Dynamics of Targeted Treatment

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Abstract

An SIS epidemiological household model is studied to understand the dynamics of targeted treatment. The household model splits the population into households, which may, for example, represent patches within a landscape or dorms within a school. Interactions between households typically occur at a much lower rate than intra-household interactions. In an agricultural setting, households are crop fields that can be infected with insect pests. Plants will recover when insecticide is applied and the insects on them are killed. Rather than using a fixed per-capita recovery or treatment rate, an individual's treatment rate will be a function of the infection level in that individual's household. This allows for targeted treatment directed towards households with larger infections. A model is developed and a moment-closure approximation approach is used to truncate the resulting infinite system of differential equations. Numerical results from the truncated system are computed and compared to stochastic simulations. It was found that targeted treatment does not change the endemic equilibrium when the population-wide treatment rate is controlled. Surprisingly, targeted treatment decreases the amount of time it takes to reach the steady state, which could be detrimental during an epidemic.

Introduction

The United States uses over 1.3 billion pounds of insecticide annually to protect its agricultural crops. Despite this, crop loss due to insects in the United States is estimated to be about two billion dollars per year [9]. Though insecticide use is currently debated, the past century's impressive crop yields owe much to the liberal use of insecticide. Humans have used insecticides for hundreds of years, but it was not until the twentieth century that cheap and effective chemical insecticides became available [5]. With these new weapons at their disposal, crop yields skyrocketed as farmers were able to effectively neutralize the damage caused by insects. Insecticides were introduced at the same time as a number of other new farming techniques including chemical fertilizers, mono-cropping and high yield grain breeds. This combined occurrence caused the "Green Revolution," the dramatic increase in world food production between 1940 and 1980; it is now possible to produce enough food to feed every human being [14].

After decades of using artificial insecticides, many insects have gained immunity from once-effective insecticides. One example is the Colorado Potato Beetle, which was effectively controlled by DDT until the the species gained immunity in the 1950's. Once DDT was no longer effective, other insecticides were used but the beetle continued to develop an immunity to each one [13]. Despite the increasing ineffectiveness of insecticides, many have argued that the insect population explosion that would result from a nation-wide ban of insecticides in the United States would lead to higher food prices, food shortages and tremendous economic damage [9, 5]. In fact, since the mass introduction of insecticides in the 1940s, crop damage due to insects has doubled [9], but commercial agriculture has evolved to the point where the application of insecticides is no longer a luxury, but a necessity.

Due to its obvious agricultural, economic, and environmental importance, the application of insecticides has been modeled by a number of researchers [11, 7]. The objective of much of this work is to maximize the effectiveness of the insecticide and minimize the cost and environmental impact. In [11, 7] crop damage caused by insects and the cost of insecticide application are modeled via a system of differential equations. As was stated in [7] one of the difficulties with modeling the effects of insecticide is that many factors, like geography and weather, may change the outcome of

a spraying.

The inherent spatial nature of any agricultural system has prompted us to consider a household model to explore the dynamics of insecticide application [8, 12, 10]. A household model groups individuals into households, which could be considered towns, dormitories, or, for this model, agricultural fields. Interaction happens mostly on the local level (within a single field) and less often between fields or between farms.

Using this framework, our paper specifically studies the dynamics of targeted treatment. Rather than spreading out a supply of insecticide throughout the entire farm, a targeting strategy seeks to define a more efficient allocation of resources. This is analogous to other studies on the effects of patch spraying as applies to herbicide applications [15] and of targeting vaccinations using SIS, SIR, and SIRS household models [1, 2].

The rest of the paper is organized as follows: in Section 1 the equations for individual households are stated and explained. From the household equations an infinite system of differential equations are derived which describe the total proportion of infected individuals across the entire population. In Section 2 the system is analyzed. Equilibrium points and their stability are derived. The infinite system of differential equations is approximated using a moment-closure. Attempting to numerically solve the truncated system led to divergent and/or negative systems which are nonsensical in this context. A reparameterization is used to force the system to stay within the correct bounds. The setup of our stochastic simulation of the system is explained. In Section 3 the results from the stochastic simulations of the model with and without targeted treatment are compared. The numerical solutions of our truncated system of equations are then compared to the simulation results. In the conclusion we discuss the implications of our results and possible directions for future work.

1 Building the Household Model

Our model is a modification of the model developed by Hiebeler [8], a continuous time SIS household model. The model splits the population into n_2 households each with n_1 individuals. The population within a household is homogeneous, and the rate at which the individuals within a household

interact is higher than the rate at which individuals in different households interact. Hiebeler's model assumes a constant per-capita recovery rate of infected individuals. To understand the effects of targeted treatment, the per-capita recovery within any given household will be changed to a linearly increasing function of the number of infected individuals within that household.

In our model, each household represents a field within a farm. The individuals will be single plants of a given crop. Plants become infected when they are infested by a crop pest that feeds on them. The plants return to the susceptible class when they are sprayed with pesticide and the pests are killed. It is assumed that the pests are killed before irreparable harm is done to the plants. This fits within the framework of the standard SIS model, since plants can move back and forth between the susceptible and infected states, depending on the presence or absence of the crop pest. As stated above, the per-capita recovery rate in each household is dependent on the number of infected individuals in that household. This is a continuous approximation of threshold spraying, spraying only when the pest density has passed a certain critical value. For example, University of Maine researchers suggest a form of targeted insecticide application to help combat the blueberry maggot fly, the chief pest of Maine's blueberry farms. To implement the strategy, fields are split into sections, each of which is monitored for pest density. Once the insect population within a certain plot passes a critical threshold, insecticide is applied to that section [6].

1.1 The Household Equations

Let I_k be the proportion of infected individuals in the kth household. Each of the households will be described by the following equation:

$$\frac{dI_k}{dt} = \underbrace{I_k\phi(1-\alpha)(1-I_k)}_{\text{intra-household infections}} + \underbrace{E[I]\phi\alpha(1-I_k)}_{\text{inter-household infections}} - \underbrace{\frac{\mu I_k^2}{Q}}_{\text{targeted treatment}} \tag{1}$$

for $k = 1, 2, ..., n_2$, where ϕ is the rate of infection, $0 \leq \alpha \leq 1$ is the probability that a given infection event occurs outside of the household, and μ is the rate of recovery. Note that when $\alpha = 1$ the entire population behaves as a globally-mixed system. $E[I]$ is the expected value of the

I_k's: $E[I] = \frac{1}{n_2}\sum_{k=1}^{n_2} I_k$. It can be interpreted as the probability that a randomly chosen individual from the entire population is infected or as the expected proportion of individuals infected within a randomly-chosen household. It should be noted that $E[I]$ is the expected value *taken over the households*. $Q = \frac{E[I^2]}{E[I]}$ is a normalization constant which allows us to compare our model to the original model without targeted treatment. In the original model, each infectious individual was treated at fixed rate μ. Because household k has $n_1 I_k$ infectious individuals, the total rate of treatment within household k was therefore $\mu n_1 I_k$, and the total treatment rate of all infectious individuals in the entire population was then

$$\sum_{k=1}^{n_2} \mu n_1 I_k = \mu n_1 n_2 E[I]. \tag{2}$$

For the model with targeted treatment, a per-capita treatment rate for an individual within household k of the form $f(I_k) = \frac{\mu I_k}{C}$ was chosen, which is a linear function of the proportion of individuals in the current household. The constant C was chosen so that the total treatment rate in the entire population would still be $n_1 n_2 E[I]$. Using the new per-capita rate, the population-wide total rate is

$$\sum_{k=1}^{n_2} f(I_k) n_1 I_k = \sum_{k=1}^{n_2} \frac{\mu n_1 {I_k}^2}{C} = \frac{\mu n_1 n_2}{C} E[I^2].$$

In order for this latter quantity to equal the value in equation (2) so that the model with targeted treatment has the same population-wide treatment rate as the previous model with constant per-capita treatment rate, we require $C = \frac{E[I^2]}{E[I]} = Q$.

