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A Mathematical Investigation of Vaccination Strategies to Prevent a Measles Epidemic

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ABSTRACT. The purpose of this project is to quantitatively investigate vaccination strategies to prevent measles epidemics. A disease model which incorporates susceptible, vaccinated, infected, and recovered populations (SVIR) is used to investigate the process of how an epidemic of measles can spread within a closed population where a portion of the population has been vaccinated. The model is used to predict the number of infections and resulting reproductive number for the measles based on a variety of initial vaccination levels. The model is further used to investigate the concept of herd immunity, which states that if a certain percentage of the population is vaccinated then it will provide protection for the entire population. Results generated from these modeling efforts suggest that approximately 95% of the population should be vaccinated against the measles in order to establish a herd immunity.

1. Introduction

The measles is a highly contagious acute viral airborne disease that can result in serious complications and even death. The disease is believed to have been first discovered and reported by Rhazes, a Persian alchemist, philosopher, musician and mathematician in the year 910 (Cohen, 2008). Seven centuries later, a British physician named Thomas Sydenham further described the symptoms, course, and complications of the disease. Following these early studies that identified the measles, a vaccine has been discovered and adopted. There have also been numerous other studies on the transmission, severity, and possible elimination (through vaccination) of the measles. A few of these such studies are outlined below.

In 1967, Sencer et al. (1967) conducted a study for an epidemiologic basis for eradication of measles. The results of their 4 year study indicated that any community that could raise its immune thresholds to a readily attainable amount could effectively eradicate the disease. A study by Orenstein et al. (1985) led to the overall objective of field evaluation of vaccine efficacy. Their research describes the epidemiological techniques available for measuring vaccine efficacy and recommends a practical approach to their use, and concludes that screening techniques are the most useful and rapid means of determining whether there is a problem with a vaccine. Other studies that have been conducted to investigate the eradication or elimination of the measles include De Quadros et al. (1998) and Cutts et al. (1999).

In addition to the field studies and other biologically based investigations, there have also been several investigations of the measles that were conducted using quantitative models. Deterministic and stochastic models used to simulate transmissions and events of a measles epidemic have been

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used to investigate control options for the measles (Anderson et al., 1992), as well as regional effects on measles transmission (Sattenspiel and Dietz, 1995). Kribs-Zaleta and Velasco-Hernández (2000) researched a simple vaccination model with multiple endemic states. This research concluded that if the aim is to prevent epidemic outbreak, then a large initial number of infective persons can cause a high epidemic level to rise suddenly even if the vaccine-reduced reproduction number is below its norm. Other mathematical models have been created to investigate vaccinations, reproductive numbers, and optimal distribution of vaccines (Scherer and McLean, 2002; Liu et al., 2008). Another such model quantitatively investigated the spread of the measles in New Zealand using an age based structure for the population (Roberts and Tobias, 2000).

Since most epidemiological models are similar in structure, research into a mathematical model for measles can benefit from studies that have been conducted for other diseases. In a study pertaining to influenza outbreaks, Bowman & Gumel created a model to determine the optimal vaccine allocation strategies for minimizing disease burden subject to a fixed cost of the vaccination program (Bowman and Gumel, 2006). The objective of this study is to use the model that was developed to address several scenarios of interest with respect to optimal vaccination allocation strategies. As a conclusion, the model was used to evaluate different vaccination strategies and optimized allocation in order to minimize disease burden when vaccination resources are scarce.

As outlined above, there have been numerous studies conducted to investigate measles epidemics, some of which use quantitative methods. But not all of the research studies involving the measles have been beneficial to the scientific community and the general population. In 1998, Wakefield et al. (1998) presented a study that claimed that adminstration of the measles vaccine to children could lead to the development of autism in said children. This study received a lot of media attention and led to the rise of an anti-vaccination movement in which parents were not inclined to vaccinate their children due to the perceived link between the vaccine and autism. The study has since been retracted from publication, and other studies have been published which contradict their findings (Taylor et al., 1999; Godlee et al., 2011), but the initial publication is still cited by those who are reluctant to vaccinate against the measles.

The resulting anti-vaccination movement is noteworthy because of the potential detriment that it poses to public health. As recently as the year 2000, the Centers for Disease Control had declared that measles had been eliminated from the United States. Elimination does not indicate that the disease has completely vanished, but rather that there are fewer than 1 reported case for every 1,000,000 people per year (Papania et al., 2014). However, with many people deciding not to vaccinate in line with the anti-vaccination movement, there have been increased reports of outbreaks across the country, including an outbreak that originated at Disneyland in December, 2014 (Berman, 2015; Halsey and Salmon, 2015).

Motivated by the recently reported outbreaks of the measles, the purpose of this study presented is to examine an SVIR model of measles transmission and use it to investigate the concept of herd immunity by running simulations at different vaccination levels. The phenomenon known as herd immunity occurs when a sufficient percentage of the population is vaccinated against a disease, thus inhibiting the ability for the disease to spread among the rest of the population. When herd immunity is achieved, the vaccinated and unvaccinated portions of the community are both protected from disease outbreaks.

2. Materials and Methods

2.1. Computational Resources

All simulations were executed utilizing MATLAB® R2015b (The Mathworks, Inc., Natick, MA). The system of equations was integrated using Euler's Method in a code created for this project. Simulations were run on a Toshiba Satellite C55t-B5230 laptop with Windows 8.1 installed as an operating system.

2.2. Mathematical Models

This investigation of the effect of vaccination rates on a potential measles epidemic utilizes a standard SVIR epidemiological model. The overall population is divided into these four components: susceptible people (S) who have not been vaccinated against the disease and may contract it if they come in contact with an infected person, vaccinated people (V) who have received an inoculation against the disease, which greatly reduces the likelihood of catching it when they come in contact with an infected person, infected people (I) who have the disease and the ability to infect others through contact, and the recovered population (R) which represents individuals who have contracted the disease and recovered from it.

