly the exposed population begins to enter the infected population. In Figure 6, the distribution of how long it took exposed individuals to show symptoms is graphed and this data is how μ is determined. Each day, a random number is picked

between 2 and 21 as the "average" number of days it takes to become infected. The chances of each number being selected are dependent upon their frequency in that graph. μ for that day is then the reciprocal of the random number. Since so many days are simulated in our model, this distributed selection process will reflect a real-life frequency.

The second negative rate of change is the vaccination rate of the exposed population. The α value is calculated the same as it is for the susceptible rate, which is Eq (6). Since Ebola has no signs of infection during the exposed stage, our group believes that the virus is in a stage of its life cycle that would create a negligible chance of an unsuccessful treatment if it were administered during this period, therefore α does not decrease.

7.2.3 Infected Rate

The rate of change of the immune population can be modeled with the following equation:

$$\frac{dI}{dt} = \mu E - \gamma I - \gamma(1-v)I \quad (3.3)$$

Where γ is the average infection recovery rate, I is the infected population, and v is chance of survival with no treatment. There is one term being added to the infected population while two terms are being removed. The positive term is the addition of exposed humans to the infected population. The first negative term is the rate at which we are able to move infected individuals into treatment. During the first few days before vaccination and treatment begin, this term is zero. Also, we neglect the second negative term until we begin treatment because the percentage of the infected population that dies during their first few days of being infected is almost negligible, which can be seen in Figure 7. On the first day of treatment in the area, we will move as many infected people into treatment as possible. Q is a fixed number for a treatment area and is the number of available spots in the ETCs. If I is greater than Q , then we can move only Q humans from I into treatment. If I is less than or equal to Q , then we can move all of I into treatment. So we can model the change of the infected population for the first day of treatment with the following:

$$\begin{cases} \frac{dI}{dt} = \mu E - Q, & I > Q \\ \frac{dI}{dt} = \mu E - I, & I \leq Q \end{cases} \quad (3.3a)$$

$$(3.3b)$$

This is **only** the case for the first day of treatment and vaccination. After this day, all three terms from Eq. (3.3) begin to affect the infected population. γ is still going to be equal to the reciprocal of the length of treatment (12 days). If all of the treatment sites Q are filled up, then we can't actually move γI infected individuals into treatment because that will make T , the population in treatment, be greater than Q . Since this isn't realistically possible, the change in the infected population needs to be limited by the relationship of Q and T .

After the first day of treatment, we can model the infected population by the following equations:

$$\begin{cases} \frac{dI}{dt} = \mu E - \gamma Q - \gamma(1-v)(I - \gamma Q), & T = Q \\ \frac{dI}{dt} = \mu E - \gamma T - (Q - T) - \gamma(1-v)(I - \gamma T - (Q - T)), & T < Q \end{cases} \quad (3.3c)$$

$$(3.3d)$$

In order to understand the relationship between Q and T correctly, we have to think about where the population leaving I is headed. They are either headed to treatment or they are going to die. We know that every day a certain number of the people in the treatment population T are either going to leave the treatment center healthy or die. This will be equal to γT , the average recovery rate times the treated population. So if the treatment centers are full on that day, when $T = Q$, then γQ leave the treatment center. Our model puts as many infected people as possible into treatment by subtracting γQ from the I population. The amount of infected people left is now equal to $I - \gamma Q$. v is representative of the chance of survival without treatment. v is similar to μ because it changes depending on how many days you have lived through the virus so far. We choose a random day value based on the distribution of people still alive on given days after becoming infected with Ebola (see Figure 7), and then we choose a value for v based on that random day. Since we perform this randomization every day of the model, it eventually has the correct distribution of v values. Our model uses this chance of survival to calculate the amount of infected people that die if they aren't going to receive treatment. To determine the untreated infected population that dies, the remaining infected population, $I - \gamma Q$, is multiplied by its chance of dying, $1 - v$, and γ .

In most cases, the treatment centers are able to treat the virus faster than it can spread, so T eventually becomes less than Q . If this happens, that means more infected individuals are able to enter the treatment centers. We know that γ of the population T leaves the treatment centers each day, so γT individuals are able to enter T from I . And if T is less than Q , then the difference between them is the number of extra spots open for treatment. So another $(Q - T)$ individuals can move from I to T . In total, we subtract $\gamma T + (Q - T)$ individuals from I and put them into treatment. After this movement, the number of infected deaths based on v is calculated using the leftover infected population, $(I - \gamma T - (Q - T))$.