1.2 Global Behavior: The Moment Equations

In addition to studying the individual households, we are interested in the global behavior of the epidemic. To do this we derive a differential equation for the proportion of infected individuals

across the entire population, $E[I]$. To derive $\frac{dE[I]}{dt}$ we use the definition of expected value:

$$\begin{aligned}\frac{dE[I]}{dt} &= \frac{d}{dt}\left(\frac{1}{n_2}\sum_{k=1}^{n_2} I_k\right) \\ &= \frac{1}{n_2}\sum_{k=1}^{n_2}\frac{dI_k}{dt} \\ &= (\phi - E[I]\phi\alpha)E[I] - \left(\frac{u}{Q} + \phi(1-\alpha)\right)E[I^2]\end{aligned} \tag{3}$$

This equation depends on the second moment of I, $E[I^2]$, which is the probability that two individuals selected within a given household are infected. In general the wth moment of I, $E[I^w]$, is the probability that w individuals picked (with replacement) from a single household are all infected. In fact, the rate of change of any moment of I will depend on other moments, thus we derive the equation for the wth moment of I:

$$\begin{aligned}\frac{dE[I^w]}{dt} &= \frac{d}{dt}\left(\frac{1}{n_2}\sum_{k=1}^{n_2} I_k^w\right) \\ &= \frac{1}{n_2}\sum_{k=1}^{n_2} wI_k^{w-1}\frac{dI_k}{dt} \\ &= w\left[(\phi(1-\alpha) - E[I]\phi\alpha)E[I^w] - \left(\frac{u}{Q} + \phi(1-\alpha)\right)E[I^{w+1}] + \phi\alpha E[I]E[I^{w-1}]\right].\end{aligned} \tag{4}$$

This gives us an infinite system of ordinary differential equations describing the moments of I. Note that because we are controlling the population-wide recovery rate, equation (3) above is the same as in the model with fixed per-capita treatment rate [8]. However, equations for the higher moments differ from the original model, leading to different dynamics for $E[I]$. For further discussion of the differences in the moment equations of the two models, see Appendix D.

2 Analysis

2.1 Deriving the Equilibria

To find the endemic equilibrium, we use (1) to get the equations for any two households j and k, set them equal to 0 and solve for $\frac{\mu}{Q}$

$$\begin{aligned}\frac{\mu}{Q} &= \frac{I_j\phi(1-\alpha)(1-I_j)+\phi\alpha E[I](1-I_j)}{I_j^2}\\ \frac{\mu}{Q} &= \frac{I_k\phi(1-\alpha)(1-I_k)+\phi\alpha E[I](1-I_k)}{I_k^2}.\end{aligned}$$

Setting these two equations equal, cross-multiplying, and combining like terms gives

$$0=\phi(1-\alpha)\left[I_jI_k^2(1-I_j)-I_kI_j^2(1-I_k))\right]+\phi\alpha E[I]\left[I_k^2(1-I_j)-I_j^2(1-I_k)\right].$$

which we can factor as

$$0=(I_k-I_j)\left[\phi(1-\alpha)I_jI_k+\phi\alpha E[I](I_k+I_j-I_kI_j)\right].$$

Thus there exists at least one equilibrium where $I_k=I_j$ for all $k=1,2,...n_2$ and $j=1,2,...n_2$, implying that there exists at least one equilibrium where all households tend towards the same infected density. This equality can be substituted into any of the n_2 household equations to solve for the equilibria in which all households have equal infected populations.

$$\begin{aligned}0 &= I_j\phi(1-\alpha)(1-I_j)+\phi\alpha E[I](1-I_j)-\frac{\mu I_j^2}{Q}\\ 0 &= I_j\phi(1-\alpha)(1-I_j)+\phi\alpha\frac{1}{n_2}\sum_{i=1}^{n}I_i(1-I_j)-\frac{\mu I_j^2\sum_{i=1}^{n}I_i}{\sum_{i=1}^{n}I_i^2}\\ 0 &= I_j\phi(1-\alpha)(1-I_j)+\phi\alpha I_j(1-I_j)-\frac{\mu I_j^2I_j}{I_j^2}.\end{aligned}$$

Ignoring the trivial equilibrium, $I_j = 0$, this reduces to

$$I_j = 1 - \frac{\mu}{\phi}.$$

A proof that the above endemic equilibrium is the unique endemic equilibrium can be found in Appendix C. Using the value for I_k we get the endemic equilibrium for the moments of I

$$E[I^w] = \left(1 - \frac{\mu}{\phi}\right)^w$$

when $\mu < \phi$. It is easy to verify that this satisfies the equation for the w^{th} moment. Interestingly, α has no effect on the long-term endemic equilibrium, implying that the household model tends towards the mean-field endemic equilibrium. The mean-field assumption states that space does not matter, each household has an equal proportion of infected individuals. In other words, $E[I^w] = (E[I])^w$. So despite the inherent spatial nature of our model, the endemic equilibrium is the same as the mean-field equilibrium where space is not important. Also, the endemic equilibrium for the model with targeted treatment is the same as the model without targeted treatment. So targeting treatment has no effect on the proportion of infected individuals as $t \to \infty$.

2.2 Stability of Equilibria

2.2.1 Stability of Disease-Free Equilibrium

Due to Q, evaluating the Jacobian for the household equations (1) is difficult. To help make this easier to analyze we first scale time by the sum of the squares of the household infection levels

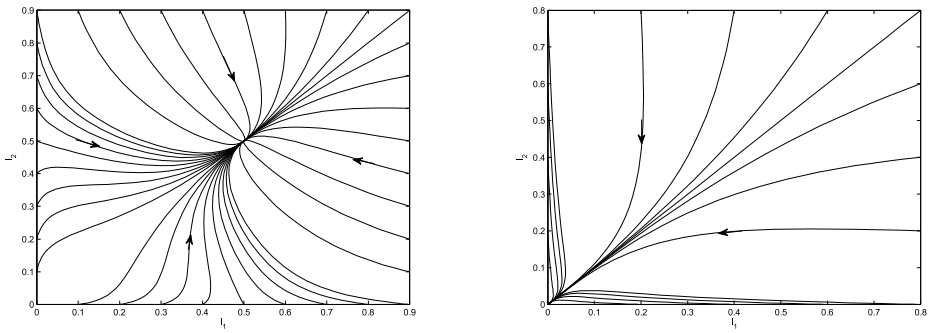
$$\frac{dt}{\sum_{i=1}^{n_2} I_i^2} = d\tau. \tag{5}$$

