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Modeling Network Worm Outbreaks

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MODELING NETWORK WORM OUTBREAKS

by

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ABSTRACT

Due to their convenience, computers have become a standard in society and therefore, need the utmost care. It is convenient and useful to model the behavior of digital virus outbreaks that occur, globally or locally. Compartmental models will be used to analyze the mannerisms and behaviors of computer malware. This paper will focus on a computer worm, a type of malware, spread within a business network. A mathematical model is proposed consisting of four compartments labeled as *Susceptible*, *Infectious*, *Treatment*, and *Antidotal*. We shall show that allocating resources into treating infectious computers leads to a reduced peak of infections across the infection period, while pouring resources into treating susceptible computers decreases the total amount of infections throughout the infection period. This is assuming both methods are receiving resources without loss. This result reveals an interesting notion of balance between protecting computers and removing computers from infections, ultimately depending on the business executives' goals and/or preferences.

TABLE OF CONTENTS

LIST OF FIGURES

CHAPTER 1: INTRODUCTION

Due to their convenience, computers have become a standard in society and therefore, need the utmost care. Not only in a physical sense (hardware) but in a mental state as well (software). This paper will focus on the latter which is affected by malware, although there have been cases of hardware malfunctions from malware programs such as Stuxnet [1] or programs from the group, Equation [2]. Due to the dynamic similarities between digital and biological virus disease spread, we shall utilize compartmental models [3]. By modeling the spread of a worm in a business computer network, we will attempt to find the most optimal method at minimizing a worm's total spread.

We start by formulating a basic model with a combination of other models used in both biological and digital virus spreads. Once the simple case has been investigated and concluded, we will examine further by introducing another process/flow in the compartmental model which introduces the possibility of computers avoiding the infection phase.

Since we are trying to model a worm outbreak over a local business network, we are going to make some assumptions to simplify the model. We must have the company maintain normal business operations, otherwise the company could simply shut down temporarily and pour all resources into fixing the error immediately. By assuming continuous business operations, our model can better simulate a virus spread since infections can still occur. Our model is based off a single virus outbreak as opposed to a multiple worms/viruses spreading throughout the network. Lastly, we require the company to have some sort of software capability of combating the network worm.

CHAPTER 2: MODEL PRELIMINARIES

Compartmental models was introduced in 1927 by Kermack and McKendrick [3] to simulate a biological disease spread among a population at a basic level. Compartmental modeling is the characterization of different phases an individual experiences during a disease spread. The governing equations of the model are formed by the different rates at which subjects transfer between different phases/compartments. The first form of this model that was introduced was the *SIR* (*Susceptible-Infectious-Removed*) model.
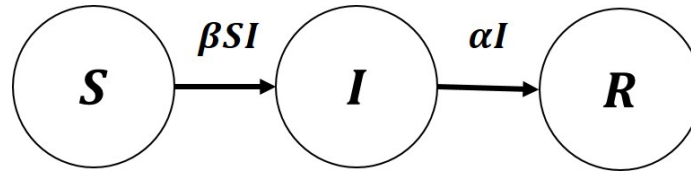
Figure 2.1: Compartmental flow diagram of the *Susceptible-Infectious-Removed* model

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - \alpha I$$
$$\frac{dR}{dt} = \alpha I,$$

where $S = S(t)$, $I = I(t)$, $R = R(t)$ represent the number of susceptible, infectious, and recovered, respectively, individuals at time t; β is the *disease transmission coefficient* and α is the *recovery rate*. Some interesting things can be determined by examining the model carefully. Long term sizes among the different compartments can be determined. Interestingly enough, the susceptible class, S, does not necessarily tend to 0 but can remain finite as $t \to \infty$, i.e., $S_\infty > 0$. [This is proven later in the paper from a similar model.] Here we denote $f_t := f(t)$ and $f_\infty := \lim_{t \to \infty} f(t)$.

However, the infection class, I, will tend to 0 and the removed class, R, will tend to $N - S_\infty$, where N is the total population size. Aside from the final sizes of the compartments, there is a parameter called the basic reproduction number, which can tell us whether the disease spread will be an epidemic or not, denoted as $\mathcal{R}_0$ [4] [5]. It turns out for this particular model, $\mathcal{R}_0 = \frac{\beta S(0)}{\alpha}$ with the following conclusions:

$$\mathcal{R}_0 \leq 1 \Rightarrow \text{no epidemic}$$

$$\mathcal{R}_0 > 1 \Rightarrow \text{epidemic.}$$

Note that these conclusions are typical with basic reproduction numbers in general.