The change in the infected population, probably the most complicated rate, has one more thing that needs to be considered. When the number of open spots in treatment is larger than the number of infected individuals left, our model would cause population I to become negative. Since it is impossible to have a negative population, we need to create a guardian at zero that prevents this from happening. If the change in population I ever makes it negative, we should check it with the following:

$$\frac{dI}{dt} = \mu E - I, \quad \text{if } I + \frac{dI}{dt} < 0 \quad (3.3e)$$

Simply put, this equation will send all of population I to treatment, and then create a new infected count from population E .

7.2.4 Treatment Rate

The rate of change of the population being treated can be modeled with the following equation:

$$\frac{dT}{dt} = \gamma I - \gamma c T - \gamma(1 - c)T \quad (3.4)$$

Where c is the survival rate with treatment. The value of c is similar to v , except that it is modified by the treatment received. The values for c came from Eq. (1), and can be seen on Figure [4] and Table 2. It is important to note that the change in T is equal to zero before we begin treatment and vaccination. The day we begin treatment, the change is modeled by the following:

$$\begin{cases} \frac{dT}{dt} = Q, & I > Q \\ \frac{dT}{dt} = I, & I \leq Q \end{cases} \quad (3.4a)$$

$$\begin{cases} \frac{dT}{dt} = Q, & I > Q \\ \frac{dT}{dt} = I, & I \leq Q \end{cases} \quad (3.4b)$$

This shows that if I is less than the number of open treatment spots then the whole infected population enters treatment, but an amount greater than Q can not enter treatment on the first day.

The change in population T is very similar to the change in population I . If you simplify Eq. (3.4), the c term disappears and γT is subtracted from γI . When T is equal to Q , then γQ patients leave the treatment centers that day. Therefore, only γQ people can enter treatment from I . The changes of T can be modeled by the following:

$$\begin{cases} \frac{dT}{dt} = \gamma Q - \gamma c Q - \gamma(1 - c)Q, & T = Q \end{cases} \quad (3.4c)$$

$$\begin{cases} \frac{dT}{dt} = \gamma T + (Q - T) - \gamma c T - \gamma(1 - c)T, & T < Q \end{cases} \quad (3.4d)$$

It is better to leave the $\gamma c Q$ and $\gamma(1 - c)Q$ terms separated in the equations above because they end up going to two different populations, Immune and Dead.

There is one more situation we need to consider: When I is about to become a negative number but we prevent it from crossing over zero with Eq. (3.3e). When we do this, it also modifies the rate at which T changes to the following equation:

$$\frac{dT}{dt} = I - \gamma c T - \gamma(1 - c)T, \quad \text{if } I + \frac{dI}{dt} < 0 \quad (3.4e)$$

7.2.5 Immune Rate

The rate of change of the immune population can be modeled with the following equation:

$$\frac{dH}{dt} = \gamma c T + \alpha S + \alpha E \quad (3.5)$$

Where the first term comes from the treatment population that became healthy, and the second and third term come from the vaccinations of the susceptible and exposed populations, respectively. The rate at which H changes is always greater than or equal to zero and is simple to understand where the values come from. First, H is not going to change during the first few days of our model because no treatment or vaccination occurs. On the first day, the rate will now be equal to the two vaccination rates, αS and αE , but it will not include the first term because there is nobody in treatment yet. After the first day, H will **always** increase at this rate.

7.2.6 Death Rate

The rate of change of the dead population can be modeled with the following equation:

$$\frac{dD}{dt} = \gamma(1 - v)I + \gamma(1 - c)T \quad (3.6)$$

Where the first and second terms come from the deaths associated with the infected and treatment population respectively. The death rate is also a very simple model because deaths in our model only come from two populations: Infected and Treatment. An assumption was made in the Infected section that stated no deaths occurred from Ebola during the first few days of our model. To reiterate why this was assumed, Figure 6 shows that the drop in living infected people during the first couple days of infection was very small compared to the total population, making these deaths almost negligible in the long run of our model.