The equations then become

$$\frac{dI_k}{d\tau} = \phi(1-\alpha)I_k\sum_{i=1}^{n_2} I_i^2 + \frac{\phi\alpha}{n_2}\sum_{i=1}^{n_2} I_i\sum_{j=1}^{n_2} I_j^2 - \mu I_k^2\sum_{i=1}^{n_2} I_i \tag{6}$$

$$- \phi(1-\alpha)I_k^2\sum_{i=1}^{n_2} I_i^2 - \frac{\phi\alpha}{n_2}I_k\sum_{i=1}^{n_2} I_i\sum_{i=1}^{n_2} I_i^2. \tag{7}$$

From the transformed system it is clear that $I_1 = I_2 = ...I_{n_2} = 0$, the disease-free equilibrium, is an equilibrium point of our system that exists for all parameter values. We are unable to evaluate the stability of this equilibrium using standard linearization methods since the Jacobian of (6) evaluated at the disease-free equilibrium results in the zero matrix. Therefore this point is non-hyperbolic; the Jacobian has at least one eigenvalue equal to zero. In fact, all eigenvalues at this point are zero. For $n_2 = 2, 3$ we can analyze the stability of the disease-free equilibrium numerically using phase-plane portraits. For the phase portraits of a two household system, see figure 1. We have omitted the portraits for $n_2 = 3$, as they are very similar to the portraits for the $n_2 = 2$ case. For more analysis of the disease-free equilibrium see Appendix B.



(a) (b)

Figure 1: Trajectories for a two-household system are shown for two different parameter settings, and for a variety of initial conditions. (a) Initial conditions had either I_1 or I_2 equal to 0 or 0.9, with the other varying between 0 and 0.9, with parameter values $\phi = 2, \mu = 1$, and $\alpha = 0.1$. All trajectories approach the endemic equilibrium $I_1^* = I_2^* = 0.5$. (a) Initial conditions had either I_1 or I_2 equal to 0 or 0.8, with the other varying between 0 and 0.8, with parameter values $\phi = 1, \mu = 2$, and $\alpha = 0.1$. All trajectories approach the disease-free equilibrium $I_1^* = I_2^* = 0$.

2.2.2 Stability of the Endemic Equilibrium

To analyze the endemic equilibrium, we applied the blowing up transformation [4, 3] to (6)

$$\begin{aligned} I_1 &= I_1 \\ I_2 &= V_2 I_1 \\ &\vdots \\ I_k &= V_k I_1 \\ &\vdots \\ I_{n_2} &= V_{n_2} I_1 \end{aligned}$$

This transformation is one to one for all $I_1 > 0,\ I_k \geq 0$ for all $k = 2, 3...n_2$. Therefore we can analyze all relevant equilibria except the disease-free using this system. Now for each V_k, we solve for $\frac{dV_k}{dI_k}$. Using the definition of the above transformation and the product rule

$$\frac{dI_k}{dI_1} = \frac{\frac{dI_k}{d\tau}}{\frac{dI_1}{d\tau}} = \frac{dV_k}{dI_1} I_1 + V_k$$

from which we get

$$\frac{dV_k}{dI_1} = \frac{-V_k(\frac{dI_1}{d\tau}) + \frac{dI_k}{d\tau}}{I_1(\frac{dI_1}{d\tau})}.$$

Using (6) and letting $V_1 = 1$ it directly follows

$$\begin{aligned} V_k' &= (1 - V_k)\left[I_1 \left(\phi(1-\alpha) V_k \sum_{i=1}^{n_2} V_i^2 \right) + \frac{\phi\alpha}{n_2} \sum_{i=1}^{n_2} V_i \sum_{i=1}^{n_2} V_i^2 + \mu V_k \sum_{i=1}^{n_2} V_i \right] \\ I_1' &= I_1 \left[(1 - I_1) \sum_{i=1}^{n_2} V_i^2 \left(\phi(1-\alpha) + \frac{\phi\alpha}{n_2} \sum_{i=1}^{n_2} V_i \right) - \mu \sum_{i=1}^{n_2} V_i \right]. \end{aligned}$$

The Jacobian of this system evaluated at the endemic equilibrium is

$$J = \begin{bmatrix} n(-\phi+\mu) & \frac{\mu}{\phi}(1-\frac{\mu}{\phi})(\phi+2\phi\alpha-\mu) & \frac{\mu}{\phi}(1-\frac{\mu}{\phi})(\phi+2\phi\alpha-\mu) & \dots & \frac{\mu}{\phi}(1-\frac{\mu}{\phi})(\phi+2\phi\alpha-\mu) \\ 0 & -n(\phi+\alpha\mu) & 0 & & 0 \\ 0 & 0 & -n(\phi+\alpha\mu) & & 0 \\ \vdots & & & \ddots & \vdots \\ 0 & 0 & 0 & 0 & -n(\phi+\alpha\mu) \end{bmatrix}$$

and since it is upper triangle, has eigenvalues of $\lambda_1 = n(-\phi+\mu), \lambda_2 = n(-\phi+\mu), ..., \lambda_{n_2} = n(-\phi+\mu)$. Therefore we have proven the following:

Proposition 1. *The endemic equilibrium is asymptotically stable if and only if $\mu < \phi$.*

2.3 Moment-Closure Approximation

To learn more about our model, we studied the infinite system of differential equations describing the moments of I. But integrating an infinite systems of differential equations is generally a daunting task. It is often necessary to use approximations to truncate at some order w. We will use the conditional closure approximation from [8]. The conditional closure approximates the wth moment using the $w-1$ and $w-2$ moments.

$$E[I^w] \approx \frac{(E[I^{w-1}])^2}{E[I^{w-2}]}$$

To derive this approximation, we first break $E[I^w]$ into two pieces

$$\begin{aligned} E[I^w] &= P(\text{first } w-1 \text{ individuals infected}) * P(\text{last individual infected}|\text{first } w-1 \text{ individuals infected}) \\ &\approx P(\text{first } w-1 \text{ individuals infected}) * P(\text{last individual infected}|w-2 \text{ before } w^{\text{th}} \text{ are infected}) \\ &= E[I^{w-1}] * \frac{E[I^{w-1}]}{E[I^{w-2}]} \\ &= \frac{(E[I^{w-1}])^2}{E[I^{w-2}]}. \end{aligned}$$

So we are assuming that the state of the last infected individual does not depend on the first selected individual, but still depends on the $w-2$ individuals selected ahead of them.

We use this closure to truncate our infinite system to three equation and approximate $E[I]$. In the paper on which this work was based [8], the approximation was sometimes done using two equations. We are unable to do that here; truncating at two equations with the conditional closure approximation reduces the targeted treatment system to the original model with constant per-capita recovery. Therefore we must use at least three equations to explore differences in dynamics between the two models.