A schematic of the flow between the four populations is presented in figure 2.1. People are born into the susceptible population at a rate of η people per day, and people in the susceptible population die at a rate of δS per day. Additionally, people in the susceptible population are vaccinated (and move to the vaccinated population) at a rate of ωS per day, and they become infected through contact with an infected person at a rate of αSI per day. Similar interactions can be noted among the other populations. It is from these interactions that we are able to derive our system of differential equations. The rate of change for any given population will be equal to the rate(s) that people are entering the population minus the rate(s) at which people are leaving the population. Using the susceptible population as an example, people enter the population through birth and leave the population through vaccination, infection, or death. This would lead to the differential equation $\frac{dS}{dt} = \eta - \omega S - \alpha SI - \delta S$. Employing a similar strategy for the other populations, we obtain the following system of differential equations.

$$\frac{dS}{dt} = \eta - \omega S - \alpha SI - \delta S \tag{2.1}$$

$$\frac{dV}{dt} = \omega S - \alpha(1-\sigma)VI - \delta V \tag{2.2}$$

$$\frac{dI}{dt} = \alpha SI + \alpha(1-\sigma)VI - \delta I - \mu I - \gamma I \tag{2.3}$$

$$\frac{dR}{dt} = \gamma I - \delta R \tag{2.4}$$

Within this system of equations, η represents the birth rate (people/day), ω represents the vaccination rate (1/day), α represents the infection rate (1/(people*day)), δ represents the natural death rate (1/day), σ represents the efficacy of the vaccine (unitless), μ represents the measles-induced death rate (1/day), and γ represents the recovery rate (1/day).

In addition to the susceptible, vaccinated, infected, and recovered population values at any given time which will be found upon solving the system of differential equations, we would also like to keep track of the total number of new infections that occur over any given time period. In order to

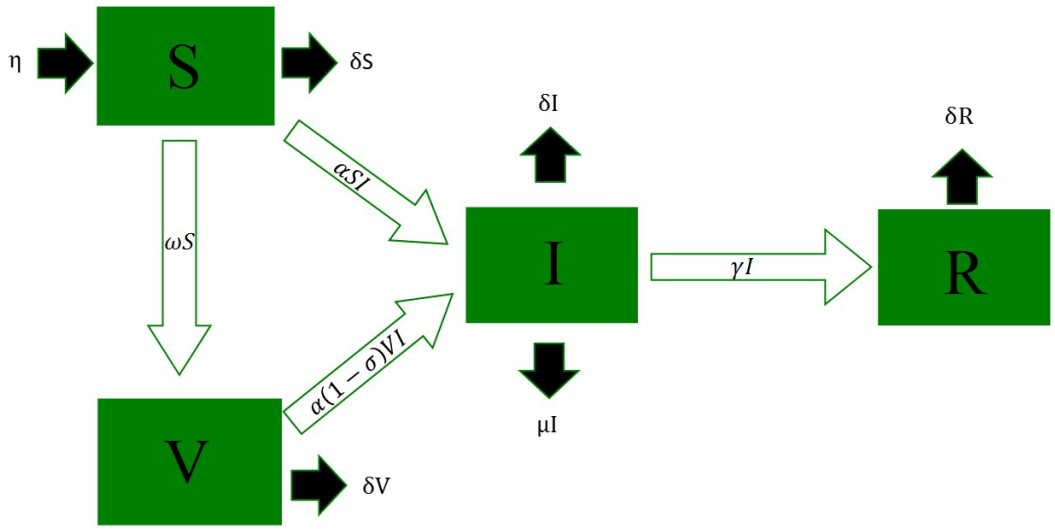


FIGURE 2.1. Schematic of the dynamics of the populations in a basic SVIR model

TABLE 3.1. Parameter values used to simulate closed population dynamics

Parameter	Description	Value	Source
N	Total Population	1,000,0000	assumed
ω	Vaccination rate of susceptible population	0	assumed
α	Infection rate of the Measles	0.95/N	CDC
σ	Efficacy of measles vaccine	0.98	CDC
γ	Recovery rate of infected population	1/18	CDC

keep track of the total number of infections, we add one more differential equation to our system, which is the sum of the new infection terms.

$$\frac{d\Psi}{dt} = \alpha SI + \alpha(1-\sigma)VI \tag{2.5}$$

Equations 2.1 through 2.5 comprise the system which we will solve under various parameterizations to investigate the effect of vaccination on a potential epidemic of the measles.

3. Simulations for a Closed Population

We begin our efforts by examining the results of our system of equations as applied to a closed population. A closed population is one in which nobody enters the system through birth, and nobody leaves the system through death. Thus, we assume that η, δ, and mu are all equal to zero. With no births or deaths occuring, there will always be a constant overall population spread among our four population categories. That is, $S + V + I + R = N$ for all time values in the simulation. Other parameter values used to generate the following results were obtained through a literature search and are reported in table 3.1.

We assume that the vaccinated population is comprised entirely of individuals who were vaccinated initially. That is, no individuals receive a vaccination after the initial outbreak ($\omega = 0$). Simulations were conducted with an arbitrary population size of 1,000,000 individuals. There is initially one infected individual in the population ($I(0) = 1$). The initial susceptible population is defined as $S(0) = (1 - v_p)N$ and the initial vaccinated population is $V(0) = v_pN$, where v_p is the percentage of the population that was initially vaccinated. The initial condition for the recovered population is $R(0) = 0$, as there are initially no recovered individuals.

To investigate the effect of the initial vaccination percentage on the dynamics of a potential measles epidemic, we conducted simulations with v_p=25%, 50%, 75%, and 95%. Results from these simulations within the closed population are presented in figure 3.1.

Upon examination of figure 3.1(a), we see that after about 20 days, there is a sharp decline in the susceptible population, and subsequent increase in the infected population. By about 30 days, the susceptible population approaches zero. The measles outbreak within a population with a 25% initial vaccination rate leads to 809,659 total infections under the assumptions of this model. The results depicted in figure 3.1(b) show similar dynamics to those from the 25% vaccination. The susceptible population also undergoes a rapid decline, beginning around 25 days, and reaching zero at approximately 45 days. The total number of infections when 50% of the population are initially vaccinated is 588,361.