We will mention other models by introducing different types of compartments. First, a biological disease model labeled the *SITR* (*Susceptible-Infectious-Treatment-Removed*) with a new compartment, T relating to a treatment class/phase [6, Chapter 2]. The other model is a computer virus spread model labeled *SAI* (*Susceptible-Antidotal-Infectious*) that adds a new compartment labeled A to represent an antidotal class/phase [7]. Our model will be a composition between the *SITR* and the *SAI* models used while keeping the computer virus spread scenario in mind. Thus we have our model, the *SITA* model (*Susceptible-Infectious-Treatment-Antidotal*).

CHAPTER 3: OUR MODEL AND ANALYTICAL STUDIES

Our model, represented by Figure 3.1, is described by the following ordinary differential equations:

$$\frac{dS}{dt} = -\beta SI - \tau_S S \tag{3.1}$$

$$\frac{dI}{dt} = \beta SI - \tau_I I \tag{3.2}$$

$$\frac{dT}{dt} = \tau_S S + \tau_I I - \gamma T \tag{3.3}$$

$$\frac{dA}{dt} = \gamma T, \tag{3.4}$$

where $S = S(t)$, $I = I(t)$, $T = T(t)$, $A = A(t)$ represent the number of susceptible, infectious, treatment, and antidotal computers, respectively, at time t; β is the *disease transmission coefficient* and γ is the *treatment rate*. Notice, we have two control parameters, τ_S and τ_I, which are defined as *protection rate* and *removal rate* respectively. These are the parameters we would ultimately like to optimize.

Theorem 3.1. *Solutions of (3.1) - (3.4) with non-negative initial conditions $S_0, I_0, T_0, A_0 \geq 0$, satisfy $S(t), I(t), T(t), A(t) \geq 0$.*

Proof. By the standard theory of differential equations [8], the solution to (3.1)-(3.4) with non-negative initial conditions $S_0, I_0, T_0, A_0 \geq 0$ exists uniquely. From (3.1), $\frac{dS}{dt}|_{S=0} = 0$ implying $S(t) \geq 0$ for all $t \geq 0$. Similarly, from Equations (3.2) - (3.4), we have $\frac{dI}{dt}|_{I=0} = 0$, $\frac{dT}{dt}|_{T=0} \geq 0$, and $\frac{dA}{dt}|_{A=0} \geq 0$. Thus $I(t) \geq 0$, $T(t) \geq 0$, and $A(t) \geq 0$ for all $t \geq 0$. $\qquad\square$
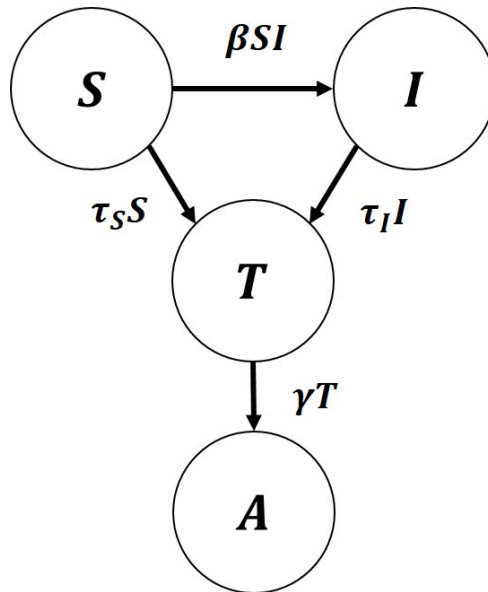
Figure 3.1: Compartmental flow diagram of the *Susceptible-Infectious-Treatment-Antidotal* model

Similar to the basic reproduction number $\mathcal{R}_0$ from the *SIR* model, we will find the ***control* *reproduction number***. From (3.2), we have

$$\frac{dI}{dt} = \beta SI - \tau_I I = (\frac{\beta S}{\tau_I} - 1)\tau_I I,$$

thus we define

$$\mathcal{R}_c = \frac{\beta S_0}{\tau_I}. \tag{3.5}$$

Similarly, we have

$$\mathcal{R}_c \leq 1 \Rightarrow \text{no epidemic}$$

$$\mathcal{R}_c > 1 \Rightarrow \text{epidemic}.$$

5

Before going further, let us quickly define the total population size.

Definition 3.2.

$$N = S + I + T + A,$$

where N is the total number of computers.

Adding (3.1) - (3.4) gives $\frac{dN}{dt} = 0$, which implies $N(t) = N_0$ for $t \geq 0$.

One of our global results we have is the final size in the treatment class.