2.4 Numerical Analysis

As stated above, the conditional closure approximation allows us to reduce our infinite system to a system with three equations. MATLAB's `ode45` function was first used to attempt to numerically solve the system of three differential equations. However, the resulting solutions included negative and/or divergent values for the various moments of I. It was speculated that this was in part due to the vastly different magnitudes of the state variables $E[I]$, $E[I^2]$, and $E[I^3]$ in the three equations. We therefore studied a modified system of three equations, involving the derivatives of $E[I]$, $E[I^2]/E[I]$, and $E[I^3]/E[I^2]$, which were more likely to have similar magnitudes. However, this change did not resolve the problem. The problem was then assumed to be due to inherent instabilities in the numerical methods used. Therefore the higher moments were reparameterized in such a way that they were unable to diverge or become negative. This reparameterization was developed by Hiebeler in [8] to deal with the same type of ill-behaved system.

To start, we use Hölder's Inequality

$$E[|XY|] \leq E[|X^p|]^{\frac{1}{p}} E[|Y^q|]^{\frac{1}{q}} \quad \text{where } \frac{1}{p} + \frac{1}{q} = 1. \tag{8}$$

We can neglect the absolute values, as all of our random variables are non-negative. Substituting in the values of $p = w, q = \frac{w}{w-1}$ with $X = 1$ and $Y = I^{w-1}$ yield

$$(E[I^{w-1}])^{\frac{w}{w-1}} \leq E[I^w]. \tag{9}$$

We also use the fact that all expected values are taken over proportions:

$$\begin{aligned} \sum_{k=1}^{n_2} I_k^w &\leq \sum_{k=1}^{n_2} I_k^{w-1} \\ \frac{1}{n_2}\sum_{k=1}^{n_2} I_k^w &\leq \frac{1}{n_2}\sum_{k=1}^{n_2} I_k^{w-1} \\ E[I^w] &\leq E[I^{w-1}]. \end{aligned}$$

This gives us bounds on $E[I^w]$, so it can be written as

$$E[I^w] = E[I^{w-1}] - \frac{E[I^{w-1}] - (E[I^{w-1}])^{\frac{w}{w-1}}}{1 + g(\beta_w)}. \tag{10}$$

where

$$g(\beta_w) = \begin{cases} \beta_w^2, & \text{if } \beta_w \geq 0 \\ 0, & \text{otherwise} \end{cases}$$

Therefore when $\beta_w = 0$, $E[I^w] = (E[I^{w-1}])^{\frac{w}{w-1}}$ and when $\beta_w \to \infty$, $E[I^w] \to E[I^{w-1}]$. We can solve for β_w using the above equation for $E[I^w]$

$$\beta_w = \sqrt{\frac{E[I^w] - (E[I^{w-1}])^{\frac{w}{w-1}}}{E[I^{w-1}] - E[I^w]}}.$$

We can now differentiate to obtain a differential equation for β_w

$$\begin{aligned} \frac{d\beta_w}{dt} &= \frac{1}{2\beta_w(E[I^{w-1}] - E[I^w])^2}\Big(\frac{dE[I^w]}{dt}E[I^{w-1}](1 - E[I^{w-1}]^{\frac{1}{w-1}}) \\ &+ \frac{dE[I^{w-1}]}{dt}(E[I^{w-1}]^{\frac{1}{w-1}}\Big(\frac{w}{w-1}E[I^w] - \frac{1}{w-1}E[I^{w-1}] - E[I^w]\Big)\Big)\Big). \end{aligned}$$

$\frac{dE[I]}{dt}$, $\frac{dE[I^2]}{dt}$, $\frac{dE[I^3]}{dt}$ can now be rewritten, using (10) and (4), as a function of $E[I]$, β_2, and β_3. With (10), $\frac{d\beta_2}{dt}$ can be rewritten in terms of β_2, $E[I]$, and $E[I^2]$. Using (10) and the moment closure approximation, $\frac{d\beta_3}{dt}$ can be rewritten in terms of β_3, $E[I^2]$, and $E[I^3]$. Therefore we have a closed

system in $E[I]$, β_2, and β_3 which can be solved numerically. This system does *not* give completely accurate values for $E[I]$. If more accuracy is desired, the process done here can be generalized to a truncation at the w^{th} moment.

2.5 Stochastic Simulations

Stochastic simulations were run for the model both with and without targeted treatment. Both models were run using the same parameters. We used these simulations to evaluate the numerical results from our moment closed system. In order to reduce stochastic variability, all simulations results were averaged over twenty runs. A table displaying the parameter values used in our simulations is shown below.

Parameter	Value used for simulations
n_1	200
n_2	200
h_0	5
c_0	6
ϕ	2
μ	1
α	.001, .01, .05, .1, 1
maxtime	60

It should be noted that maxtime = 300 was used for $\alpha = 0.001$, as it took much longer for the system to reach equilibrium in that case. h_0 and c_0 are the initial number of infected households and the initial number of infected individuals in each infected households, respectively.

As can be seen above, ϕ and μ were constant throughout all of the simulations. We assumed $\mu < \phi$ as the disease-free equilibrium is stable otherwise, a situation we were not interested in.

The code for the simulations can be found in Section A of the Appendix.

3 Results

3.1 Numerical Solutions

By truncation and reparameterization we were able to numerically solve our system using MATLAB's `ode45` function. The system was solved numerically and plotted against the average of 20 stochastic simulations, all with matching parameters and initial conditions. Figures (2) and (3) are graphs of the system with $\alpha = 0.1$ and $\alpha = 0.01$ respectively.

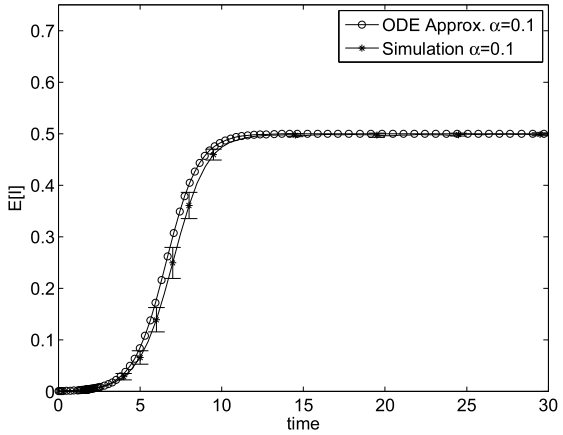


Figure 2: Comparing the Results of Numerical Solution and Stochastic Simulation for $\phi = 2$, $\mu = 1$, and $\alpha = 0.1$. Error bars show ± 1 Standard Error of $E[I]$.

In Figure (4), time taken to reach 5% of equilibrium for both the numerical solution and the simulations is plotted for varying α. For α sufficiently large, the system truncated by the conditional closure does an excellent job of predicting the simulated results. For smaller α, the truncated system is not a good predictor of the stochastic simulation. The reason for which we can get from Hölder's inequality (8).