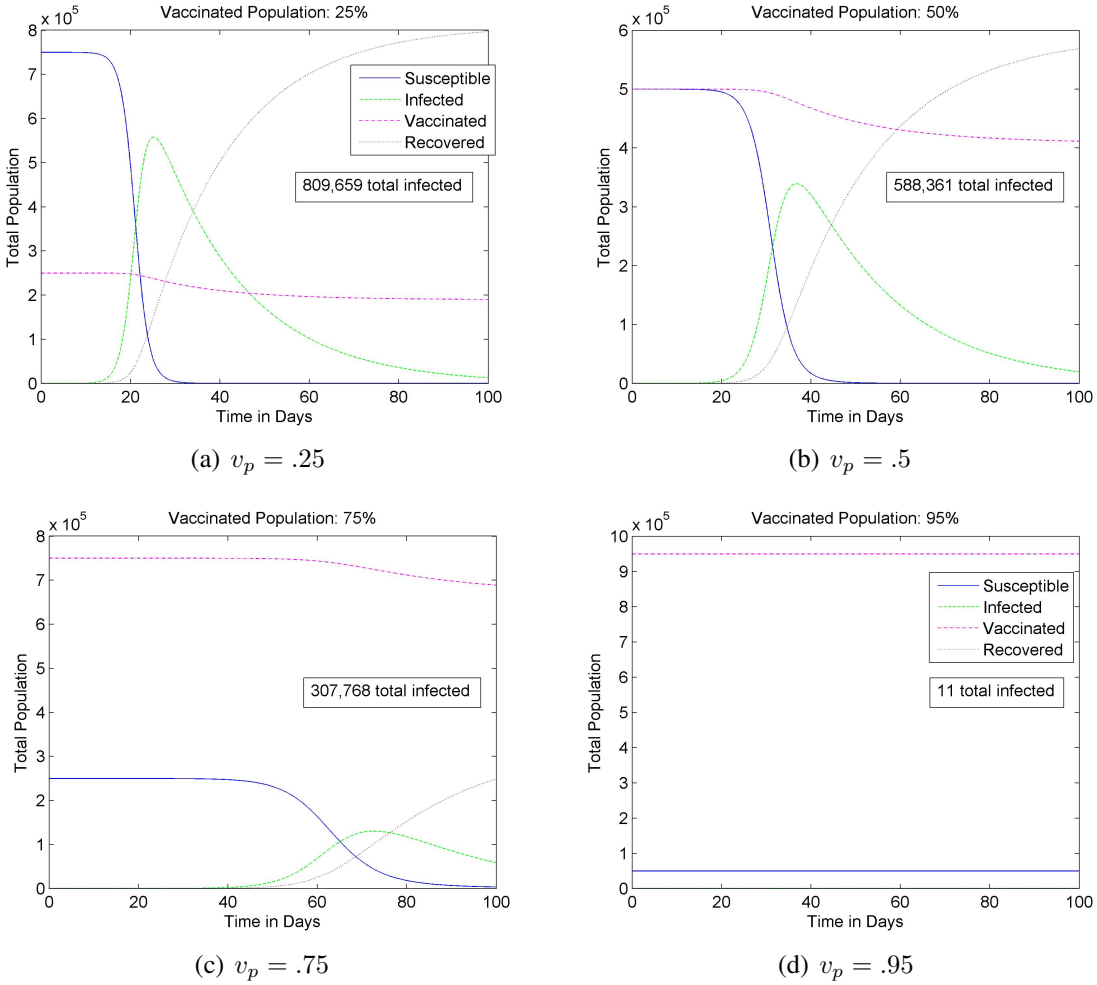
Increasing the initial vaccinated population to 75% of the total population seems to delay the outbreak. Where the outbreak began around 20 and 25 days for the lower vaccination levels (25% and 50%), the susceptible population does not begin a rapid decline until about 45 days, and does not appear to reach zero until about 100 days. The simulation with 75% of the initial population vaccinated leads to 307,768 total infections. Note that for each of the results examined thus far, once the outbreak occurs, the vaccinated population also begins to decrease. This is because the measles vaccine is not 100% effective, and people who are vaccinated may still get infected when an outbreak occurs.

Finally, if we examine figure 3.1(d), which represents the results of the simulation where 95% of the initial population was vaccinated, we notice that the populations remain relatively constant over the 100 day time period. This graph illustrates what would be expected when a herd immunity is achieved. There are a sufficient number of the overall population vaccinated against the disease that it impedes an outbreak and protects those individuals who are unvaccinated.

4. Simulations for an open population

After completing the simulations for a closed population, we then turn our efforts to an open population. We assume that the natural death rate δ is 2.23×10^{-5} per day, the measles induced death rate, μ, is 2×10^{-5} per day, and the natural birth rate, η, is 34 people per day. (The birth rate was determined by multiplying 3.4×10^{-4} times the overall population, N.) Note that the birth rate was adjusted by an order of magnitude from the value reported in **?** to ensure that the population is growing. We originally used the value as reported, but because it was so close to the death rate, the results mirrored those of a closed population. All other parameter values and initial conditions were assumed to be the same as those used for the closed population model.