There are three equations for deaths depending on the situation on a given day:

$$\begin{cases} \frac{dD}{dt} = \gamma(1 - c)Q + \gamma(1 - v)(I - \gamma Q), & T = Q \quad (3.6a) \\ \frac{dD}{dt} = \gamma(1 - c)T + \gamma(1 - v)(I - \gamma Q - (Q - T + \gamma T)), & T < Q \quad (3.6b) \\ \frac{dD}{dt} = \gamma(1 - c)T, \quad if \quad I + \frac{dI}{dt} < 0 \quad (3.6c) \end{cases}$$

7.3 Montserrado, Liberia

Using the model for Montserrado, Liberia produces the graph in Figure 8. The initial conditions associated with Montserrado are the following:

- Population: $1,010,970^{19}$
- Basic Reproduction Rate: 1.59^{20}
- Initial Infected Population: 131^{14}
- Treatment Spots: 150^{19}

7.3.1 Results

The outbreak occurring in Montserrado was taken care of with ease by our model, reducing the infected population to zero within 2 months. The model produced the following data:

- Only 57 people died from the Ebola virus, which is approximately 43.5% of the initially infected population.
- 223 people received treatment for Ebola during the first month of the outbreak, effectively eliminating the chance of it making a comeback in the area.
- one year after the start of the vaccination process, 310,000 people are in the immune population, which is about 30.7% of the total population. When $R_0 = 1.59$, 37.1% of the population needs to be vaccinated in order to reach herd immunity, so within the next couple months Montserrado will be able to avoid future outbreaks of Ebola.

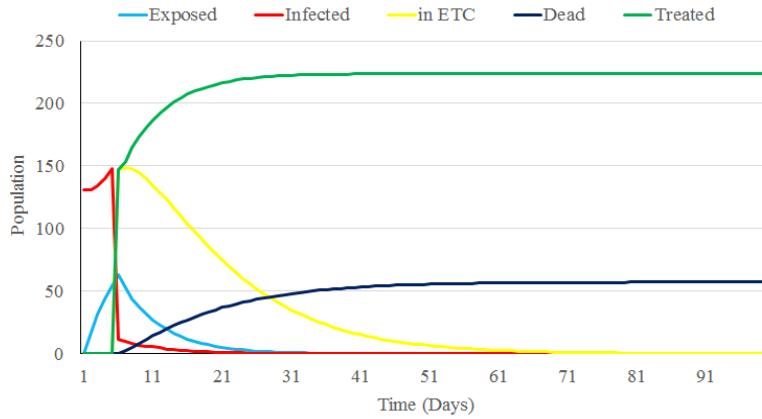


Figure 8: *Model of Spread and Treatment in Montserrat*

7.4 Freetown, Sierra Leone

Using the model on Freetown, Sierra Leone produces the graph in Figure 9. The initial conditions are as follows:

- Population: $1,200,000^{19}$
- Basic Reproduction Rate: 2.53^{20}
- Initial Infected Population: 220^{14}
- Treatment Spots: 248^{19}

7.4.1 Results

After applying our eradication model to the situation in Freetown, the entire infected population had progressed through treatment and out of all the ETC's within the first 3 months. Here are some other statistics associated with the treatment of this are:

- Our model predicted 163 total deaths during this outbreak, which is 74.1% of the initial infected population. This is acutally a pretty impressive result since the basic reproduction number was one of the higher reported numbers since the West African Ebola scare started and the initial infected population was 89% of the treatment spots available.
- At the beginning of the simulation of the model, the treatment centers went through a week period where the ETC's were packed full. Luckily, they were able to bounce back and treat enough people to recover from the increasing infected population before it was too late. This makes the dead percentage of the initial population even more impressive.

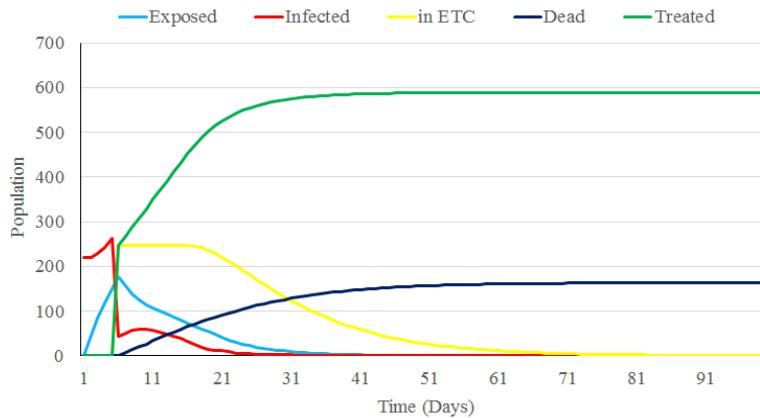


Figure 9: Model of Spread and Treatment in Freetown

- 539,690 people were immune by the year of the first year of simulation, while the population reached herd immunity a little bit before halfway through the next year of the simulation.

