Diseases that get "carried" away: The effect of carriers on the spread of an infectious disease.

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Math 201

28 May 2010

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Abstract

When studying the spread of an infectious disease, it is important to consider the existence of infected individuals who do not exhibit symptoms. These "carriers" are still capable of spreading the disease, but are asymptomatic, meaning they do not visibly display any symptoms, and are thus difficult to identify as infected. They will affect the spread of the infectious disease and must be taken into account as a third class of people who do not fall into the usual categories of those who are noticeably infected and those who are susceptible to the disease. Note that this is different from an asymptomatic disease, one in which all infected individuals are asymptomatic, and display no perceptible symptoms. The purpose of this paper is to use differential equations to model the change that arises in a closed population within which an infectious disease is present. By comparing the spread of both a disease that produces only symptomatic infected persons and a disease that allows for a certain fraction of the infected to be asymptomatic, we observe the extended effects of this added class.

Problem Statement

We study the spread of an infectious disease in a closed population. We must first understand the behavior of a disease in which all those infected at time t, $I(t)$, show clear symptoms of the disease. We assume the remainder of the population is susceptible to said disease and is represented at time t by $S(t)$. By modeling the spread of such a disease in Model I, we propose a second population in which asymptomatic individuals, or carriers, are present. By comparing these two models, we are able to observe the effects of transmission of the disease in a population with carriers on the population dynamics.

First, we model a universally symptomatic disease, which accounts for several parameters relevant to the spread of the disease, such as the rate of infection and the rate of recovery. We thus gain an understanding of the disease spread. Using the equations established in Model I, we introduce a new variable representing carriers, and denote it by $C(t)$.

In developing our models, we assume the following:

- The first model considers a population with two subgroups, and the second considers three subgroups
- A population is defined as the total group of people; a subgroup consists of a group of people with a similar relation to the disease (i.e. infected, susceptible, carrier)
- The disease is infectious but curable, meaning the infected can recover and do not die (i.e. no death population)
- There is no acquired immunity for the disease, meaning those who have recovered from the disease return to the susceptible population
- The diseases occur in a closed population and no change in population size due to births or deaths
- In both models, the disease is spread by direct contacts that occur randomly within the population in which susceptible and infected (either symptomatic or asymptomatic) are uniformly distributed

Mathematical Models

Model I: The Spread of a Disease with Universally Perceptible Symptoms (Non-asymptomatic)

We consider a model in which all infected individuals exhibit outward manifestations and are contained in a closed environment. As previously stated, we assume that the disease is infectious and curable where immunity is not possible. We introduce the independent variable t , which is the time in days. The population is divided into two subgroups: those that are infected and symptomatic, $I(t)$, and the remaining members of the population who are not infected but are susceptible, $S(t)$. These two subgroups can then be said to represent the entire population and are related by

$$S(t) + I(t) = 1, \tag{1}$$

where $S(t)$ and $I(t)$ are both fractions of the total population.

We assume that the disease is spread by person-to-person (direct) contact from infected to susceptible individuals and that such contacts occur randomly within the population of uniformly distributed individuals. Therefore, the number of contacts is proportional to the product of S and I, and since $S=1-I$ (Eq. 1), we may write the initial value problem as follows:

$$\frac{dI}{dt} = \alpha I(t)(1 - I(t)) \quad , \qquad I(0) = I_o, \tag{2}$$

where α is the infectivity, and I_0 represents the number of individuals initially infected on day 1.

Solving the initial value problem, (2), using the method of separation of variables we obtain the solution

$$I = \frac{I_o}{I_o + (1 - I_o)e^{-\alpha t}} \quad . \tag{3}$$

Looking at a longer period of time let us consider the limit of the solution as $t \to \infty$ .Then,

$$\lim_{t \to \infty} \frac{I_o}{I_o + (1 - I_o)e^{-\alpha t}} = \frac{I_o}{I_o} = 1. \tag{4}$$

Since the limit approaches 1 (as also shown in Figures 1-3), this means that over time the disease will have spread over the entire population and eventually everyone will become infected and remain so. That is, in this model recovery is not possible.

Analysis of Model I

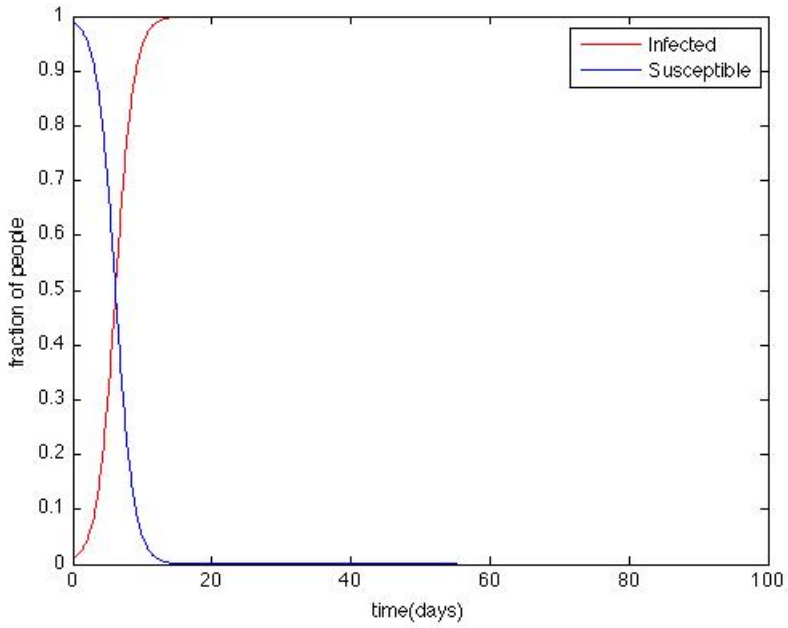


Figure 1: Graphical from of the solution modeled by equation (3), modeling a population of 500 in a closed environment with the contact rate $\alpha = .75$ and $I_0 = 1$.