The results from the simulations of the open population model are presented in figure 4.1. The dynamics of the model for each vaccination level are similar to those reported for the closed population. In the open population, the 25% and 50% levels also predict an outbreak starting between



(a) $v_p = .25$ (b) $v_p = .5$

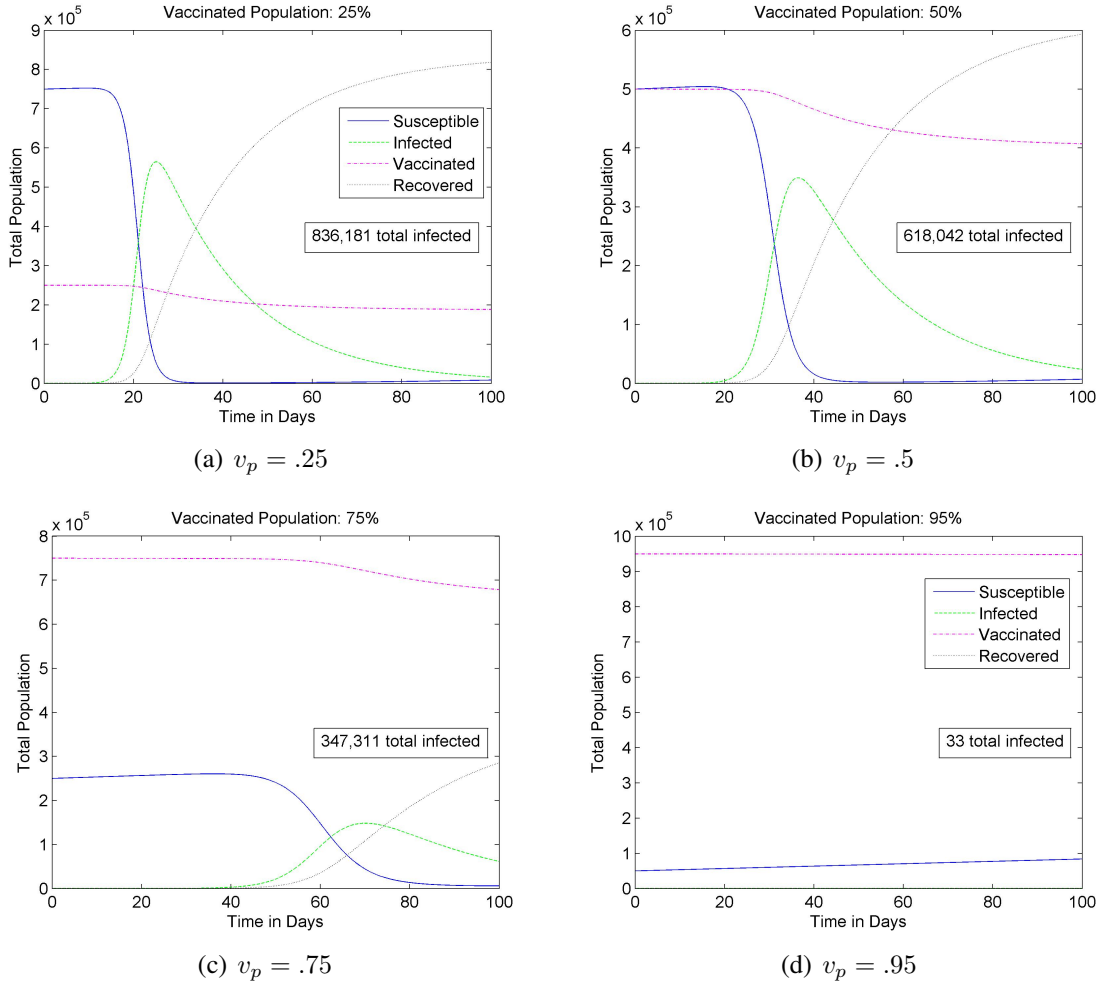
(c) $v_p = .75$ (d) $v_p = .95$

FIGURE 3.1. Simulations of a measles outbreak for a variety of initial vaccination percentages. Each graph depicts the susceptible population (solid line), the vaccinated population (dashed line), the infected population (dash-dot line), and the recovered population (dotted line) versus time.

20 and 25 days, with zero susceptible individuals by 30 and 45 days, respectively. The total number of infections is predicted to be 836,181 when 25% of the population is vaccinated, and 618,042 when 50% of the population is vaccinated.

The results for the 75% vaccination level in an open population are also similar to those of the closed population counterpart. When examining figure 4.1(c), we see that an outbreak still occurs and all of the susceptible population are eventually infected, but it takes longer for the outbreak to begin and end. For the open population, it is predicted that 347,311 total infections will occur when 75% of the population are vaccinated.

In each case, it seems that the biggest difference between the simulations for the closed and open populations is that there are more infections in the open population. This is because we made the assumption that our open population is growing (the birth rate is larger than the death rate), and when the population is growing, there will be more individuals who are susceptible to the disease. This is possibly best illustrated in the results from the open population with 95% of the



(a) $v_p = .25$ (b) $v_p = .5$

(c) $v_p = .75$ (d) $v_p = .95$

FIGURE 4.1. Simulations of a measles outbreak for a variety of initial vaccination percentages. Each graph depicts the susceptible population (solid line), the vaccinated population (dashed line), the infected population (dash-dot line), and the recovered population (dotted line) versus time.

population vaccinated. As with the 95% vaccination rate in the closed population, the number of total infections predicted for the open population is drastically reduced, and it appears that herd immunity has been achieved. While the graphs for the closed population seemed to be horizontal lines, we can see that the graph of the susceptible group in the open population has a positive slope. Again, this occurs because of our assumption that the population is growing.

5. Investigation of the Reproductive Number

A useful analysis of epidemiological models is the investigation of the reproductive number. The reproductive number of an epidemiological system can be thought of as a threshold between the system reaching a disease-free equilibrium versus an equilibrium dominated by infection. The reproductive number can be thought of as the number of secondary cases produced in a population

by a typical infective individual. If the reproductive number is less than 1, then an infected individual produces less than one new infected individual through the course of its infectious period, and the infection will eventually die out. If the reproductive number is greater than one, then each infected individual further infects (on average) more than one other person during the course of the infectious period, allowing the infection to spread (Diekmann et al., 1990; Van den Driessche and Watmough, 2002).

Because our system incorporates vaccinated individuals, we will compute the vaccinated reproductive number, R_v, using the next generation method outlined in Van den Driessche and Watmough (2002), Bowman and Gumel (2006), or Browne et al. (2014). The reproductive number R_v is defined to be the spectral radius of FV^{-1}, where F and V are matrices representing the new infection and transfer terms in the model, respectively.