7.5 Conakry, Guinea

Our eradication of Ebola model produced the graph in Figure 10 after being applied to Conakry, Guinea. Here are the initial conditions we used for Conakry's Ebola outbreak:

- Population: 1,667,864¹⁹
- Basic Reproduction Number: 1.51²⁰
- Initial Infected Population: 153¹⁴
- Treatment Spots: 132¹⁹

7.5.1 Results

When our SEITHD model of treating Ebola outbreaks was implemented on Conakry, only 88 people died during the first few months of the simulation. Even though the basic reproduction number was one of the lowest ever reported for Ebola, it is still impressive that the area only lost 57.5% of the initially infected population since it began higher than the number of treatment spots. Here is some other data that came from the treatment simulation:

- Within one month, the infected population was completely gone and the people in treatment was dropping rapidly. Within the next month everyone was out of treatment.

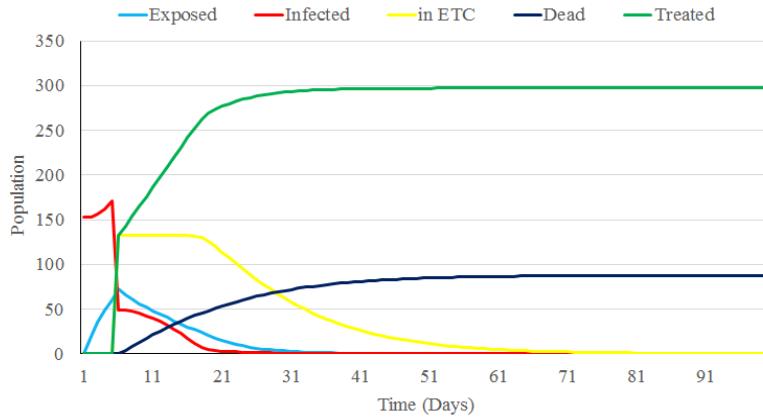


Figure 10: *Model of Spread and Treatment in Conakry*

- Less than 300 people needed treatment during this outbreak. This shows how much more important the basic reproduction rate is over other factors like the size of the population.

7.6 Lola, Guinea (No Treatment Centers)

The area of Lola, Guinea is not modeled by our generic eradication model. Since there are no treatment centers, we cannot quarantine the infected population and it makes it harder to isolate the virus. Instead of being able to take the infected population out of the transmission pool and treat them, we are unable to directly treat the infected, leaving them to expose more of the susceptible population. This creates more transmission of the virus and therefore more deaths. In order to simulate the spread of Ebola in Lola, a modified version of our SEITHD model without the T population, called a SEIHD model, is used. After running a SEIHD model with the following initial conditions, we produce a graph of some of the populations in Figure 11:

- Population: 124,000¹⁹
- Basic Reproduction Number: 1.51²⁰
- Initial Infected Population: 15¹⁴
- Treatment Spots: 0¹⁹

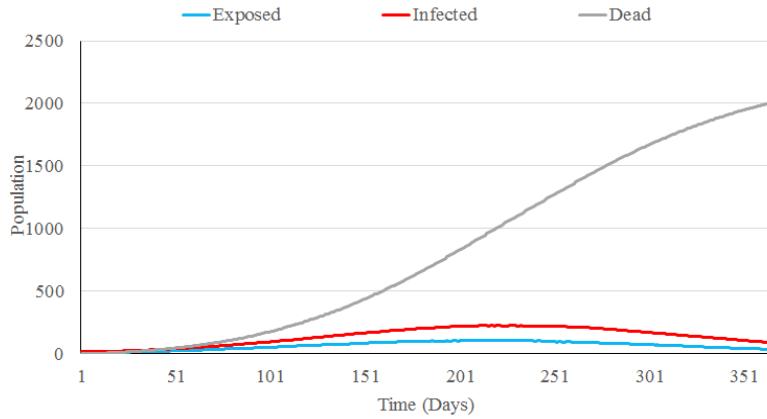


Figure 11: Model of Ebola Spread in Conakry without proper treatment

7.6.1 Results

The results of this outbreak are very troubling for the population of Lola, but it is easy to see why the increase in deaths occur. If the I population is never able to get close enough to zero to stop the spread of the virus, then there will continue to be exposed and infected individuals. The deaths continue to rise for a year, but then begin to level off after the susceptible population becomes the smaller population that limits the transfer of the virus. The problem is that once it finally leveled off, the death toll was above 2,000 people, roughly 2% of the area's population, even though they were able to immunize their population at a speedy rate.