Lemma 3.3 (Treatment Final Size)**.** *The number of computers in treatment over time approach zero, i.e., $T(t) \to 0$ as $t \to \infty$.*

Proof. Assume a small set of computers are never completely treated, $T_\infty > 0$, then

$$\int_0^\infty T(t)dt = \lim_{t \to \infty} \int_0^t T(s)ds = \infty.$$

Integrating (3.4) from $t = 0$ to $t = \infty$ gives

$$A_\infty - A_0 = \gamma \int_0^\infty T(t)dt = \infty.$$

Thus, $A_\infty = \infty$, but A_∞ is bounded above by N_0, namely, $A_\infty \leq N = N_0$. This is a contradiction, and hence $T_\infty = 0$. $\qquad\square$

3.1 Case 1: Protection Denied

Here, we are assuming no computers are being pulled from the *Susceptible* class into the *Treatment* class, meaning no computers being protected from the worm virus spread, i.e., $\tau_S = 0$. We would

like to determine final sizes for our classes and if there are any equilibrium solutions in the model.

Lemma 3.4 (Equilibrium Solution). *If $\tau_S = 0$, there is a line of equilibrium solutions.*

Proof. At equilibrium, the rate of change vanishes. Thus, setting (3.1)-(3.2) to zero gives

$$\frac{dS}{dt} = -\beta SI = 0$$
$$\frac{dI}{dt} = (\beta S - \tau_I)I = 0.$$

Solving S and I leads to $I = 0$ and $S = \alpha$ for any non-negative constant $\alpha \leq N_0$. Thus $(S, I) = (\alpha, 0)$ for $0 \leq \alpha \leq N_0$ form a line of equilibrium solutions. $\qquad\square$

Lemma 3.5 (Infectious Final Size). *If $\tau_S = 0$, the number of infectious computers over time approach zero, i.e., $I(t) \to 0$ as $t \to \infty$.*

Proof. Assume a finite amount of computers remain infected over the infection period, i.e., $I_\infty > 0$, then

$$\int_0^\infty I(t)dt = \lim_{t \to \infty} \int_0^t I(s)ds = \infty.$$

Integrating (3.4) gives

$$A_\infty - A_0 = \gamma \int_0^\infty T(t)dt.$$

On the other hand, integrating (3.3) yields

$$T_\infty - T_0 = \tau_I \int_0^\infty I(t)dt - \gamma \int_0^\infty T(t)dt,$$

7

thus

$$\gamma \int_0^\infty T(t)dt = T_0 - T_\infty + \tau_I \int_0^\infty I(t)dt.$$

Since $T_0 = T_\infty = 0$, we have

$$A_\infty = \tau_I \int_0^\infty I(t)dt = \infty, \tag{3.6}$$

which contradicts with the fact that $A_\infty \leq N_0$. $\qquad\square$

Theorem 3.6 (Susceptible Final Size). *If $\tau_S = 0$, the number of susceptible computers over time is positive.*

Proof. From (3.1) and (3.2), we obtain

$$dI = (\frac{\tau_I}{\beta S} - 1)dS.$$

Integrating from $t = 0$ to $t = \infty$ gives

$$\int_0^\infty dI(t) = \int_0^\infty (\frac{\tau_I}{\beta S} - 1)dS(t).$$

Thus, we have

$$I_\infty - I_0 = \frac{\tau_I}{\beta} \ln S_\infty - S_\infty - \frac{\tau_I}{\beta} \ln S_0 + S_0.$$

Since $I_0 \approx 0$, $I_\infty = 0$ and $\mathcal{R}_c = \frac{\beta S_0}{\tau_I}$, we have

$$\mathcal{R}_c(1 - \frac{S_\infty}{S_0}) = \ln S_0 - \ln S_\infty. \tag{3.7}$$

8

Hence, we conclude $S_\infty > 0$. □

Another important result we would like to examine is the total amount of infections over the entire infection period.

Theorem 3.7. *If $\tau_S = 0$, the total amount of infections, also known as final disease size (FDS), of the worm outbreak has the relation*

$$FDS = \tau_I \int_0^\infty I(t)dt.$$

Proof. With S_∞ satisfying the relation (3.7), note that

$$S_\infty + I_\infty + T_\infty + A_\infty = N,$$

where N is the total population size. Since $I_\infty = T_\infty = 0$, it follows that $A_\infty = N - S_\infty$, representing the total amount of infections. This is equivalent to removing those that were not infected in the total population. By (3.6), we have $N - S_\infty = A_\infty = \tau_I \int_0^\infty I(t)dt$.

□

This makes sense, since τ_I represents the removal rate (computer per unit time), $\frac{1}{\alpha}$ represents the average infection period (time per unit computer), and $\int_0^\infty I(t)dt$ represents the amount of infections across the disease outbreak, with repeats. So dividing by the average infection period removes repeats and gives us our result.

3.2 Case 2: Protection Applied

Now we are assuming computers are being protected from the computer worm transferring susceptible computers into a treatment class by way of digital quarantine. As in the first case, we shall prove some final size results. Firstly, we study the first two equations of our system, i.e., (3.1) and (3.2).