To find the matrices F and V, we start by considering the Jacobian of the equations representing infected individuals. Since our system only has one equation representing infected individuals, the Jacobian would be as follows.

$$\begin{aligned} J &= \frac{\partial}{\partial I}\left(\frac{dI}{dt}\right) \qquad (5.1)\\ &= \frac{\partial}{\partial I}\left(\alpha SI + \alpha(1-\sigma)VI - \delta I - \mu I - \gamma I\right)\\ &= \alpha S + \alpha(1-\sigma)V - \delta - \mu - \gamma \end{aligned}$$

(5.2)

This Jacobian can be written as the difference between F and V ($J = F - V$), where F is comprised of the terms that arise from new infections, and V is comprised of the terms that are leaving the population due to the various transitions (death, recovery, etc.). For our system, we will have the following.

$$J = \alpha S + \alpha(1-\sigma)V - (\delta + \mu + \gamma) \Rightarrow F = \alpha S + \alpha(1-\sigma)V, V = \delta + \mu + \gamma \qquad (5.3)$$

Thus, FV^{-1} would take the following form.

$$FV^{-1} = \frac{\alpha S + \alpha(1-\sigma)V}{\delta + \mu + \gamma} \qquad (5.4)$$

Plugging $\alpha = 0.95/N$ and our initial conditions into equation 5.4 above, we would have

$$FV^{-1} = \frac{\frac{0.95}{N}(1-v_p)N + \frac{0.95}{N}(1-\sigma)v_pN}{\delta + \mu + \gamma}, \qquad (5.5)$$

which simplifies to yield

$$FV^{-1} = \frac{0.95(1-v_p\sigma)}{\delta + \mu + \gamma}. \qquad (5.6)$$

The vaccinated reproductive number will be the spectral radius of FV^{-1}. Since our result is a constant, the spectral radius will just be $R_v = \frac{0.95(1-v_p\sigma)}{\delta+\mu+\gamma}$.

Now that we have derived a formula for the vaccinated reproductive number, we can compute it for various vaccination levels. Table 5.1 displays the vaccinated reproductive numbers for the open and closed populations for the vaccination levels whose results were shown in figures 3.1 and 4.1. From the table, we can see that as the vaccination percentage increases, the reproductive number decreases. at 95% vaccination, where our results seemed to indicate that a herd immunity

TABLE 5.1. Vaccinated reproductive numbers for the open and closed population models

v_p	R_v (closed)	R_v (open)
0.25	12.91	12.9
0.5	8.72	8.71
0.75	4.53	4.52
0.95	1.18	1.17

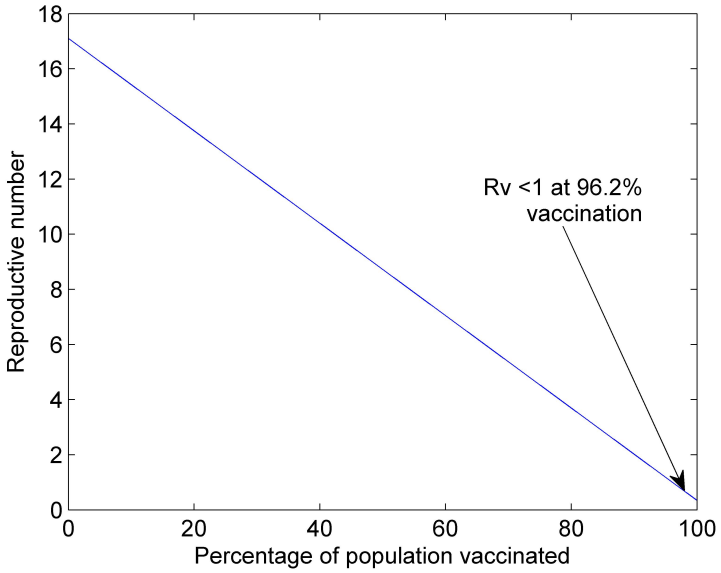


FIGURE 5.1. Reproductive number based on initial vaccination percentage

was achieved, the reproductive number is 1.17 and 1.18 for the closed and open populations, respectively. Since these values are still larger than 1, the disease will not actually die out, although it may be slowed enough to efficiently protect the entire population.

Furthermore, a graph of the vaccinated reproductive number based on the percentage of the population that was initially vaccinated is depicted in figure 5.1. This graph shows a linear relationship between the vaccination percentage and the reproductive number. Examination of the results used to produce this graph show that the reproductive number first falls below 1 (0.978) when approximately 96.2% of the population are vaccinated.

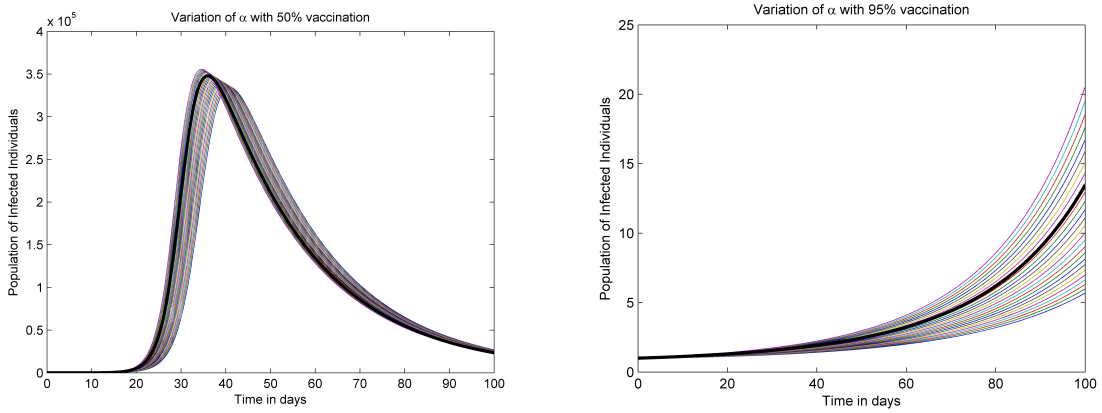
6. Conducting a Visual Sensitivity Analysis

Having demonstrated that approximately 96% of the population would need to be vaccinated to achieve herd immunity under the reported assumptions, the SVIR model can also be utilized to investigate the effects that each parameter has on the model results. A basic visual sensitivity analysis can be conducted by allowing each model parameter to vary (while the other parameters remain the same) and comparing the model results that are generated for each parameter value. This sensitivity analysis can be used to demonstrate how a change in any given parameter affects

the model results, and can further be used to identify which parameters have the greatest effect on the model results.