7.7 R_0 Value Results

When looking at the initial conditions that used to set up our eradication model, R_0 is very important because it effects the efficacy of our model, and the degree to which our strategy can be called successful. First, it is the basis of how the rate of vaccination is chosen. If a vaccination rate is not chosen depending on R_0 , then the rate may not be enough to combat the transmission of the disease and the susceptible population may move faster toward the exposed population rather than the immune population.

The transmission rate, β , is also directly dependent upon the basic reproduction number. Since transmission is the replication of the disease, this means a lot of information farther down the road will be dependent upon this R_0 value, such as death rate. Our model shows how the effects of an increasing reproduction number causes many factors to increase, the worst being the total number of deaths. Figure 12 shows the relationship between the total deaths and reproduction number with everything else held constant. The jump between R_0 lines is an average of .3, and the spacing is only increasing as the magnitudes increase.

Soon the total deaths will be uncontrollably high if R_0 increase enough. With this being said, R_0 is one of, in not the most, important initial condition we consider.

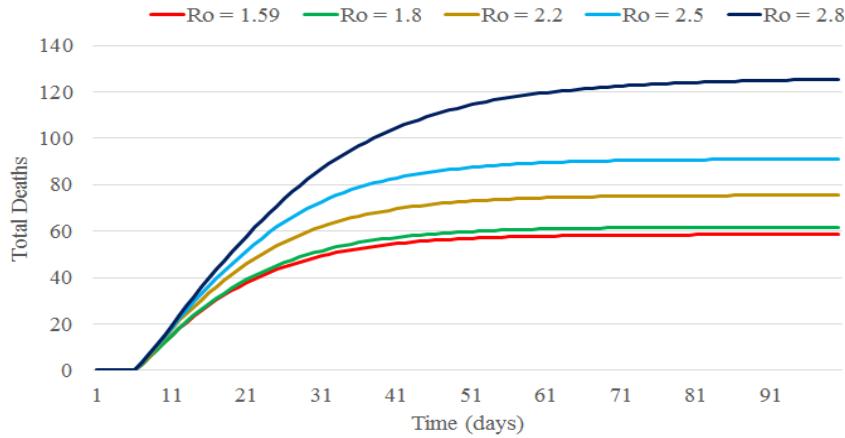


Figure 12: Relationship between Basic Reproduction Number(R_0) and Total Deaths modeled for Montserrado, Liberia

8 Cost Analysis

Reducing the costs related to administering our medicine to the areas effected by Ebola is critical to its rapid eradication. The inaccessability of remote West African villages, the virulence of the virus and the lack of available healthcare makes combating the disease problematic and raises the cost of its treatment.

We can calculate the total cost for a given country by the following equation:

$$C_T = C_M + C_D + C_A$$

where C_M is the manufacturing cost, C_D is the distribution cost, and C_A is the administrative cost. Countries that require a larger amount of treatment and vaccine and have poorer transportation infrastructure will ultimately cost more to control their Ebola epidemics.

8.1 Manufacture

The manufacturing cost of multi-dose vials is far cheaper than their single-dose counterparts and even oral and intranasal administration methods. This is due in part because filling costs and packaging costs at the time of manufacture are lower. Overall, manufacturing costs for multi-dose formats are about 2.5 times lower than for single-dose. Wastage is also a critical factor to be considered when discussing

the cost of the vaccine. Despite the higher incidence of vaccine wastage in multi-dose vaccinations, the cost-benefits of using multi-dose vials are higher than those of single-dose vials.⁵ Additionally, because the number of vaccinations per day will be predicted prior to arrival, the multi-dose vaccination can be used to maximum effectiveness and number of required staff, which reduces wastage costs. The high degree of vaccination demand warrants the use of high-dose injection vials.

The manufacturing cost can be approximated by the following equation:

$$C_M = Tx + Vy$$

where T is the number of treated patients, V is the total number of vaccinated people, x is the manufacturing cost of a single-dose vial, and y is the manufacturing cost of a multi-dose vial. When examining this equation, we can see that given the total number of people we need to treat and vaccinate (derived from our model), to reduce manufacturing costs, we need to produce single and multi-dose vials that optimize the relationship between cost and wastage. Therefore, by choosing the quality of which our injections, we can pick a price of manufacture for which a vaccine wastage and cost are optimized. We also notice that since we only use multi-dose vials for people who are to be vaccinated, our manufacturing costs are significantly reduced.⁵