In the above Figure 1, the infected and susceptible subgroups are equal at about 10 days, after which the fraction of infected exceeds the fraction susceptible. This graph also shows that within 20 days the entire population is infected.

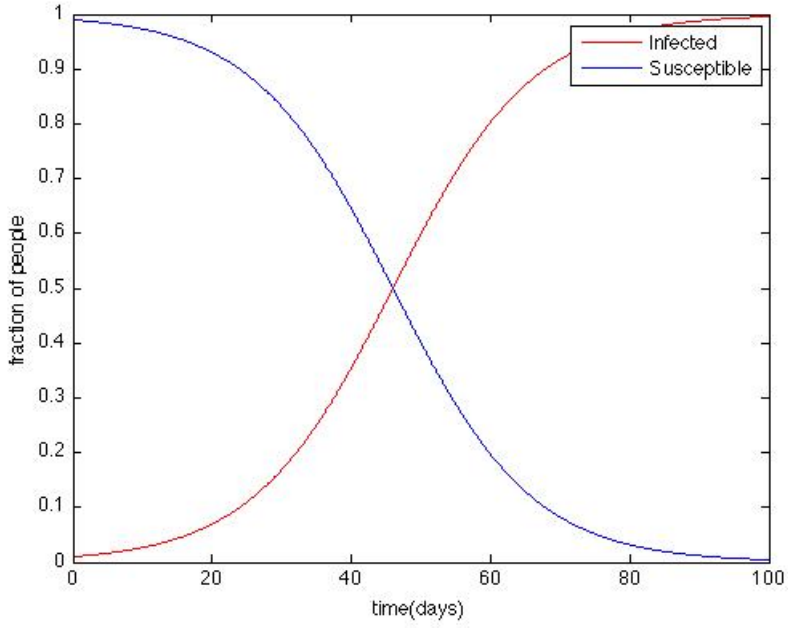


Figure 2: Graphical representation of the solution represented by equation (3), modeling a population of 500 in a closed environment with the contact rate $\alpha = 0.10$ and $I_0 = 1$.

In this figure $I = S$ at about 40-45 days with the entire population becoming infected after about 80 days. Since the only parameter altered between Figures 1 and 2 is the contact rate, these graphs demonstrate that the contact rate has a large impact on the spread of a disease through a population. The higher the contact rate and infectivity, the more rapid the spread of the disease.

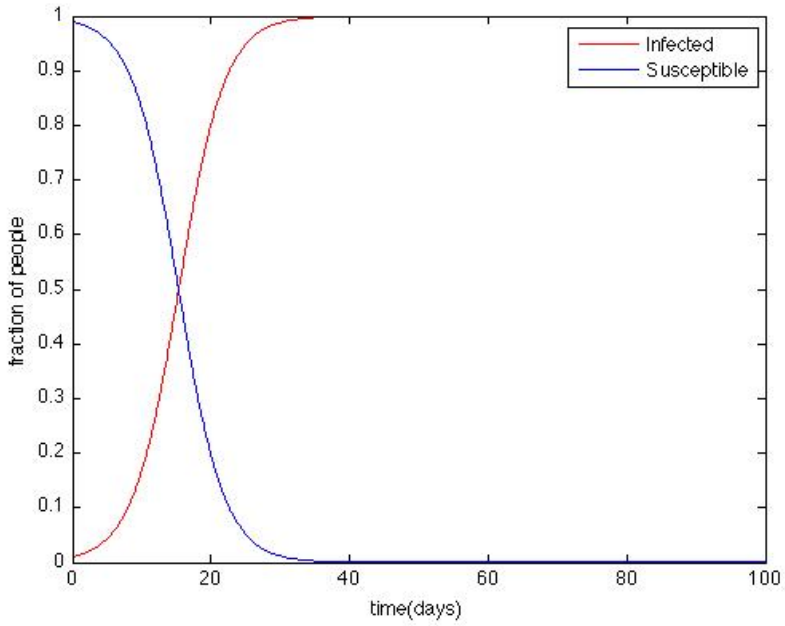


Figure 3: Graphical representation of the solution represented by equation (3), modeling a population of 500 in a closed environment with the contact rate = .30. This models an actual effect of influenza using the infection rate from a study from the Canadian Journal of Infectious Diseases in 2008.

Figure 3 illustrates the application of this model using actual data from the spread of a disease.

Looking at Figures 1-3 and considering the impact of the parameter , we observe that as α, the contact rate, increases, the fraction of individuals infected increases at a faster rate, as would be expected logically.