Let $X = I^{w/2}$, $Y = I^{w/2-1}$, $p = 2$, and $q = 2$. So $XY = I^{w-1}$, $X^p = I^w$, and $Y^q = I^{w-2}$. Using Hölder's inequality,

$$\begin{aligned} E(I^{w-1}) &\leq (E[I^w])^{1/2}(E[I^{w-2}])^{1/2} \\ (E[I^{w-1}])^2 &\leq E[I^w]E[I^{w-2}] \end{aligned}$$

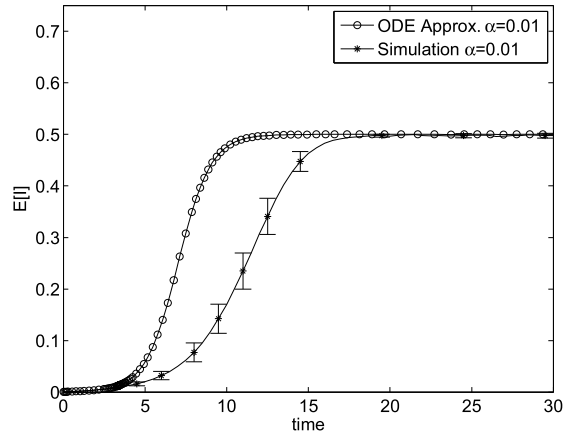


Figure 3: Comparing the Results of Numerical Solution and Stochastic Simulation for $\phi = 2$, $\mu = 1$, and $\alpha = 0.01$. Error bars show ± 1 Standard Error of $E[I]$.

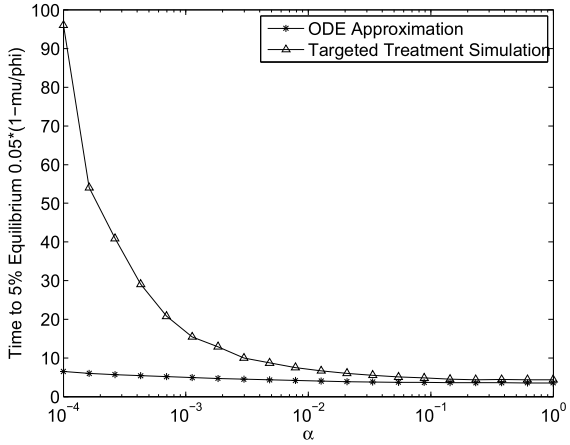


Figure 4: Comparing time to reach 5% of equilibrium for numerical solutions and stochastic simulations of targeted treatment. Clearly as α becomes smaller, the numerical solutions do not predict the dramatic increase in time need to reach 5% of equilibrium predicted by the simulations.

which gives us

$$\frac{(E[I^{w-1}])^2}{E[I^{w-2}]} \leq E[I^w]. \tag{11}$$

This shows that conditional closure underestimates $E[I^w]$, which means our three equation system truncated by the conditional closure is underestimating $E[I^4]$. But a multiple of $E[I^4]$ is subtracted

from the equation for $\frac{dE[I^3]}{dt}$, thus the conditional closure is subtracting *too little* from $\frac{dE[I^3]}{dt}$. So $E[I^3]$ under conditional closure is growing too quickly. But, by the same logic, $E[I^2]$ is now growing too slowly as a multiple of $E[I^3]$ that is growing too quickly is being subtracted from $\frac{dE[I^2]}{dt}$. So $E[I^2]$ under the conditional closure is growing too slowly. Applying the same logic again we see that $\frac{dE[I]}{dt}$ has a too-small multiple of $E[I^2]$ being subtracted from it, thus $E[I]$ is growing faster than it should, which is the behavior that we observe.

The higher moments are a measure of clustering, which occurs at a higher rate for smaller α. But the moment that has the most impact on the magnitude of $E[I]$, $E[I^2]$, is underestimated by the conditional closure approximation. For small enough α the truncated system does not place enough importance on clustering and consequently grows too quickly.

3.2 Simulation Results

Due to the inaccuracy of the numerical solutions of the truncated system we used simulation results to compare the differences between the original model and the model with targeted treatment. The simulations were used to understand the effects of varying α. Though α has no effect on the steady-state, it does affect the speed at which the equilibrium is approached. We first compare the model with and without targeted treatment for varying α.

Figure 5 and 6 show that, for both models, time to equilibrium is significantly increased for smaller and smaller values of α. The largest difference between the two models is clear: targeted treatment approaches equilibrium noticeably faster for all $\alpha < 1$. When $\alpha = 1$, which is shown in Figure 7, there no noticeable difference between the original model and the targeted treatment model.

When $\alpha < 1$ the model with targeted treatment reaches equilibrium faster than the model with constant per-capita treatment. To further exhibit this difference, simulations of the two models for the same α are plotted on the same axes, which can be seen in Figures 8, 9, and 10. To summarize the difference between the two models, Figure 11 shows the time to 5% of equilibrium for both models for varying values of α.

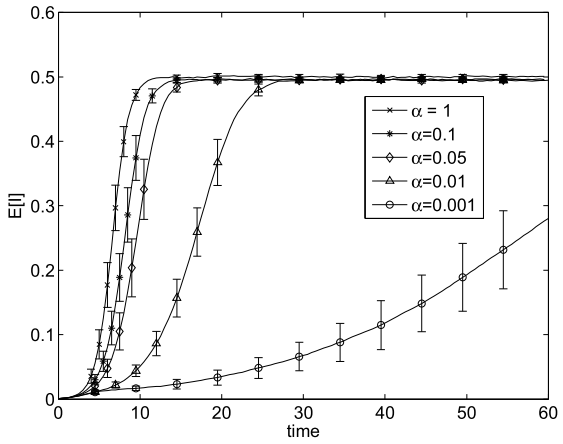


Figure 5: Each curve is the average of 20 simulation runs with $\alpha =$ values of $1, 0.1, 0.05, 0.01, 0.001$ and with constant per-capita recovery. This is a reproduction of the simulation results in [8]. Runs with $\alpha = 0.001$ leveled off after approximately 120 timesteps. Error bars show ± 1 Standard Error of $E[I]$.

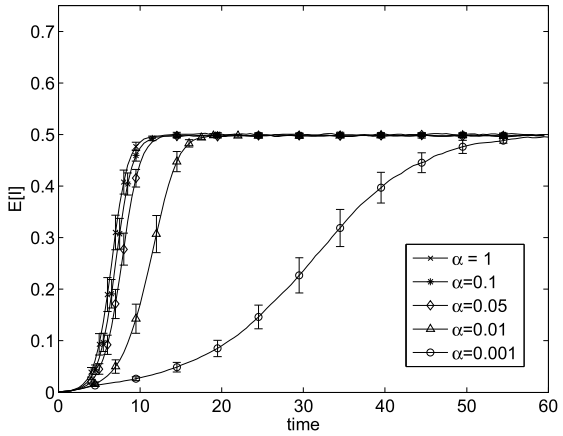


Figure 6: Each curve is the average of 20 simulation runs with α values of $1, 0.1, 0.05, 0.01, 0.001$ and with targeted treatment. Runs with $\alpha = 0.001$ leveled off after approximately 60 timesteps. Error bars show ± 1 Standard Error of $E[I]$.