8.2 Distribution

The distribution of our vaccine will be initialized at Ebola Treatment centers (ETCs), seen in Figure 3 . From these initial points, we will use the main and secondary roads, and waterways to transport the vaccine. There are a total of 44 total ETCs throughout Western Africa.¹⁴ In addition to limited facilities, the fact that many areas in this region lack proper infrastructure hinders our ability to quickly reach remote areas if an outbreak occurs. Transportation to remote areas of Liberia, Sierra Leone and Guinea will be more costly as it will require transportation of the medicine and personnel using poorer roads and access points. Additionally, it will cost more time to reach these areas, which will give the Ebola virus more time to spread before we can effectively contain it. Finally, the cost of storage will increase as the amount of time of transportation increases. The cost of distribution, C_D , can be approximated by the following equation:

$$C_M = mz + Ta + Vb$$

where m is the total amount of miles traveled, z is the cost of travel per mile, a and b are the costs associated with storage of single and multi-dose vials respectively, and T and V are total number of treated and total number of vaccinated, over a certain time frame, respectively. Considering the mz term, we know that the z term is constant for the selected mode of transportation. The m term can be reduced by selecting an initial distribution site that will optimize for the amount of miles we must travel to spread the vaccine. The costs of storing our medicine is dependent on the volume of each dose. Since multi-dose storage costs are lower, we have reduced the cost of storage by applying the multi-dose medicine to the majority of the population.⁵

8.3 Administration

Because intradermal vaccination requires administration by a medical professional, the staffing of the Ebola Treatment Centers will necessitate precise calculation for the treatment plan.

The administrative costs, C_A , are calculated by the following equation:

$$C_A = Tn + Vk$$

where n is the cost associated with the wages of the staff that treats patients, k is the cost associated with the wages of the staff that vaccinates the population, and T and V are the total number of treated people and vaccinated people over a predicted time frame. To reduce the first term on the right side of the equation, we can designate each healthcare official a number of patients to treat and care for to optimize the cost vs. treatment rate. Also, if our treatments are successful and if there is an adequate rate of treatment, the total number of staff designated for treatment will decrease. So increasing rate of treatment will reduce net administration costs associated with treatment. Although we are maximizing the amount of people we vaccinate everyday, Vk stays nearly constant over our projected time because we continue vaccination of the population until a certain percentage of the population is vaccinated.

9 Conclusions

The consideration of various aggravating and random factors within our SIETHD model allows us to more accurately predict the spread of a disease within a population. Given our eradication method of relying on ETCs for treatment and reaching herd immunity to end the epidemic and threat of future outbreaks, there is a clear threshold at which our solution is successful. Once the basic reproduction number, and number of initially infected victims is elevated to an insurmountable degree, the hope of eliminating the Ebola outbreak becomes untenable. Our model can correctly simulate this threshold of efficacy based on the factors of initial population, initial number of infected individuals, transmissibility of the virus, and number of treatment sites available. Our model optimizes the feasible factors which can be controlled, and operates very successfully under all but the most realistically catastrophic circumstances. Additionally, our model sets guidelines by which we can optimize the time, cost and method of eradicating the disease. Given what we have observed, eradication strategy can be implemented in the current state of affairs with success.

10 Model Analysis

10.1 Model Strengths

There are a number of areas in which our model excels and allows us to simulate a realistic outbreak scenario. Many important factors in the spread of the Ebola virus have been considered, and allow us to draw strong conclusions about the nature of the virus and the course of action which should be taken to combat it.

10.1.1 Realistic Population Response

Our model takes into account several factors that account for the realistic spread of the Ebola virus, and its reaction to our eradication strategy. Our model considers real distributions of incubation and symptomatic times in exposed patients and can very accurately model the death rate of the virus given its virulence. Our model holds a high-level of realism in its regard to its dynamic variability of the transmission rate, which varies with the immune population to simulate the effects of herd immunity.

10.1.2 Available Information

Our model is based off of four initial conditions; population, basic reproduction number, number of available beds at local treatment centers, and number of initially infected victims. These factors are mostly well-known and can be verified quickly through multiple sources. This makes our model and its conclusions very accurate.

The basic reproduction number and the number of initially infected were the least accurate variables used, but values obtained from multiple sources allowed us to test a wide range of outbreak scenarios, and ultimately test the efficacy of our treatment plan.

In order to test our success rate against our two varying initial conditions, we set the total population and number of treatment spots to generic numbers based on the values most frequently recorded. We tested basic reproduction numbers from 1.5 to 4 with a step size of .1 and initial infected population values from 10 to 500 with a step size of 10. Our model output a table of our success rates at each variation of (R_0, I_{init}) which provided a good surface plot of the data seen in Figure 13.