Lemma 3.8. *If $\tau_S > 0$, there is a unique equilibrium solution at $(S, I) = (0, 0)$ for (3.1) - (3.2).*

Proof. Set equations (3.1) - (3.2) to zero,

$$\frac{dS}{dt} = -\beta SI - \tau_S S = S(-\beta I - \tau_S) = 0$$

$$\frac{dI}{dt} = \beta SI - \tau_I I = I(\beta S - \tau_I) = 0.$$

Since $\tau_S > 0$, $\tau_I > 0$, it follows $S(t) = I(t) = 0$. $\square$

Theorem 3.9 (Final Size Relation)**.** *If $\tau_S > 0$, $(0, 0)$ is globally asymptotically stable for (3.1) - (3.2).*

Proof. First, we show local asymptotic stability. The Jacobian is

$$J = \begin{pmatrix} -\beta I - \tau_S & -\beta S \\ \beta I & \beta S - \tau_I \end{pmatrix}$$

and its evaluation at $(0, 0)$ gives

$$J_{(0,0)} = \begin{pmatrix} -\tau_S & 0 \\ 0 & -\tau_I \end{pmatrix}.$$

Since $\tau_S, \tau_I > 0$, we see that the matrix has two eigenvalues, $-\tau_S$ and $-\tau_I$. Thus, $(S, I) = (0, 0)$ is locally asymptotically stable [9]. For global attractivity, adding (3.1) - (3.2) gives

$$(S + I)' = -\tau_S S - \tau_I I \leq -\tau(S + I),$$

where $\tau = \min\{\tau_S, \tau_I\} > 0$. Integrating the above inequality gives

$$S(t) + I(t) \leq ce^{-\tau t}.$$

By letting $t \to \infty$, we have $S(t) + I(t) \to 0$, as $t \to \infty$. Since $S(t) \geq 0$, $I(t) \geq 0$, we have $\lim_{t \to \infty} S(t) = 0$ and $\lim_{t \to \infty} I(t) = 0$, i.e., $(S(t), I(t)) \to (0, 0)$ as $t \to \infty$. Local stability and global attractivity together imply the global stability of $(0, 0)$. $\qquad \square$

We have established that the solution of our model approaches a steady state solution. Now the question becomes, can we adjust these control parameters to minimize the final virus size. Another interesting aspect we will examine is reducing the maximum amount of infections over the infection period, meaning a company may want a control strategy such that the maximum number of computers that can be infected is bounded above by some threshold. Let us first prove the existence of a maximum with the following:

Lemma 3.10. *The number of infectious computers $I(t)$ achieves its maximum value at some moment $\hat{t}$, and the maximum value is denoted as $I_{max} = I(\hat{t})$.*

Proof. The trivial case is true, i.e., $I(t) = 0$. If not, notice the behavior on $I(t)$ and how it depends on the control reproduction number, $\mathcal{R}_c$, this leads into two cases.

<u>Case 1</u>: Let $\mathcal{R}_c \leq 1$, i.e., $\beta S_0 - \tau_I \leq 0$. Since $\frac{dS}{dt} < 0$ for all $t \geq 0$, we have $S(0) = S_0 \geq S(t)$.

Hence, $\beta S - \tau_I \leq \beta S_0 - \tau_I \leq 0$. From (3.2), we have

$$\frac{dI}{dt} = I(\beta S - \tau_I) \leq I_0(\beta S_0 - \tau_I) \leq 0.$$

Thus, $I(t)$ is monotone non-increasing and $I_{max} = \max_{t \geq 0} I(t) = I(0) = I_0$.

Case 2: Let $\mathcal{R}_c > 1$, i.e., $\beta S_0 - \tau_I > 0$. By equation (3.2),

$$\frac{dI}{dt}\Big|_{t=0} = I_0(\beta S_0 - \tau_I) > 0.$$

This means that the amount of infections are increasing initially, producing an epidemic. However, since $S(t)$ decreases, i.e., $\frac{dS}{dt} = -\beta SI - \tau_S S < 0$ whenever $S > 0$, there exists $t = \hat{t}$ such that $\beta S(\hat{t}) - \tau_I = 0$, and $\beta S(\hat{t}) - \tau_I < 0$ for $t > \hat{t}$. Hence $\frac{dI}{dt} = I(\beta S - \tau_I) < 0$ for $t > \hat{t}$. This implies $I(t)$ increases when $t < \hat{t}$ and decreases when $T > \hat{t}$, yielding a maximum value of $I(t)$ at $t = \hat{t}$. $\qquad\square$

To examine this result,we shall first derive a useful expression.