However, there exists another parameter to consider. As more individuals are infected with the disease and *I(t)* grows, individuals are also leaving the infected category by being cured . We must then introduce a second proportionality factor, β, which accounts for the change in the number of those susceptible to the disease. The relationship between these factors and the population fractions is depicted in the following diagram.

Diagram of Model I

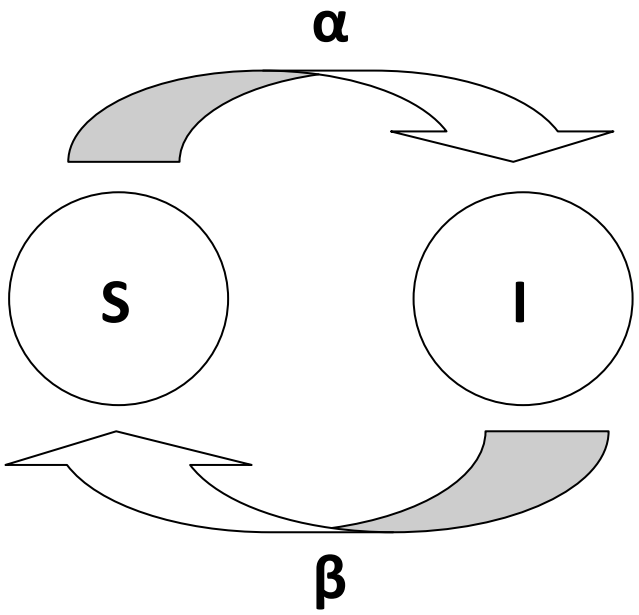


Diagram 1: This flow chart depicts Model I, where S is the susceptible fraction, and I is the infected fraction of the population. α is the infectivity, or the rate at which susceptible individuals become infected, and β is the recovery rate, or the rate at which infected individuals are cured, and return to the susceptible fraction of the population.

Therefore, consider the equation

$$I'(t) = \alpha I(t)(1 - I(t)) - \beta I(t),\ I(0) = I_o, \tag{5}$$

where $\alpha, \beta > 0$ and t is the time measured in days.

In this case α still represents the contact rate, but β represents the removal rate of individuals moving from the infected to susceptible subgroup. There this situation is the same as modeled by equations (2)-(4) and Figures (1)-(3) except the parameter α now becomes (α-β).

Solving this initial value problem using separation of variables, we obtain the solution

$$I(t) = \frac{(\alpha - \beta) I_o e^{(\alpha - \beta)t}}{(\alpha - \beta - \alpha) I_o + \alpha I_o e^{(\alpha - \beta)t}}. \tag{6}$$

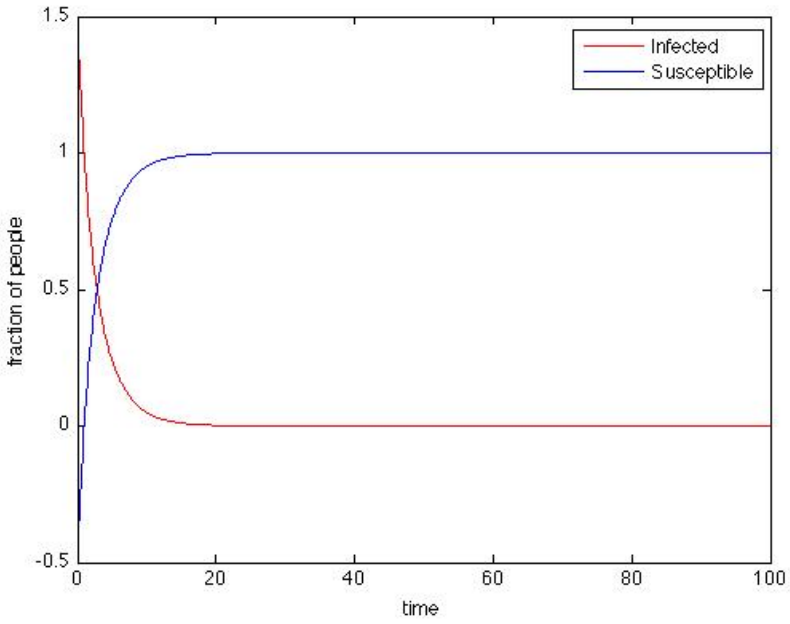


Figure 4: This figure is the graphical representation of the solution represented by equation (6). This models a population of 500 with α=0.1, β=.4, and I_0=1.

In this graph the rate of removal from the infected class is greater than the infective contact rate. As shown by Figure 4, the infected and susceptible subgroups become equal after 1-3 days, which can be considered a fast rate, and eventually the susceptible proportion > infected proportion. Equilibrium appears to be reached within 15-20 days.