10.2 Model Limitations

Our model attempts to achieve a high degree of realistic accuracy when simulating the outcomes of the current Ebola outbreak and our efforts to contain it. Even so, there are a number of areas in which the scope of our model is limited. We can draw important conclusions from the data we have acquired, but the following are factors for which our model does not correctly account and for which we cannot provide accurate solutions.

10.2.1 Herd Immunity In Our Model

Our model takes a strong cue from the idea that herd immunity is the ultimate goal of a vaccination program and that achieving herd immunity will defeat the spreading potential of a disease. Our model attempts to discern when this occurs after the vaccination program and how this affects the transmissibility (R_0 and β) of the disease. Unfortunately, our model does not account for the exponential nature of transmissibility. When isolating R_0 , many researchers have found that once herd immunity has been reached, and the disease has transformed from epidemic to endemic, there is a logarithmic decline in the R-value of the disease.

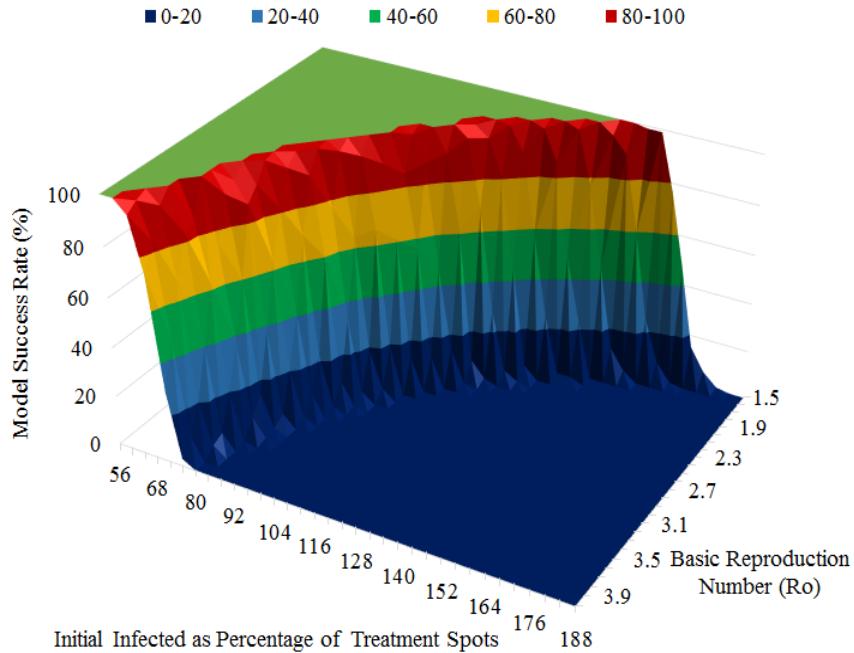


Figure 13: *Model Success Rate*

While our model predicts the decline of R_0 and β with implementation of disease control measures, it does not model it in this logarithmic way.

10.2.2 Population Density

Likely the most significant factor that was excluded from our model was the direct correlation between population density and transmissibility. It follows that the degree to which a virus is infection is directly related to the number of individuals in the direct vicinity of the outbreak, and larger population densities increasing the likelihood of interactions between individuals. In this same vein, our model can only predict the interactions of a homogeneous population. Our model considers population, but not the area to which they are confined, which limits our model's accuracy.

10.2.3 Contact Networks

The most advanced models of disease spread in epidemiology consider transmissibility by probabilistic contact interactions that build infection networks. In particular, these models consider each individuals distinct role in a larger population. These models weigh herd immunity greatly as a factor in reducing the spread of disease, because the implementation of vaccination helps reduce the probability of long contact networks forming, much as would occur in real life. If this model

were to aim to be as realistic as possible, and perhaps map the spread of disease over time, it would likely have implemented this concept of contact networks expanding from an epicenter.

10.3 Improving The Model

Recognizing the limitations of our model, we are able to consider how certain improvements would provide better simulations. For example, if our model accounted for how the change in transmission rates correlate to population density and probabilistic interactions, nonhuman viral reservoirs, and improper burial practices, it would have a higher degree of real-world accuracy. Since most areas have nonhomogeneous population distributions, this improved model would be able to simulate the spread in both cities and remote areas much more accurately. In addition, a model that could account for more realistic contact networks would be able to give valuable information on how to expedite the eradication process. These improvements would allow for a higher degree of optimization and accuracy in an eradication strategy.

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- ²¹ UN. Cartographic section. Availableat:<http://www.un.org/Depts/Cartographic/english/htmain.htm>, 2007.