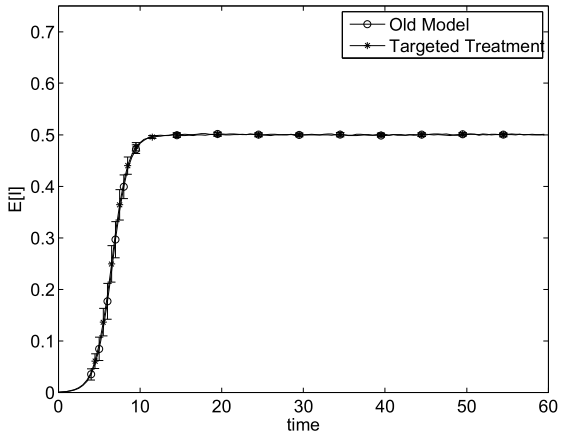


Figure 7: Constant Per-Capita Recovery and Targeted Treatment Models with $\alpha = 1$. Error bars show ± 1 Standard Error of $E[I]$.

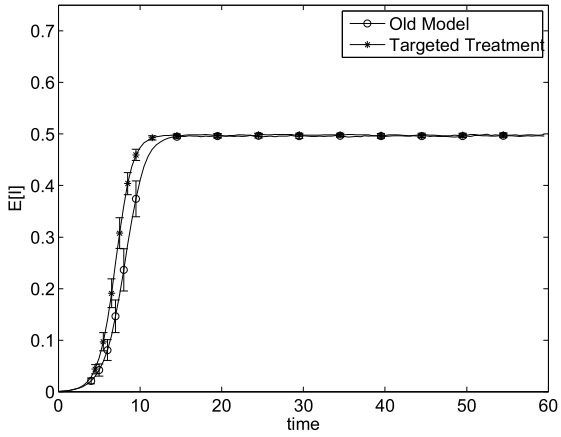


Figure 8: Constant Per-Capita Recovery and Targeted Treatment Models with $\alpha = 0.1$. Error bars show ± 1 Standard Error of $E[I]$.

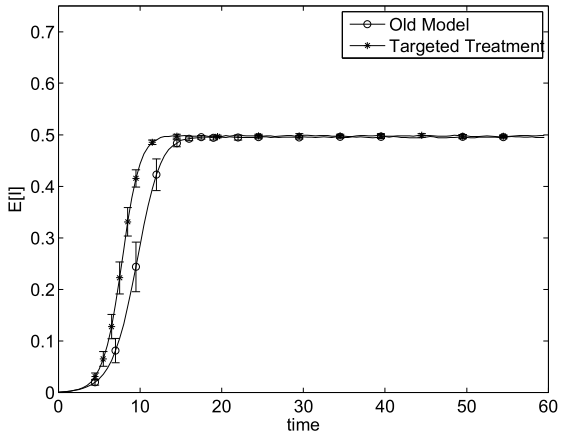


Figure 9: Constant Per-Capita Recovery and Targeted Treatment Models with $\alpha = 0.05$. Error bars show ± 1 Standard Error of $E[I]$.

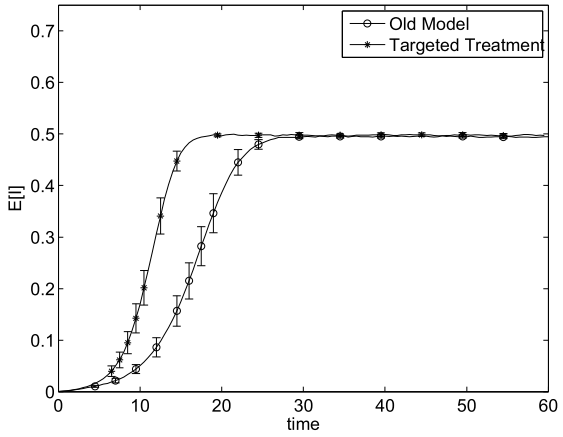


Figure 10: Constant Per-Capita Recovery and Targeted Treatment Models with $\alpha = 0.01$. Error bars show ± 1 Standard Error of $E[I]$.

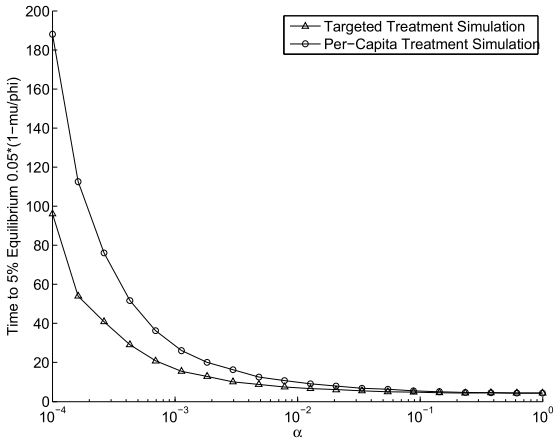


Figure 11: Time needed to reach 5% of equilibrium for model with constant per-capita treatment and with targeted treatment.

4 Conclusions

The lesson we learn from this model is simple: targeted treatment increases the speed at which a population reaches the endemic equilibrium. Since in most cases it would not be desirable to reach an endemic equilibrium quickly, targeted treatment is actually a harmful strategy. There are several ways to intuitively understand why targeted treatment decreases time to equilibrium. First, the targeted treatment reduces variance between households more quickly than the constant per-capita model. Households with higher infected densities are hit hardest, but as this happens the houses with lower infected densities are allowed to grow. Thus the system tends quickly toward the mean-field equilibrium, which has no variance. Secondly, assuming relatively small values of α, most infection attempts in a household with a high infected density are wasted. If most contacts are intra-household, then high infected households will often have infected-infected interactions, resulting in no change in the infected population. But in the households with lower infected densities, most infection attempts result in a new infected individual. Therefore targeting households with higher infected densities ignores the spreaders that can do the most damage.

The other interesting product of this research is the development of a spatial recovery term. With the simple function we explored, we found the process of recovery can impede the dynamics that α creates in the system. Not only are the simulations and numerical solutions predicting that targeted treatment will push a population to equilibrium faster, they also increased show localized contact (i.e. small values of α) cannot slow the time to equilibrium in the targeted treatment system as much as it did in the original model with constant per-capita recovery.

The application to our general insecticide model is clear. Targeted spraying of insecticide on fields with the highest insect pest density could actually serve to decrease farmers' yields. To better understand the the dynamics of this type of spraying strategy, several modifications should be studied. Insecticide offers a period of immunity to the plants that are sprayed. Our model assumed that the instant the insects were removed from the plant, they could potentially return. Also, it would be interesting to change the model to SIR. In our model, once the insect pest was removed from a plant, it returned to susceptible (healthy). Actually, sometimes a plant will become so damaged by the insects that it will either die or become worthless to the farmer. There are many

ways to expand on this model to better understand the dynamics of insect pest control.

From the results of this work, it is possible to hypothesize that reversing the targeted treatment to attack smaller infections and ignoring large infections could be the best solution for this problem. Using the same logic as above, targeting houses with lower density would in all likelihood increase variance. 'Reverse-targeting' would let households with high proportions of infected individuals continue to grow while stamping out the infections in houses with low proportions of infection. Of course, one need not only consider linear functions. A per-capita treatment rate of $\frac{\mu}{I_k+\delta}$ would target households with lower infected densities, like the strategy described above, but would not completely neglect the households of high infected density. It could also be possible to consider a compound recovery term where infections are treated according to a function, yet infections also recover naturally at some rate.