Lemma 3.11. *(First Integral of SITA Model) Our model admits a First Integral,*

$$V(S, I) = \beta(S + I) - \tau_I \ln(S) + \tau_S \ln(I) = constant.$$

Proof. From equations (3.1) - (3.2), we have

$$\frac{dI}{dS} = \frac{\beta SI - \tau_I I}{-\beta SI - \tau_S S} = \frac{-I(\beta S - \tau_I)}{S(\beta I + \tau_S)},$$

thus,

$$\frac{\beta I + \tau_S}{I} dI = \frac{\beta S - \tau_I}{S} dS.$$

12

Integrating gives

$$\int (\beta + \frac{\tau_S}{I})dI = \int (-\beta + \frac{\tau_I}{S})dS,$$

namely,

$$\beta I + \tau_S \ln(I) = -\beta S + \tau_I \ln(S) + c,$$

where c is the constant of integration. Solving for c gives

$$\beta(S + I) - \tau_I \ln(S) + \tau_S \ln(I) = c.$$

$\square$

This gives us a very important result, for any time t, the resulting values for S and I satisfy this expression. Consequently, this means we can choose any points in time and plug into the derived expression.

Corollary 3.12. *We have the following result:*

$$\beta(S_0 + I_0) - \tau_I \ln S_0 + \tau_S \ln I_0 = \beta(\hat{S} + I_{max}) - \tau_I \ln \hat{S} + \tau_S \ln I_{max}, \tag{3.8}$$

where $\hat{S} = S(\hat{t})$ with $I(\hat{t}) = I_{max}$.

With this corollary, we would like to observe some behavior regarding the maximum number of infections at any point in time in relation to our control parameters, τ_S, τ_I. Hence, we have the following theorem:

Theorem 3.13. *Assume that $\mathcal{R}_c > 1$, then the maximum number of infections across the infection*

13

period, at time $t = \hat{t}$, is decreasing if there is an increase in the protection rate and/or the removal rate, τ_S and τ_I respectively.

Proof. Differentiating (3.8) with respect to τ_S gives

$$\ln I_0 = \beta \frac{\partial I_{max}}{\partial \tau_S} + \ln I_{max} + \frac{\tau_S}{I_{max}} \frac{\partial I_{max}}{\partial \tau_S}.$$

Thus,

$$\frac{\partial I_{max}}{\partial \tau_S} = \frac{\ln \frac{I_0}{I_{max}}}{\beta + \frac{\tau_S}{I_{max}}}.$$

Since $I_{max} > I_0$, we have $\ln \frac{I_0}{I_{max}} < 0$. Therefore $\frac{\partial I_{max}}{\partial \tau_S} < 0$, implying that I_{max} is decreasing as τ_S increases. On the other hand, differentiating (3.8) with respect to τ_I gives

$$-\ln S_0 = \beta \frac{\partial I_{max}}{\partial \tau_I} - \ln \hat{S} + \frac{\tau_S}{I_{max}} \frac{\partial I_{max}}{\partial \tau_I}.$$

Hence,

$$\frac{\partial I_{max}}{\partial \tau_I} = \frac{\ln \frac{\hat{S}}{S_0}}{\beta + \frac{\tau_S}{I_{max}}}.$$

Since $\hat{S} < S_0$, we have $\ln \frac{\hat{S}}{S_0} < 0$. Hence, $\frac{\partial I_{max}}{\partial \tau_I} < 0$, implying that I_{max} is decreasing as τ_I increases. $\qquad \square$

This implies any more resources that are poured into extracting susceptible or infectious computers will, in fact, reduce the peak of infections.

However, we still have yet to examine what would happen to the final virus size when we tune

our parameters which will be studied numerically in the next chapter, and succeeding this is another numerical study we determine what the optimum distribution between τ_S and τ_I would be given some initial constraint, namely $\tau_S + \tau_I = constant = \tau$. Numerical simulations are carried out in the next chapter to explore these issues.

CHAPTER 4: NUMERICAL STUDIES

Matlab was used to simulate the solutions of our model using the command 'ode45', which uses an explicit Runge-Kutta method, the Dormand-Prince pair [10] [11]. The following values for our parameters were chosen: $N = 1000$, $I_0 = 100$, $S_0 = N - I_0 = 900$, $\tau_S, \tau_I \in [.5, 1]$, $\gamma = .1$, $\beta = 0.0039$, and the unit of time t is one day.