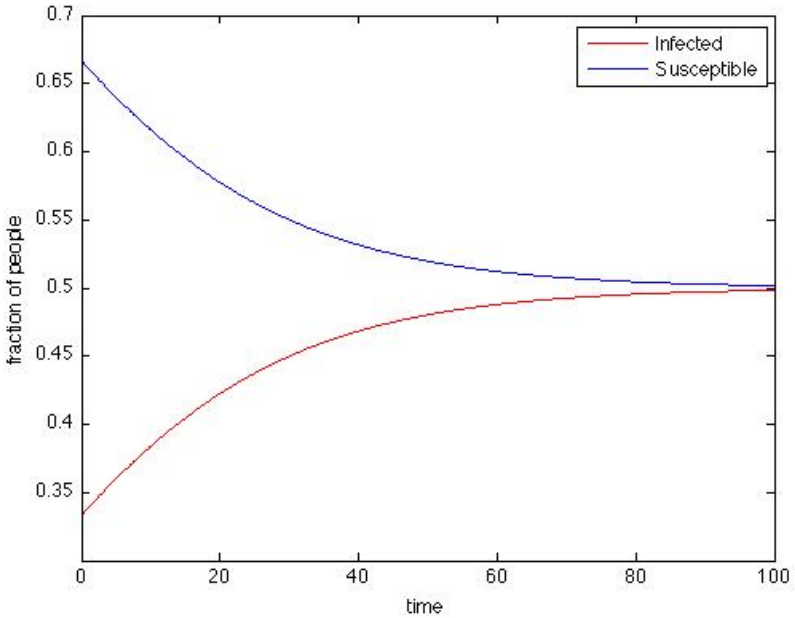


Figure 5: This figure is the graphical representation of the solution represented by equation (6). This models a population of 500 with α=0.1, β=.05, and I_0=1.

In Figure 5 the infective contact rate is larger than the removal rate from infected to susceptible. As compared to the results seen in Figure 4, the time it takes for the two proportions to even out is considerably longer, about 100 days. It is possible that is they are considerably different they may never reach equilibrium, but we will not be considering this case.

When modeling a population of 500 with α=0.5, β=0.5, and I_0=1, i.e. when the infective contact rate is equal to the removal rate from infected to susceptible, no change in the spread of the disease is observed. Therefore, for our purposes we assume that $\alpha \neq \beta$.

Pseudo-code for Figures 1-5

Define parameters a, b, I_0
Define variable t
Zeros vectors I, S, I_2, S_2
Define
I=Equation 3,
S= 1-I,
I_2= Equation 6,
S_2= 1-I_2,
Plot
End.

Model II: The Spread of a Disease with an Asymptomatic Class

Now let us consider a population in which there exist certain individuals who can transmit the disease but possess no outward manifestations. We call these individuals "carriers," or asymptomatic infected. There now needs to be a modification of (Eq. 1) to account for these individuals. Therefore,

$$S(t) + I(t) + C(t) = 1. \tag{7}$$

where *C(t)* represents the fraction of the population that is carriers. In order to minimize the fraction of infected cases, it is desired to remove these individuals from the population.

Analysis of Model II

Consider the following equations:

$$S'(t) = -\alpha_I I(t)S(t) - \alpha_c C(t)S(t) + \beta(I(t) + C(t)), \tag{8}$$

$$I'(t) = \gamma(\alpha_I I(t)S(t) + \alpha_c C(t)S(t)) - \beta I(t), \tag{9}$$

$$C'(t) = (1-\gamma)(\alpha_I I(t)S(t) + \alpha_c C(t)S(t)) - \beta C(t), \tag{10}$$

where

$$C(0) = C_o,$$
$$S(0) = S_o,$$
$$I(0) = I_o,$$

and

$$C(0) = C_0 = 1 - S_o - I_o, \tag{11}$$

where α_I and α_c represent the infective contact rates in the proportion of the populations that are infected and which are carriers, respectively, represents the rate of infected individuals being returned to the susceptible proportion (as in our simplified model), and represents the fraction of new infections that are symptomatic. The relationship between these new factors and the population fractions is depicted in the following diagram.

Diagram of Model II

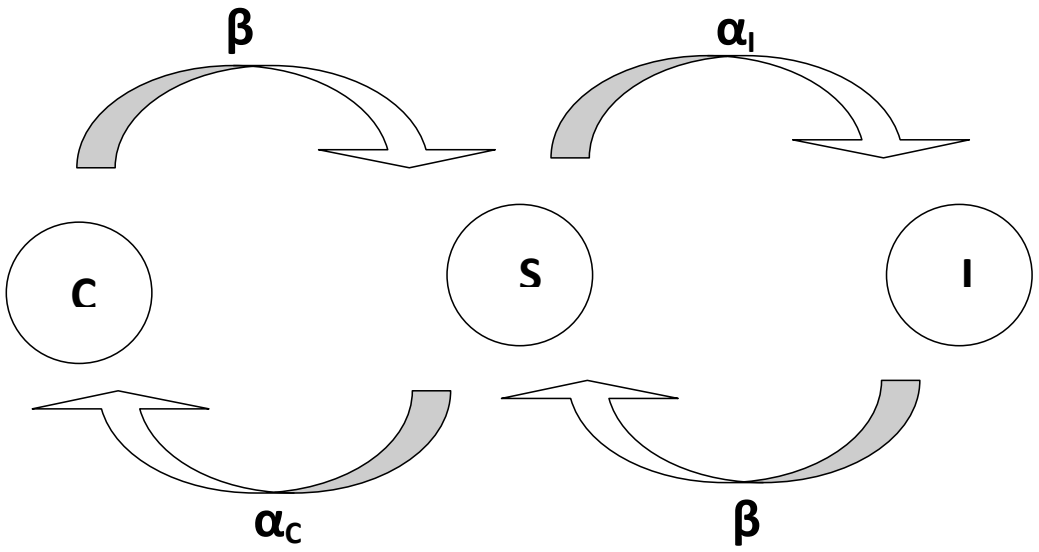


Diagram 2: This diagram depicts Model II, where C is the fraction of carriers, or asymptomatic infected individuals, S is the fraction of susceptible individuals, and I is the fraction of symptomatic infected individuals in the population. α_I is the infectivity, or the rate at which susceptible individuals become symptomatically infected, α_C is the infectivity rate at which susceptible individuals become asymptomatically infected, or carriers, and β is the removal rate of both asymptomatic and symptomatic individuals from their respective subgroups back into the susceptible population.