5 Acknowledgments

We would like to thank David Hiebeler, David Murillo, Young Lee, Tae Do, and Faina Berezovskaya for all of their hard work and time. We would also like to thank Carlos Castillo-Chavez for giving us this opportunity. The MTBI/SUMS Summer Undergraduate Research Program is supported by The National Science Foundation (DMS-0502349), The National Security Agency (DOD-H982300710096), The Sloan Foundation, and Arizona State University.

References

[1] Ball, Frank G. and Lyne, Owen D. "Optimal vaccination policies for stochastic epidemics among a population of households." *Mathematical Biosciences*. 178:333-354. 2002

[2] Becker, Neils G. and Starczak, Dianna N. "Optimal Vaccination Strategies for a Community of Households." *Mathematical Biosciences*. 139:117-132. 1997.

[3] Berezovskaya, F.; Novozhilov, A.; Kazev G. "Population Models with Singular Equilibrium." *Mathematical Biosciences*. 208: 270-299. 2007.

[4] Bruno, A.D. "Power Geometry in Algebraic and Differential Equations." Elsevier: Amsterdam, NY. 2000.

[5] Delaplane, Keith S. "Pesticide Usage in the United States: History, Benefits, Risks, and Trends." *Cooperative Extension Service/The University of Georgia College of Agricultural and Environmental*. Bulletin 1121. 2000.

[6] Drummond, Frank. "Wild Blueberry Fact Sheet." *University of Maine Cooperative Extension*. Fact Sheet 201.

[7] Hall, Darwin C. and Norgaard, Richard B. "On the Timing and Application of Pesticides." *American Journal of Agricultural Economics*. 198-201. 1973.

[8] Hiebeler, David. "Moment Equations and Dynamics of a Household SIS Epidemiological Model." *Bulletin of Mathematical Biology*. 68:1315-1333. 2006.

[9] Pimentel, David; Krummel, John; Gallahan, David; Hough, Judy; Merrill, Alfred; Schreiner, Ilse; Vittum, Pat; Koziol, Fred; Back, Ephraim; Yen, Doreen; Fiance, Sandy. "Benefits and Costs of Pesticide Use in U.S. Food Production." *Bioscience*. 28:772+778-784. 1978.

[10] Rushton, S. and Mautner, A.J. "The Deterministic Model of a Simple Epidemic for More Than One Community." *Biometrika*, 42:126-132. 1955.

[11] Talpaz, Hovav and Borosh, Itshak. "Strategy for Pesticide Use: Frequency and Applications." *American Journal of Agricultural Economics*. 56:769-775. 1974

[12] Watson, R.K. "On an Epidemic in a Stratified Population." *Journal of Applied Probability*. 9:659-666. 1972.

[13] Hare, J. Daniel. "Ecology and Management of the Colorado Potato Beetle." *Annu. Rev. Entomol. 1990.*

[14] Conway, G; Toenniessen G. "Feeding the World in the Twenty-First Century" *Nature, 1999.*

[15] Paice, Day, Rew, Howard. "A stochastic simulation model for evaluating the concept of patch spraying" *Weed Research*. 38(5): 373-388.

A MATLAB Code

```
%SIS Household Simulation with Targeted Treatment
%Filename: targetSim.m
%Written by: Isaac Michaud
%Created on 7-6-07
%Last Edited on 7-13-07
%Description: This matlab script simulates an SIS household epidemic with
%targeted recovery

numSim = 20;
for y=1:numSim
%%% Initalizations %%%

%Population Variables

n1 = 200;
%number of individuals in a household
n2 = 200;
%number of household to simulate
totalPop = n1*n2;
%total population size
Ivec = zeros(n2,1);
%Ivec stores the total number of infected individuals in each household
In1 = 6;
%initial infection per "infected household"
In2 = 5;
%initial infected households

%Model Parameters
phi = 2;
%phi is the per-capita infection rate
mu = 1;
%mu is the per-capita recovery rate
alpha = 0.1;
%alpha is the proportion of inter-household infections
death = mu/(mu+phi);
%death is the probability of a death event

%Time Variables
maxTime = 300;
%maximum time the simulation will run for
currentTime = 0;
%the current or initial time the simulation starts with
maxDt = 0.5;
```

```
% how often to record data

%Data Collection
chunksize = 500;
%Grow EI and et by this number of entries when they are full
EI = zeros(1,chunksize);
%EI vector stores the total population size at each time-event
et = zeros(1,chunksize);
%et vector stores the time of each event
vectorlengths = chunksize;
%vectorlenghts is used to keep track of the lengths of the EI and et
%vectors during the simulation

%Other
mycolors = [0 0 0 ; 0 1 0];
%mycolors is the color scheme for displaying the household image
%[black;green]
counter = 1;
%counter is used to keep track of the number of events
graph = 1;
%set to 1 for graphs, 0 for no graphics



%***Part 1***% Initialize the Infected households

[s,idx] = sort(rand(n2,1));
%create n2 indexes randomly
p = idx(1:In2);
%place randomly selected indexes into a vector


Ivec(p) = In1;
%initialize the selected households with In1 infected individuals
totalI = sum(Ivec);
%totalI is the total population of infected individuals in all households



EI(counter) = totalI;
%initlize the first entry of EI to the total population of infected
%individuals at currentTime
et(counter) = currentTime;
%initlize the first entry of et to currentTime
```

```
%***Part 2***% Simulate the Model

while(currentTime < maxTime)

    lambda = totalI*(phi+mu);
    %lambda is the total rate of the poisson process
    deltaTime = exprnd(1/lambda);
    %calculate the time to the next event
    currentTime = currentTime + deltaTime;
    %increment time by the time it took to get to the next event

%%%*****Code Used To Simulate Original Model*******%%%

    %pIvec = Ivec./totalI;  %Method used to fine random recovery
    %pvec = pIvec;          %Method used choose random infection

%%%*****Code Used To Simulate Targeted Treatment***%%%

    pvec = Ivec./totalI;
    %pvec holds the proportions of the total infected population each
    %household has
    pIvec = (pvec.^2)*(sum(pvec)/sum(pvec.^2));
    %compute the new pIvec that targets higher infection densities

%%%*****End of Modified Code*****%%%

    %%Choose What Event Happens%%

    if ( rand < death )
        %test if the event was a recovery

        house = nvalsrnd(pIvec);
        %selects a household to have an individual recover in; note: this
        %is using a weighted average
        Ivec(house) = Ivec(house) - 1;
        %subtract the infected individual from the household's infected
        %list
        totalI = totalI - 1;
        %subtract the infected individual from the total infected
        %population

    else
```

```
        if ( rand < alpha)
            %test to see if the infection is inter-household

            %select a random household to have the infection targeted at
            targetHouse = floor(rand*n2)+1;

            if( rand < ((n1-Ivec(targetHouse))/n1) )
                %test to see if the infection is successful

            Ivec(targetHouse) = Ivec(targetHouse) + 1;
            %if the infection is successful, increment the appropriate
            %variables
            totalI = totalI + 1;
            end
        else

            house = nvalsrnd(pvec);
            if ( rand < ((n1-Ivec(house))/n1) )
                %test to see if the in household infection was successful
                Ivec(house) = Ivec(house) + 1;
                %if the infection is successful, incremment the appropriate
                %variables
                totalI = totalI + 1;

            end

        end

    end
    if (currentTime - et(counter) > maxDt)
        % time to record data
      [currentTime totalI/totalPop std(Ivec/n1) min(Ivec/n1) max(Ivec/n1)]
      counter = counter + 1;
      %increment the counter because the event has accured
      if (counter == vectorlengths)
          %if there has been enough events to full the data arrays, then
          %augment them to make more space
vectorlengths = vectorlengths + chunksize;
EI = [EI zeros(1,chunksize)];
et = [et zeros(1,chunksize)];

      end
      EI(counter) = totalI;
      %record the current population of infected individuals in the EI
      %vector
```

```
      et(counter) = currentTime;
      %record the current time in the et vector
  if ( graph == 1 )
      imageArray = zeros(n1,n2);
      %construct the image array which will allow the user to see the
      %status of the households after each event
      for k = 1:n2
        imageArray(1:Ivec(k),k) = 1;
      end
      figure(1);
      pcolor(imageArray), shading flat, colormap(mycolors);
      %display the image array
      drawnow
  end
     end
  if ( totalI == 0)
      %breaks Loop if there are no more infected individuals
      break;
      %the simulation does not need to run if there are no infected
      %individuals to infect others
  end

end
et = et(1:counter);
%truncate data vectors to only contain recorded data
EI = EI(1:counter);
save('EI.txt', 'EI', '-ascii','-append');
%export the results to a text file
save('times.txt', 'et', '-ascii','-append');
end
```