4.1 Control Strategies Unconstrained

In Section 3.2, we established the fact that our peak infections should decrease if there is an increase of the protection rate and/or the removal rate. Numerical simulations are carried out for varying values, [0.5, 1], of removal rate τ_I and protection rate τ_S. Specifically, Figure 4.1 shows the decrease of peak infection for fixed τ_I with varying τ_S, Figure 4.2 shows the decrease of peak infection for fixed τ_S with varying τ_I, and Figure 4.3 displays the change of peak infection in terms of varying τ_S and varying τ_I.
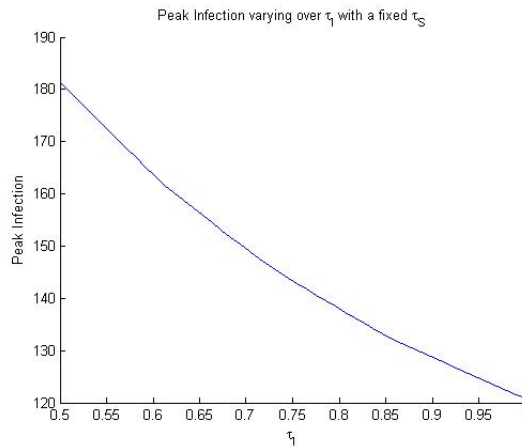

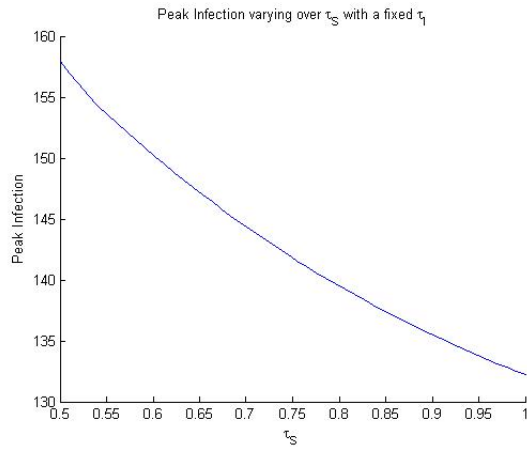Figure 4.1: Peak infections with varying removal rate and fixed protection rate

Figure 4.2: Peak infections with varying protection rate and fixed removal rate
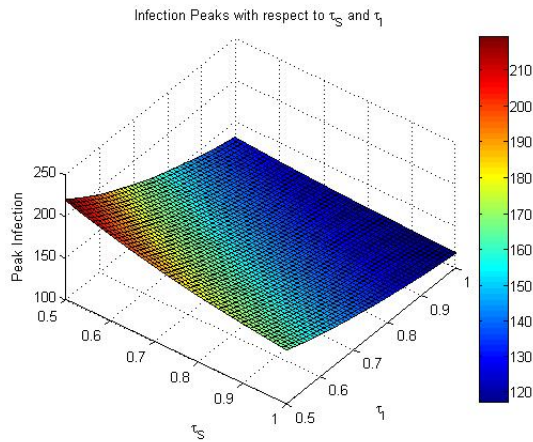

Figure 4.3: 3-D surface plot of peak infections with varying protection and removal rates

The next set of plots (Figures 4.4-4.6) show the relation between the final disease size and control parameters τ_S and τ_I. In particular, this complements the missing qualitative study on this. All simulation results show the monotone dependence, that is, increasing τ_S or τ_I would decrease the final disease size, and we only show one typical set of plots here.
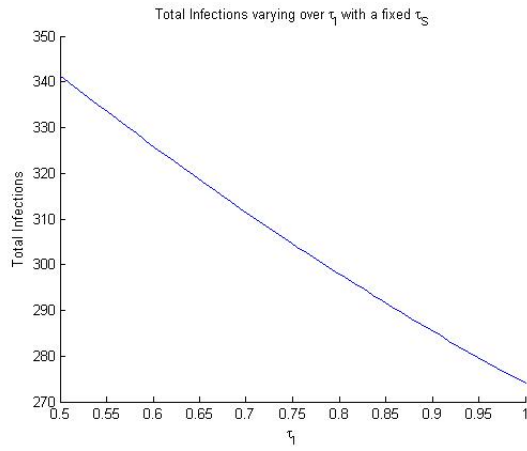
Figure 4.4: Final disease sizes with varying removal rate and fixed protection rate
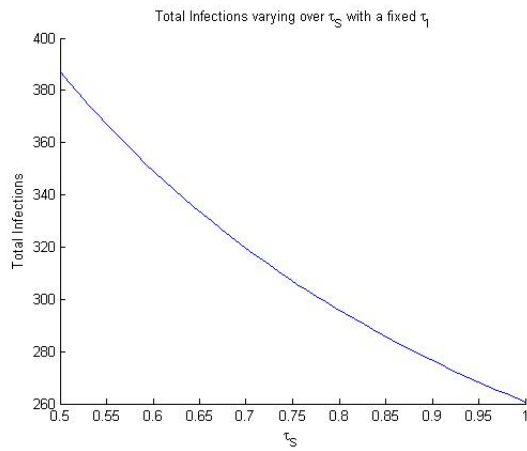