Also, it is necessary to consider

$$\alpha_I \neq \alpha_c, \tag{12}$$

since the degree of disease transmission from carriers may differ from the degree of transmission by the infected individual due to their lack of symptoms.

The removal from the infected and carrier proportions can be assumed to be the same with the rate β. By using Euler's method, we observe the following trends represented in Figures 6-8.

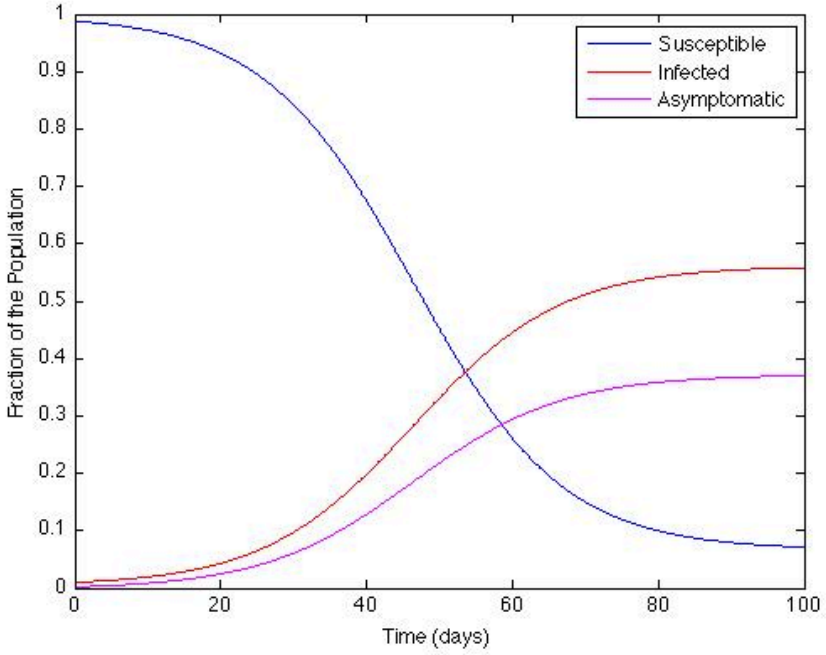
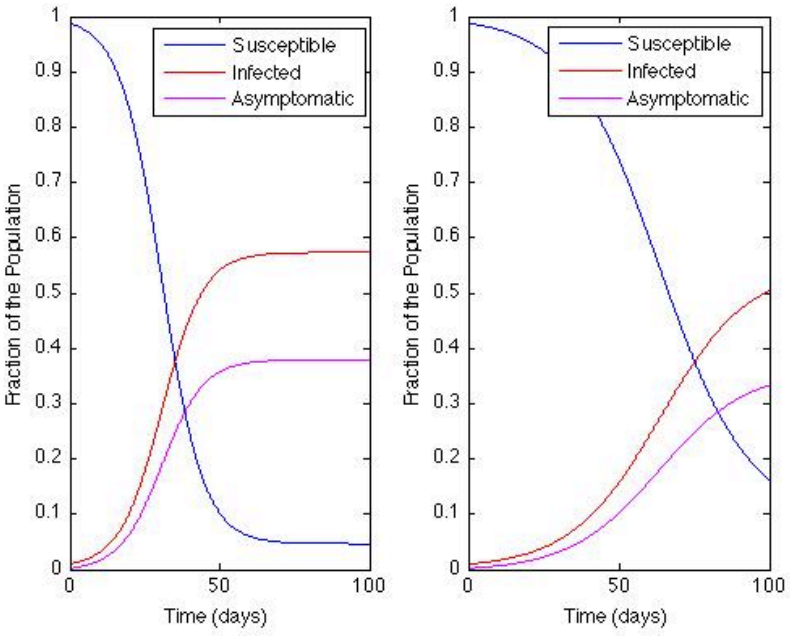


Figure 6: This shows the progression of Model II by using equations (8)-(10) in which the parameters have values: α_I=.075, α_c=.15, β=.007. and γ=.6. The total population is closed and the period of time is 100 days. This graph was created with MatLab.