For this investigation, parameters that are used to determine the reproductive number ($\alpha, \delta, \gamma, \mu$, and σ) were varied and the resulting total number of infections and reproductive numbers were examined. Each parameter was assigned a range that was either based on a range that was presented in literature (γ), based on biologically feasible values (α and σ), or increased and decreased by an order of magnitude (δ and μ). For each parameter, 25 equally spaced values from the parameter range were used to generate the reported results. Figures 6.1 - 6.5 depict the sensitivity results of the infected population for each parameter. The original results are depicted on these graphs by a solid black line. The sensitivity results for the reproductive number are displayed in Figure 6.6. A summary of the parameter ranges and their corresponding total infections and threshold vaccination percentage can be found in Table 6.1.

The infection rate parameter, α, was varied from 0.85 to 1. The results for the infected population that were generated for α values in this range are depicted in Figure 6.1. Examination of the graph generated with 50% of the population vaccinated indicates that lower values of α result in a slight delay in the occurrence of the major outbreak, although it appears that a similar number of people get infected. The results for the 95% vaccination rate also show that higher values of α result in a higher infected population. The range for the total number of people infected when α was varied from 0.85 to 1 was 607,876 to 623,672 (respectively) for a population with 50% vaccination coverage, and 17.696 to 48.91 (respectively) for a population with 95% vaccination coverage. Figure 6.6(a) depicts the variation of the reproductive number for this range of the α parameter. From this graph, we see that there is greater variability in the results of the reproductive number for smaller vaccination percentages. The threshold needed to achieve an R_v value of less than one ranged from 95.3% to 96.4% for this range of α values.

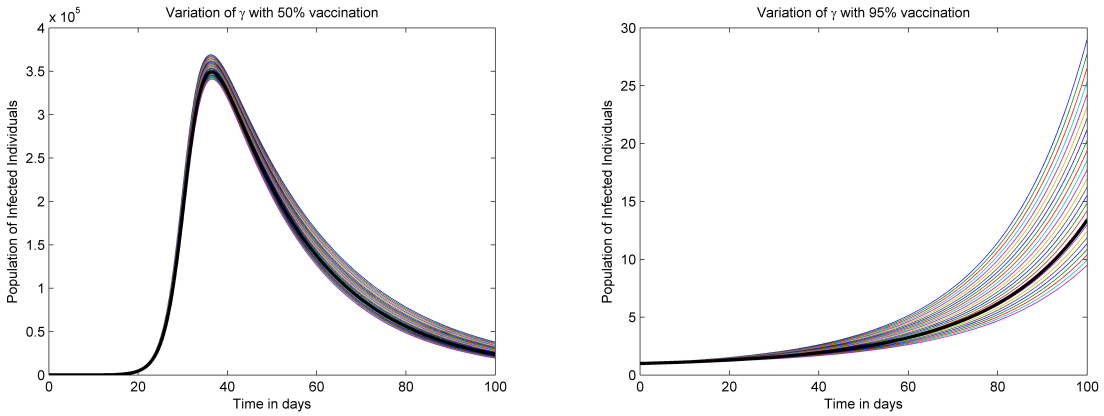


(a) $v_p = .50$ (b) $v_p = .95$

FIGURE 6.1. Simulations of a measles outbreak with the infection parameter α ranging from .85 to 1.

The recovery rate parameter, γ was reported (for Disease Control et al., 2008) to fall between 1/21 and 1/17. Figure 6.2 depicts the infected population results generated using this range for the γ parameter. As γ increases (recovery time decreases), the number of infected individuals decrease. The range for the total number of people infected when γ was varied from 1/21 to 1/17 was 633,209 to 612,695 (respectively) for a population with 50% vaccination coverage, and

60.97 to 27.04 (respectively) for a population with 95% vaccination coverage. Additionally, the variation in the reproductive number can be viewed in Figure 6.6(b). Similar to the results for α, we see more variation of the reproductive number when the percentage of the population that has been vaccinated is relatively low. Simulations showed that the threshold needed to achieve herd immunity with this range for the γ parameter ranged from 96.9% to 95.7%.

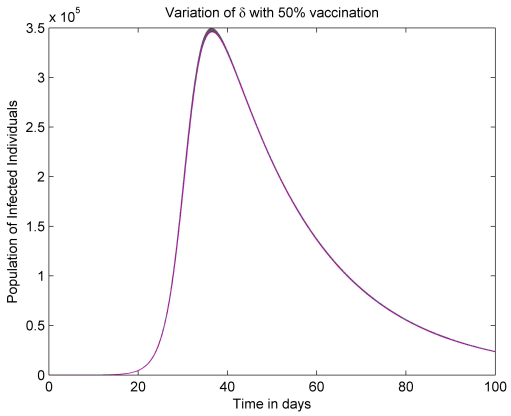


(a) $v_p = .50$ (b) $v_p = .95$

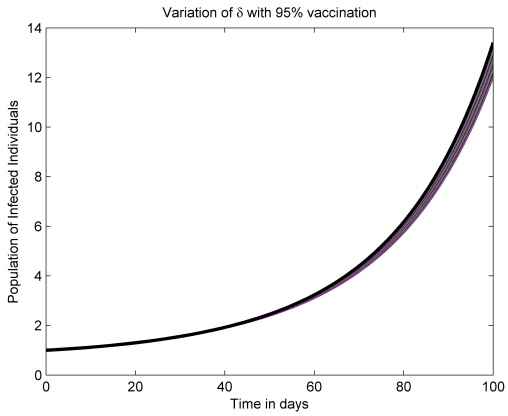
FIGURE 6.2. Simulations of a measles outbreak with the recovery rate parameter γ ranging from 1/21 to 1/17