Figure 4.5: Final disease sizes with varying protection rate and fixed removal rate
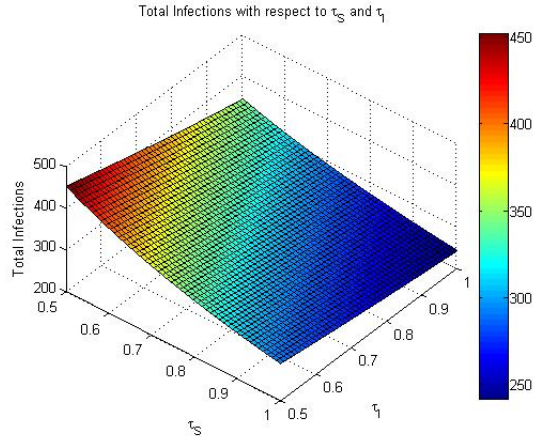
Figure 4.6: Final disease sizes with varying protection and removal rates

4.2   Control Strategies Constrained

Since the resources to treat or to protect computers is often limited, it is more interesting to consider the case with a constraint on τ_S and τ_I. In this section, we may assume $\tau_S + \tau_I = \tau$, where τ is a positive constant representing the available resources for either treatment or protection. We would like to determine which parameter values, τ_S and τ_I can minimize peak infections and final disease size . In the simulations, we chose $\tau = 1.5$.

For Figure 4.7, the peak infection values tend to be smaller as τ_S and τ_I become larger. However, based on the curvature, one can see that the smallest value is located around the upper left corner of the contour image. Since we only focus on values satisfying our constraint, the most upper left value on the black line represents our peak infections minimum. In this case, the control parameter values that correspond to this minimum peak infection would be $\tau_I = 1, \tau_S = 0.5$. That is, if more sources were allocated to removing infectious computers from the functional network, the lower the maximum number of infections that can be across the entire infection period.
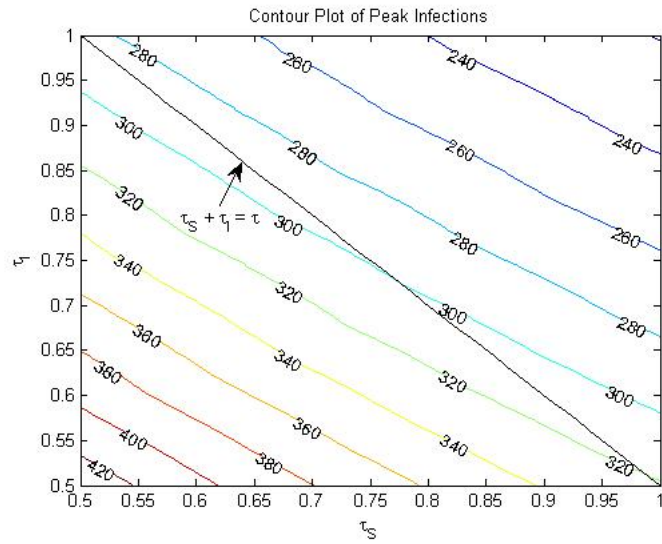
Figure 4.7: Contour plot of the peak infections over τ_S and τ_I with constraint

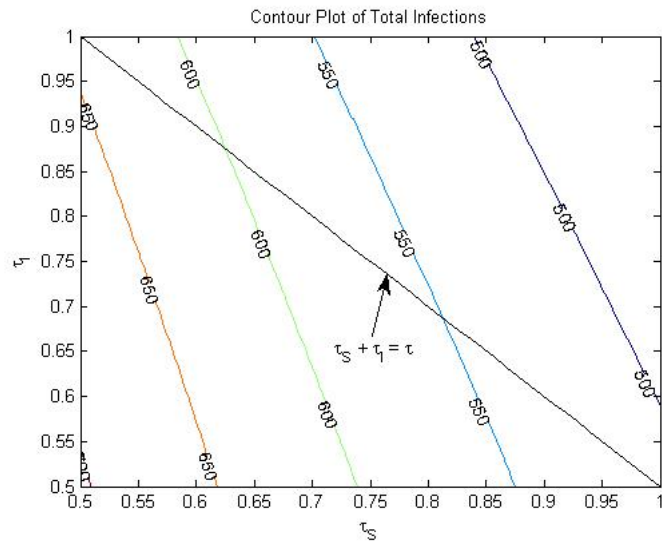Figure 4.8: Contour plot of the final virus size over τ_S and τ_I with constraint

For Figure 4.8, this behaves similarly to our peak infections contour in regards to a lower final virus size being caused by an increase in both τ_S and τ_I. However, the curvature on this contour plot angled toward the lower right. Following along the constraint line, we find that the minimum for

the final virus size occurs at the parameter values: $\tau_I = .5, \tau_S = 1$. Based on our initial parameters, the total amount of infections across the entire infection reaches a minimum if resources are poured into protecting more susceptible than infectious computers.