7a **7b**

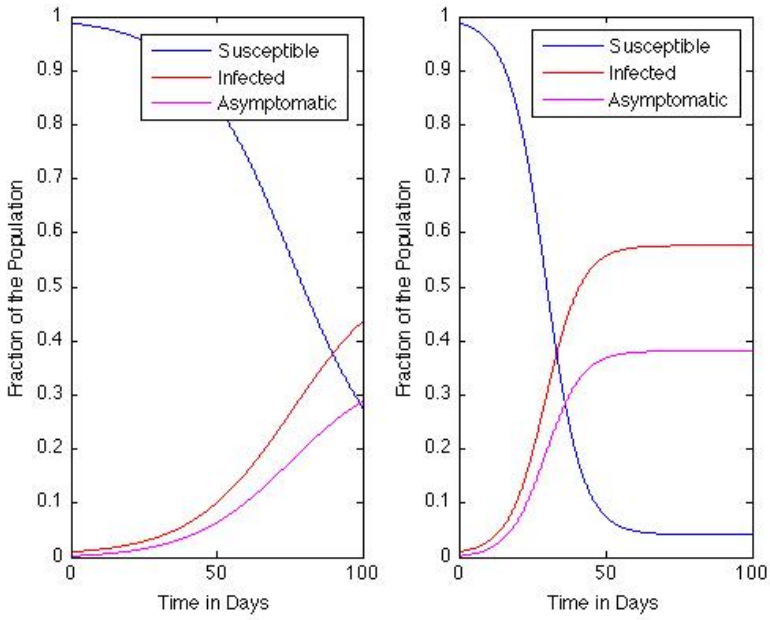
Figures 7a and 7b: : The progression of the spread of disease using equations (8)-(10) with α_I, and α_C are both equal to (a) .15 and (b) .075. The β and γ values remain constant as before. These figures were created with MatLab.

As seen in Figures 6, with set β and γ values, we observe the non-linear relationship of Equations 8-10. When choosing the infective contact rates (α_I and α_C) values, we took into account the increased chance of an asymptomatic person infecting a susceptible person. This is true because contact between an asymptomatic person and a healthy person is more likely because the asymptomatic person does not show any sign of being sick. A healthy person no longer takes care to modify their interactions with these people as they would an obviously symptomatic person, and they are more likely to become infected. As seen in the Figure 6, the susceptible population decreases as time passes and both the number of symptomatic infected and asymptomatic infected people increase over time before all the categories of people level out to what we believe to be the beginning of a steady state at around day 100.

In Figures 7a and 7b, the contact rates are set to equal values to show a situation in which susceptible people did not take care not to interact with those exhibiting symptoms more than those who were asymptomatic. When both α_I and α_C are set to .15, which is the increased contact rate that was previously used for contact with asymptomatic individuals, people are infected at a much more dramatic pace as seen in Figure 7a. The curves reach the perceived steady state around day 50, which is half the time the normal contact rates require. Although the fraction of infected persons increases at a much faster rate initially, the end results of people infected is about the same as when the contact rates are varied. Figure 7b represents contact rates in which the population avoids both classes of the infected equally. This graph shows a slower progression of the spread of the disease and results in a lower fraction of the population infected after 100 days. The curves are also not perceived to reach steady state in this situation.

Application of Model II: Measles

Consulting outside studies, we know that the Measles occasionally produces asymptomatic individuals and that the fraction of the population infected is dependant on seasonality. In the winter months, the measles has a higher contact rate of both asymptomatic and symptomatic infected persons with those susceptible mostly because of the day-to-day contact experienced by children and adolescents in school. In the summer months, however, the contact rates are noticeably lower because the children are not subjected to contact with other children as often. These effects are shown in the figures below.



8a **8b**

Figures 8a and 8b: The effect of seasonality on the progression of the spread of the measles using equations (8)-(10) during (a) summer months with α_I=.05 and α_C =.09 and (b) during winter months with α_I=.08 and α_C =.3. These figures were made with MatLab.

As seen in Figure 8a, the lower contact rates present during the summer months causes the a slower increase in those infected and a slower decrease in the fraction of the population susceptible. At the end of 100 days, steady state is not reached and the fraction of the population susceptible is about equal to the fraction of the population who are infected and asymptomatic. In Figure 8b, however, the contact rates that exist during the winter, school months are documented in relation to the spread of the measles. The drop in the fraction of people susceptible is much more dramatic than seen in 8a. Also, the curves all seem to reach steady state around day 50. The total fraction of the population infected was greater overall at the end of 100 days in the winter months than in the summer months.

Pseudo-code for Figures 6-8

Define variables T, t, dt
Define parameters B, A_1=α_I , A_2=α_C, g
Zeros vectors I, A, s

Define S(1)
.
.
For t = 0 until t = n
I(n) = I(n-1)+dt*Eqn. 9
A(n) = A(n-1)+dt*Eqn. 10
S(n) = 1-I(n)-A(n)

End
Plot.

Further Implementation and Solutions

We now solve the model with a particular solution for equations (8)-(10). We set equations (8)-(10) equal to zero and first solved for *S(t)*. Then we substituted for *S(t)* in the following equations and solved for *I(t)*, *S(t)*, and *C(t)* using separation of variables. For a more detailed description of the solution process, see Appendix A. The solutions are represented by the equations

$$I(t) = -\frac{\gamma(\gamma\alpha_I - \beta)}{\gamma\alpha_I}, \tag{13}$$

$$C(t) = \frac{(1-\gamma)(\gamma\alpha_I - \beta)}{\gamma\alpha_I}, \tag{14}$$

and

$$S(t) = \frac{\beta}{\gamma\alpha_I}. \tag{15}$$

Our next step was defining a function

$$W(t) = \alpha_I I(t) + \alpha_C C(t), \tag{16}$$

which represents all the infected population.