Ebola can now be eradicated, the World must take note

Geneva, Switzerland February 9 2015: The World Medical Association strongly urges governments and humanitarian actors to commit to an ambitious comprehensive plan to eradicate the threat of Ebola in the world. The recent creation of a new medication and vaccination to combat the disease brings the world a chance to end the epidemic while it is still in its early stages.

A resolution adopted by the Association, delegates from a dozen national medical associations called for more effort to control the deadly virus and apply the new medication to eradicate the disease. The uncontrolled spread of infectious disease like Ebola is a public health concern and a global crisis.

The Assembly called on the international community acting through the United Nations, its aid agencies and other global humanitarian groups to immediately provide the necessary supplies and equipment to protect affected communities and health care workers worldwide. This must include the mass manufacture, transport and means to administer treatment to affected communities worldwide and engage in nation-wide vaccination efforts.

The resolution declared that there is a shortage of resources, including health care workers, personal protective equipment and beds. Countries must commit resources as soon as possible, as treatment and control methods are most effective in the earliest stages of the viral epidemic spread.

The Association calls on national and local government to raise awareness about the disease and its new medication and vaccine as well as control practices which will help combat and eradicate the epidemic

The resolution commends those countries that commit resources to the urgent establishment of new treatment and isolation centers for the most heavily burdened regions of the world facing this threat.

The global community has a unique opportunity to end an emergency situation and avoid further humanitarian suffering and reduce the overwhelming economic cost facing the impoverished communities at risk. With global help, the eradication of Ebola in West Africa and the world can be one of the great accomplishments of this century.



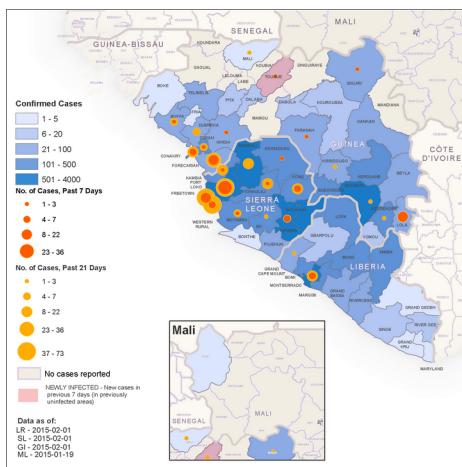
WMA

The World Medical Association

Ebola Eradication Strategy

At a glance

Affected Areas



Courtesy of the World Health Organization

Liberia	
Total Population	4,092,310
Total Requiring Treatment	≈400
Requiring Vaccination	≈2,500,000
Time Until Vaccination Goal	≈535 days
Number of Treatment Centers	18
Sierra Leone	
Total Population	6,190,280
Total Requiring Treatment	≈680
Requiring Vaccination	≈3,705,000
Time Until Vaccination Goal	≈535
Number of Treatment Centers	4
Guinea	
Total Population	10,057,975
Total Requiring Treatment	≈650
Requiring Vaccination	≈6,020,000
Time Until Vaccination Goal	≈534 days
Number of Treatment Centers	24

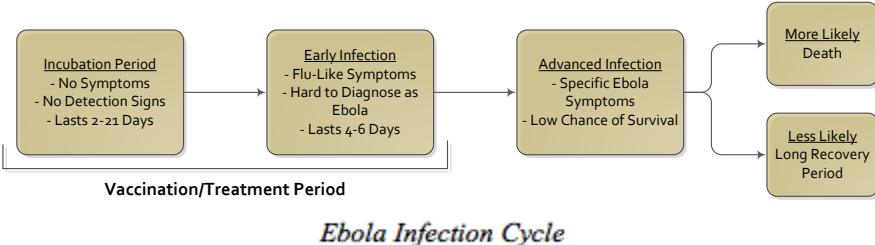
With a definitive cure for Ebola finally available, here are some fast facts that everyone should be aware of

Strategic Goals

The outbreak potential of the Ebola Virus CAN be eliminated within a year through the eradication plan.

Quarantine

- Quarantine All Infected Persons*
- The infected should be quarantined at certified Ebola Treatment Centers if possible
- Minimum treatment/quarantine time is 12 days



Treatment

- Treatment is only effective during first 7 days of symptoms, early diagnosis is key
- One patient = one injection

Vaccination

- Vaccination is the only way to stop the spread of Ebola and prevent future outbreaks*
- Immunize any and all healthy individuals
- ≈60% of population must be immunized, higher if you live in the city
- 10-dose injections should all be administered to reduce waste