Results for the sensitivity of the infected population to the natural death rate parameter (δ) and the measles-induced death rate parameter (μ) are displayed in Figures 6.3 and 6.4 respectively. The natural death rate parameter varied from 2.23×10^{-6} to 2.23×10^{-4}, and the measles-induced death rate varied from 2×10^{-6} to 2×10^{-4}. Neither parameter seemed to cause significant changes in the results when it was varied. This may be due to the magnitude of the parameters, or perhaps the short time interval that was used in these simulations. Varying δ produced a range of total infected people from 618,598 to 613,054 at the 50% vaccination level and from 34.22 to 31.717 at the 95% vaccination level. The range of parameters examined for μ resulted in less variation, causing 618,433 to 618,092 total infections at 50% vaccination and 34.465 to 33.28 total infections at the 95% vaccination rate. The sensitivity results for the reproductive number was similar between these two parameters, generating very little variation. Because of the similarity of the results, only the graph for the results generated using δ are depicted (see Figure 6.6(c)). Both parameters yielded threshold values between 96.2% and 96%.

Lastly, the system was examined for sensitivity to the vaccine efficacy parameter, σ, which was allowed to vary from 0.9 to 1. Figure 6.5 depicts the results for the infected population generated using values within this range for σ. When comparing these results to those produced for the other parameters, it is evident that the number of infected people shows the greatest amount of variation with respect to the vaccine efficacy parameter. This is especially clear when we consider the simulation that was run with 95% of the population vaccinated. The largest number of infected individuals at time = 100 days generated by the other parameters was 30. The results from the σ parameter go up to 16,000. The resulting total number of infected people generated by varying σ also showed greater variation than the results from any other parameter. The range of total infected people with 50% vaccination coverage was from 916,235 to 524,941 and from 23,897 to 8.78 with 95% vaccination coverage. The sensitivity of the reproductive number to the vaccine efficacy is

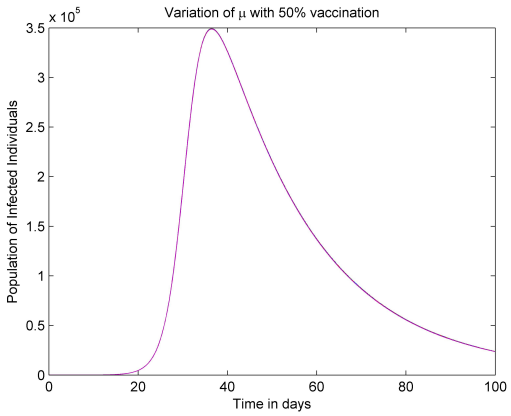


(a) $v_p = .50$

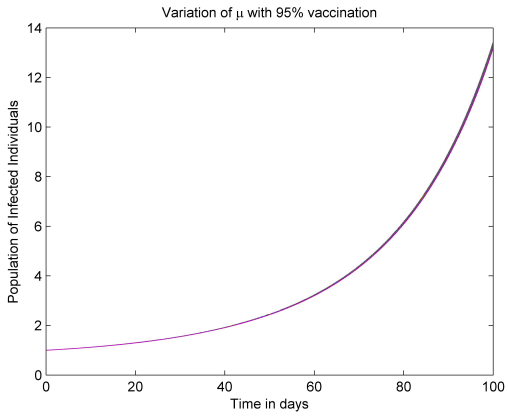


(b) $v_p = .95$

FIGURE 6.3. Simulations of a measles outbreak with the natural death rate parameter δ ranging from 2.23×10^{-6} to 2.23×10^{-4}.



(a) $v_p = .50$



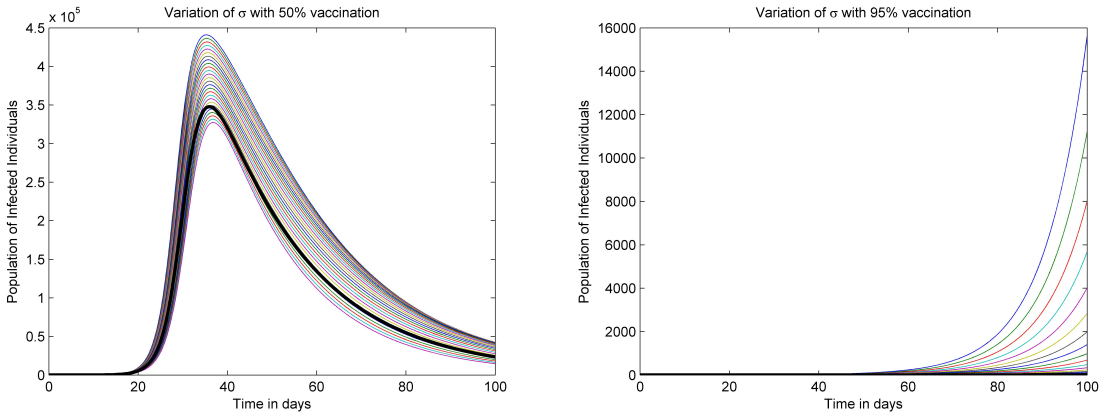
(b) $v_p = .95$

FIGURE 6.4. Simulations of a measles outbreak with the measles-induced death rate parameter μ ranging from .85 to 1.

shown in Figure 6.6(d). Unlike the other parameters, σ seems to cause a greater variation in the results for the reproductive number at higher levels of vaccination coverage. When examining the threshold needed to achieve herd immunity for this range of σ values, we found that at the low end of the range (90% efficacy), it is impossible to achieve herd immunity, and at 100% efficacy, 94.1% of the population would need to be vaccinated. These results indicate that the spread of the measles within a vaccinated population is most affected by the efficacy of the vaccine.

7. Concluding Remarks

In this paper, we have quantitatively examined a potential measles outbreak within a population of one million people with various levels of the population being vaccinated. As one might assume, our results showed that with more vaccinations, there would be fewer individuals who become



(a) $v_p = .50$ (b) $v_p = .95$

FIGURE 6.5. Simulations of a measles outbreak with the vaccine efficacy parameter σ ranging from .9 to 1.