Taking a linear combination of (9) and (10), we can then obtain

$$W'(t) = [\gamma\alpha_I + (1-\gamma)\alpha_C)S(t) - \beta]W(t), \tag{17}$$

the solution of which, using separation of variables, is expressed by

$$W(t) = W_0 \exp[\lambda \int_0^t S(u)du - \beta t], \tag{18}$$

with

$$\lambda = \alpha_I + (1-\gamma)\alpha_C, \tag{19}$$

and

$$W_0 = W(0) = \alpha_I I_0 + \alpha_C C_0 \ , \tag{20}$$

where λ represents the adjusted infectious contact rate.

Since we now have a solution representing the entire infected population we can compare this to S(t) representing the susceptible population. We can also compare this to the solution in our previous simple model, which accounts for the effect of carriers on the spread of the disease.

The equilibrium situation for the model occurs as . The equilibrium solution for the system is solved by setting the right hand sides of equations (8)-(10) equal to zero, and using (15). Assuming that W_o does not vanish, for this implies that the disease would die out and our model would not be valid, it follows that

$$\lim_{S\to\infty}(8) - (10) = \frac{\beta}{\lambda}, \tag{21}$$

$$I_\infty = \gamma(1 - \frac{\beta}{\lambda}), \tag{22}$$

and

$$C_\infty = (1-\gamma)(1 - \frac{\beta}{\lambda}). \tag{23}$$

In our further analysis we hope to demonstrate that the consequences of our model are highly dependent on the proportion of the parameters β and λ, and therefore the contact rates, and the proportion of each subgroup *I(t)*, *S(t)* and *C(t)* can be considered in terms of these parameters, and that our solution are tending toward steady states.

If the limits (21-23) tend toward high valued steady states, it means that the removal rate, β, of individuals from the infected class back to the susceptible class is high and that it keeps the infected rate from getting undesirably high. It is also higher than the contact rate, λ, meaning the size of the infected class is low. If the limits tend toward a low-valued steady state it means that the adjusted contact rate is higher than the removal rate and therefore the probability of getting infected is higher and more frequent than the removal of infected individuals back to the susceptible class. This would mean a large number of infected individuals within the population.

DISCUSSION

Through our model we demonstrated the spread of an infectious disease within a closed population. We considered two cases, one in which all infected individuals were symptomatic and one in which there were "carriers" present, or individuals with no outwardly manifesting symptoms. In both cases the spread of the disease is dependent on several parameters, including the contact rate among individuals and the removal rate of individuals leaving the infected class and returning to the susceptible fraction of the population. In the case where carriers are present, however, there are different contact rates between the symptomatic individuals and the carriers and the susceptible class. These contact rates are not the same, and the rate is higher with carriers. This is logical because when there are no visible symptoms people fail to make a conscious effort to avoid these individuals and therefore the carriers will have a higher contact rate and a higher chance of infecting someone from the susceptible class.

We specifically modeled the spread of measles and considered seasonality as a variable. Some diseases have certain seasons in which the rate of infection is higher among a population. In the case of the measles there is a higher rate of infection in the winter months when children are in school and therefore have higher day to day contact with other children. Since this contact rate is higher the amount of individuals infected increases at a faster rate and after 100 days there are more infected people in winter than in the summer.

There are several limitations to our model. First of all, one of the assumptions made, as mentioned in the Problem Statement above, is that we are considering a closed population. This is rarely if ever the case in reality. Different populations interact with one another as well as the environment on a daily basis. Also, we assume disease is spread through direct contact only. Most diseases can be spread through various other means including the air or shared surfaces. However, this would introduce many extraneous variables that would not coincide with our model, so for our purposes we exclude these possibilities. Finally, we assume that there is no immunity or deaths caused by the disease. In reality this is rarely the case. People with weaker immune systems can die from even rather not virulent diseases, babies can be born, etc. which would change the size of the population. Also, in some diseases, such as chicken pox, where there is a immunity involved after recovery or a recovery time where an individual cannot become infected again. This would again introduce another parameter into our model that we do not consider, but which can be considered and our model could be manipulated for future research o accommodate such a parameter.

CONCLUSION

Our model is a successful start in attempting to predict the course an infectious disease will take within a population. It shows the disease going to a steady state, meaning it can help predict the eventual outcome of a disease. This could help with possible preventative measures such as vaccinations, which would attempt to lower the infected class within a population at any given time. Our model shows that the spread of any disease is heavily dependent on contact rates among individuals, the rate of time it takes for an individual to recover, and whether or not individuals in the disease can be asymptomatic. This can be important general information on what to look out for to stay healthy. Knowing that asymptomatic individuals have a heavy impact on the spread of a disease and that they often quickly lead to a larger infected class can serve as a reminder to always practice good

hygiene skills with every person. Overall our model demonstrates many factors that play into the spread of a disease and being made aware of these factors can lead to solutions that can improve the general health of the population.

Table 1: Parameters used in this Report

Parameters	Representation
α	proportionality (contact rate) factor
β	removal rate from infected class to susceptible class
γ	fraction of new infections
α_I	infective contact rate in infected class
α_c	infective contact rate in carriers

Appendix A: Step-by-Step Solutions for Equations 8-10

Solving equations 8-10 for $S(t)$, $C(t)$, and $I(t)$, we did the following.