CHAPTER 5: CONCLUSIONS/FUTURE WORK

Based on our initial parameter values, we have seen that peak infections tend to decrease and are minimized when more resources are allocated into removing infectious computers to avoid further disease spread. Conversely, reducing the final virus size would require more resources being distributed into protecting susceptible computers from being infected. From the infectious distribution perspective, it would appear an increase of τ_S contracts the function since the peak infections increase but reduce the final virus size. Conversely, an increase of τ_I appears to expand the infectious distribution by lowering the peak infections but ultimately increasing the final virus size. The challenge comes into play when the decision needs to be made as to which control parameter should receive a majority of resources.

The key to determining where resources should be distributed depends on the goals of the business' shareholders. If there is to be some tolerance as to how many computers are allowed to be infected at any point in time, then resources should go into removing infectious computers, note this also implies a reduction in average infection period per computer. Imposing this type of infectious constraint would be practical if the company were to assume regular business operations while trying to meet demands on time. Alternatively, if there is a long term concern in regards to damage of the computer network, then resources should be more focused on protecting computers from infectious computers. Ultimately, the preferences and short/long term goals of the company executives affect which control strategy to take.

To allow improvement in the model, we have proposed some concepts to account for other factors not addressed initially. Inject demographic data regarding the arrival of new purchased computers or the deletion of outdated computers. One could impose a continuous constraint for the maximum

number of computers allowed to be in the *Treatment* class at any point in time, utilizing optimization techniques would be helpful here. There is the possibility of allowing some computers to never be completely treated, perhaps due to lack of resources or some business constraints; this can be thought of as a factory reset, i.e., *Treatment → Susceptible*. Initially, we had assumed the company had a means of combating the virus on computers individually, to account for previous points in time, we may allow τ_S to be piece-wise where $\tau_S = 0$ until a means of removal is acquired, thus $\tau_S = constant$ at junction $t = \bar{t}$. An interesting and more robust approach would be to allow our control parameters to vary with respect to time, i.e., $\tau_s = \tau_S(t), \tau_I = \tau_I(t)$. We can broaden our assumptions by allowing to different treatment classes to tackle susceptible and infectious computers separately. Intuitively, this seems viable since susceptible computers may just need an anti-virus update which is much faster than diagnosing and fixing an infectious computer. As a final thought, allowing the model to cover a multi-viral outbreak would provide some interesting insight into a more refined control strategy, this implemented by simply allowing computers with some new anti-virus program to be susceptible to other viruses, i.e., *Antidotal → Susceptible*. With more observational research, we hope to condition our model to be more robust.

REFERENCES

[1] Langner, R. (2011, March). Ralph Langner: Cracking Stuxnet, a 21st-century cyber weapon. [Video Link]. Retrieved from
http://www.ted.com/talks/ralph_langner_cracking_stuxnet_a_21st_century_cyberweapon

[2] Kirk, J. (2015, February 17). Destroying your hard drive is the only way to stop this super-advanced malware [Online Article] Retrieved from
http://www.pcworld.com/article/2884952/equation-cyberspies-use-unrivaled-nsastyle-techniques-to-hit-iran-russia.html

[3] W.O. Kermack, A.G. McKendrick: A contribution to the mathematical theory of epidemics. Proc. R. Soc. Lond. B Biol. Sci., **115**, (1927) 700-721.

[4] O. Diekmann, J.A.P. Heesterbeek, J.A.J. Metz, *On the definition and the competition of the basic reproduction ratio $\mathcal{R}_0$ in models for infectious diseases in heterogeneous populations*, J. Math. Biol. 28(1990) 365-382.

[5] P. van den Driessche, J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Biosci. 180(2002) 29-48.

[6] F. Brauer, P. van den Driessche, J. Wu (Eds), Mathematical Epidemiology, Lecture Notes in Mathematics, Vol. 1945, Springer, Berlin, 2008.

[7] Araujo VO. Modelagem Dinâmica de Vírus de Computador, Under-Graduate Engineering Thesis, Escola Politécnica da USP, São Paulo-Brasil; 2004.

[8] Blanchard, Paul, Robert L. Devaney, and Glen R. Hall. "Existence and Uniqueness for Systems." Differential Equations. 4th ed. Pacific Grove, CA: Brooks/Cole Pub., 2011.

[9] Blanchard, Paul, Robert L. Devaney, and Glen R. Hall. "Phase Portraits for Linear Systems with Real Eigenvalues." Differential Equations. 4th ed. Pacific Grove, CA: Brooks/Cole Pub., 2011.

[10] Forsythe, G. E., Malcolm, M. A., Moler, C. B., Malcolm, M. A., and Moler, C. B.. *Computer methods for mathematical computations*. Englewood Cliffs, N.J. : Prentice-Hall, 1977.

[11] Kahaner, D., Moler, C. B., Nash, S., Forsythe, G. E., and Nash, S.. *Numerical methods and software*. Englewood Cliffs, NJ : Prentice Hall, 1988.