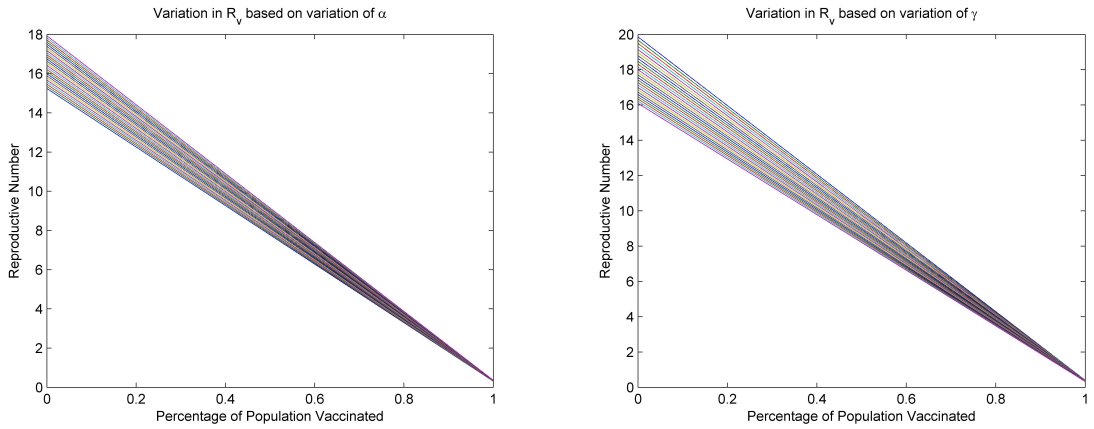
TABLE 6.1. Summary of infected individual and reproduction number results from parameter variation. Results corresponding to the lowest value in the parameter range are presented in bold.

	Parameter Range	Infected ($v_p = 50\%$)	Infected ($v_p = 95\%$)	Threshold
α	**0.85** - 1	**607,876** - 623,672	**17.696** - 48.91	**95.3** - 96.4
γ	**1/21** - 1/17	**633,209** - 612,695	**60.97** - 27.04	**96.9** - 95.7
δ	$\mathbf{2.23 \times 10^{-6}}$ - 2.23×10^{-4}	**618,598** - 613,054	**34.22** - 31.72	**96.2** - 96.0
μ	$\mathbf{2 \times 10^{-6}}$ - 2×10^{-4}	**618,433** - 618,092	**34.47** - 33.28	**96.2** - 96.1
σ	**0.9** - 1	**916,235** - 524,941	**23,897** - 8.78	**[ ]**-94.1

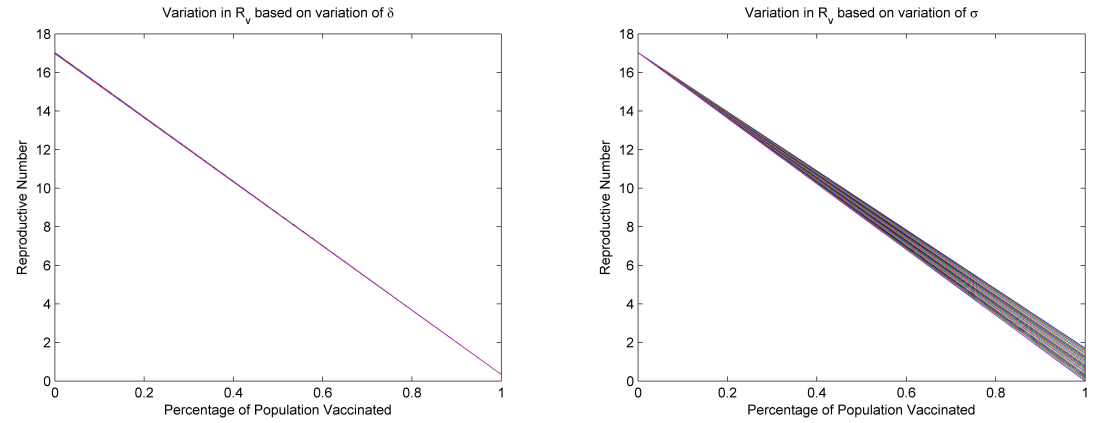
infected with the measles. Our simulations indicated that at a 25% vaccination level, approximately 800,000 of the population of 1,000,000 would become infected with the disease, while only 11-35 would be infected if 95% of the population were initially vaccinated. Further, these results demonstrated that if approximately 95% of the population were vaccinated, then the population would achieve a herd immunity and drastically reduce the effects of this highly contagious disease.

Because there are individuals in our population who cannot get vaccinated against the measles due to medical conditions (cancer patients, those with weakened immune systems, some allergies, etc.), it is important for those who are able to receive the vaccine to be inoculated in an effort to protect themselves as well as the public at large. Establishing a herd immunity effectively protects those who are unable to protect themselves (via vaccination). The results from this project support the current scientific consensus that vaccination is a necessary precaution to protect the public health.

Furthermore, a basic sensitivity analysis was conducted to determine the effect each parameter might have on the simulated infected population and reproductive number. Our study showed that the natural death rate and measles-induced death rate have the least effect on the simulation results. The system appears to be most sensitive to the vaccine efficacy parameter.



(a) variation in α (b) variation in γ



(c) variation in δ (d) variation in σ

FIGURE 6.6. Sensitivity simulations of the vaccinated reproductive number (R_v) dependent on the percentage of the population which is vaccinated (v_p) for a variety of parameter values.

It should be noted that the system of equations that were solved to produce the results in this project were formed under a variety of assumptions that do not necessarily reflect the complex interactions of a society. Future work on this project would include examination of more complicated models that could be used for a heterogenous population, examination of the interactions of multiple populations, as well as a more long-term simulation. These results were generated under the assumption that once the simulation begins, no one else would receive a vaccination. Another future project would involve allowing individuals to receive vaccination during the simulation and using that term to investigate an appropriate age for vaccination to occur. Additionally, a more formal, robust sensitivity analysis could be conducted to further examine the dynamics of the model.

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