Set

$$S'(t) = 0.$$

Then

$$0 = -\alpha_I I(t)S(t) - \alpha_c C(t)S(t) + \beta(I(t) + C(t)).$$

Rearranging, we obtain

$$\alpha_I I(t)S(t) - \beta I(t) = -\alpha_c C(t)S(t) + \beta C(t).$$

Solving for $I(t)$,

$$I(t) = \frac{-\alpha_C C(t)S(t) + \beta C(t)}{\alpha_I S(t) - \beta},$$

and rearranging,

$$I(t) = \frac{C(t)[-\alpha_C S(t) + \beta]}{\alpha_I S(t) - \beta}. \qquad \textbf{(A.1)}$$

Next, set

$$I'(t) = 0.$$

Then

$$I(t)[\beta - \gamma\alpha_I S(t) = \gamma\alpha_c C(t)S(t).$$

Rearranging, we obtain

$$0 = S(t) * \gamma[\alpha_I I(t) + \alpha_c C(t)] - \beta I(t).$$

Solving for *I(t),*

$$I(t) = \frac{\gamma\alpha_c C(t)S(t)}{\beta - \gamma\alpha_I S(t)} \text{ .} \qquad \textbf{(A.2)}$$

Now we set equations **(A.1)** and **(A.2)** equal to each other, obtaining

$$\frac{C(t)[-\alpha_C S(t) + \beta]}{\alpha_I S(t) - \beta} = \frac{\gamma\alpha_C C(t)S(t)}{\beta - \gamma\alpha_I S(t)} \text{ .}$$

Simplifying and solving for *S(t)*

$$S(t) = \frac{\beta}{\gamma\alpha_I} \text{ .} \qquad \textbf{(A.3)} \ (15)$$

We now set *C'(t)* equal to 0 to add another equation to our system of equations.

Thus,

$$C'(t) = (1 - \gamma)[\alpha_I I(t)S(t) + \alpha_c C(t)S(t)] - \beta C(t) = 0.$$

Rearranging, we obtain

$$0 = S(t)[1 - \gamma][\alpha_I I(t) + \alpha_c C(t)] - \beta C(t).$$

Solving for *S(t)*,

$$S(t) = \frac{\beta C(t)}{(1 - \gamma)(\alpha_I I(t) + \alpha_C C(t))} \text{ .} \qquad \textbf{(A.4)}$$

Setting

$$S'(t) = 0$$

again and this time solving for *S(t)*, we obtain

$$S(t) = \frac{\beta[I(t) + C(t)]}{\alpha_I I(t) + \alpha_C C(t)} \text{ .} \qquad \textbf{(A.5)}$$

We now set

$$I'(t) = 0$$

so that

$$0 = \gamma[\alpha_I I(t)S(t) + \alpha_C C(t)S(t)] - \beta I(t).$$

Rearranging, we obtain

$$0 = S(t)*\gamma[\ \alpha_I I(t) + \alpha_C C(t)] - \beta I(t).$$

Solving for *S(t)*,

$$S(t) = \frac{\beta I(t)}{\gamma[\alpha_I I(t) + \alpha_C C(t)]} \text{ .} \qquad \textbf{(A.6)}$$

We now set equations **(A.4)**, **(A.5)**, and **(A.6)** equal to each other,

$$\frac{\beta C(t)}{(1-\gamma)(\alpha_I I(t)+\alpha_C C(t))} = \frac{\beta[I(t)+C(t)]}{\alpha_I I(t)+\alpha_C C(t)} = \frac{\beta I(t)}{\gamma[\alpha_I I(t)+\alpha_C C(t)]}$$

and simplify to obtain

$$\frac{C(t)}{(1-\gamma)} = \frac{I(t)+C(t)}{1} = \frac{I(t)}{\gamma} \quad . \qquad \textbf{(A.7)}$$

From equation (7) we know that

$$I(t) + C(t) + S(t) = 1. \qquad (7)$$

Substituting equation **(A.7)** in terms of *C(t)* (the left third) for *I(t) + C(t),* and equation **(A.3)** for *S(t),* we obtain

$$\frac{C(t)}{(1-\gamma)} + \frac{\beta}{\gamma\alpha_I} = 1 \quad .$$

Rearranging, and solving for *C(t)*,

$$C(t) = \frac{(1-\gamma)(\gamma\alpha_I - \beta)}{\gamma\alpha_I} \quad . \qquad \textbf{(A.8)} \ (14)$$

Substituting this solution for *C(t)* **(A.8)** into equation **(A.7),** we obtain

$$\frac{\frac{(1-\gamma)(\gamma\alpha_I - \beta)}{\gamma\alpha_I}}{(1-\gamma)} = \frac{I(t)}{\gamma} \quad .$$

Simplifying, and solving for *I(t)*,

$$I(t) = -\frac{\gamma(\gamma\alpha_I - \beta)}{\gamma\alpha_I} \quad . \qquad \textbf{(A.9)} \ (13)$$

AUTHOR CONTRIBUTION

Analysis of Model I, Part I of Analysis of Model II, the discussion and conclusion were contributed by Joanna Izewski. The abstract, pseudo-codes, Part II of Analysis of Model II, and graphs and explanations for Model II were contributed by Emily Verbus. The flow charts and descriptions, the solving of the equations for Model II, and the Problem Statement were contributed by Maddie Sokal.

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