B Stability Analysis of Disease Free Equilibrium

Our model is

$$\frac{dE[I]}{dt} = w[(\phi(1-\alpha) - E[I]\phi\alpha)E[I^w] - \left(\frac{u}{Q} + \phi(1-\alpha)\right)E[I^{w+1}] + \phi\alpha E[I]E[I^{w-1}].$$

At the disease free equilibrium (DFE), $Q = \frac{E[I^2]}{E[I]}$ is not defined; however, the term $\frac{\mu}{Q}E[I^{w+1}]$ is

uniformly bounded whenever $E[I] > 0$. To see this, note that

$$\frac{E[I^{w+1}]}{Q} = \frac{E[I]E[I^{w+1}]}{E[I^2]} \leq \frac{E[I]E[I^w]}{E[I^2]}. \tag{12}$$

For $w = 2, 3, \ldots$, the fact that $E[I^w] \leq E[I^2]$ implies that the above quantity is $\leq E[I]$, and therefore ≤ 1. For $w = 1$, the rightmost quantity in (13) by inequality (8) above is $\frac{(E[I])^2}{E[I^2]}$, which is also ≤ 1. Thus $\frac{E[I^{w+1}]}{Q} \leq 1$ for all $w = 1, 2, \ldots$. Therefore, our system of ODEs is well defined, but the DFE is a removable singular point. We show that applying the stability analysis of a nonsingular equilibrium to a singular point results in the wrong conclusion.

We consider three cases which are small deviations from the DFE: (A) $c0/n1 \approx 0$, (B) $h0/n2 \approx 0$, and (C) $c0/n1 \approx 0$ and $h0/n2 \approx 0$. For case (A) and (C) the Jacobian is undefined, while for case (B), the Jacobian matrix evaluated at $h0/n2 \to 0$ is

$$J = \begin{bmatrix} \phi - \mu & -\phi(1-\alpha) & 0 \\ -2\mu\frac{c0}{n1} & 2(\phi(1-\alpha)+\mu) & -2(\phi(1-\alpha)+\mu\frac{n1}{c0}) \\ -3\mu(\frac{c0}{n1})^2 & 3(\phi(1-\alpha)(\frac{c0}{n1})^2 + 2\mu\frac{c0}{n1}) & 3(\phi(1-\alpha)(1-2\frac{c0}{n1}) - 2\mu) \end{bmatrix}$$

.

This Jacobian matrix always has at least one positive eigenvalue, even for the case when the recovery rate is greater than the infection rate. But our numerical solutions and stochastic simulations show that the DFE is asymptotically stable.

In the hope that the positive eigenvalue was only important to a system that considered negative values of $E[I]$, we studied the reparameterized system mentioned in the Numerical Analysis section since this reparameterization forced all values to be positive. The Jacobian for this system of three ODEs is

$$J = \begin{bmatrix} \phi - \mu & -\phi(1-\alpha) & 0 \\ 2\mu\frac{\beta_3}{\beta_2} & 2(\phi(1-\alpha)+\mu\frac{c0}{n1}\frac{\beta_2}{\beta_2^2}) & 2(\phi(1-\alpha)-\mu\frac{c0}{n1\beta_2}) \\ -3\mu(\frac{\beta_3^2}{\beta_2^2})^2 & -3\phi(1-\alpha)\frac{\beta_3^2}{\beta_2^2} & 3(\phi(1-\alpha)(1-\frac{c0\beta_3}{n1\beta_2^2}) - 2\mu\frac{c0\beta_3}{n1\beta_2^2}) \end{bmatrix}$$

.

Even for $\phi < \mu$, the above Jacobian has a positive eigenvalue. The stability analysis of a singular equilibrium is beyond our scope for now and is a subject we plan to study in the future.

C Proof of Uniqueness of Endemic Equilibrium

From (9) we know that

$$E[I^2] \geq (E[I])^2. \tag{13}$$

Next, setting the differential equation for $dE[I]/dt$ equal to 0 gives

$$E[I](\phi(1 - \alpha E[I]) - \mu) - \phi(1 - \alpha)E[I^2] = 0$$

which can be solved for

$$E[I^2] = \frac{E[I](\phi(1 - \alpha E[I]) - \mu)}{\phi(1 - \alpha)} \tag{14}$$

and finally

$$\frac{E[I^2]}{E[I]} = \frac{\phi - \mu - \phi\alpha E[I]}{\phi(1 - \alpha)}. \tag{15}$$

Note that we are assuming $E[I] > 0$ in the above, as we are looking for an endemic equilibrium. Inequality (13) and (15) imply

$$\frac{\phi - \mu - \phi\alpha E[I]}{\phi(1 - \alpha)} \geq E[I],$$

which can be solved for

$$E[I] \leq 1 - \frac{\mu}{\phi} \tag{16}$$

Next, from (11) we know that

$$\frac{(E[I^{w-1}])^2}{E[I^{w-2}]} \leq E[I^w],$$

which we can rewrite (using $w = 3$) as

$$\frac{E[I^3]}{E[I^2]} \geq \frac{E[I^2]}{E[I]}. \tag{17}$$

Setting the differential equation for $dE[I^2]/dt$ equal to 0 gives

$$(\phi(1-\alpha)-\phi\alpha E[I])E[I^2]+\phi\alpha(E[I])^2-\left(\phi(1-\alpha)+\frac{\mu E[I]}{E[I^2]}E[I^3]\right)=0,$$

which can be solved for

$$\frac{E[I^3]}{E[I^2]}=\frac{(\phi(1-\alpha)-\phi\alpha E[I])E[I^2]+\phi\alpha(E[I])^2}{\phi(1-\alpha)E[I^2]+\mu E[I]}.$$

Using equation (14) to substitute in for $E[I^2]$ in the above equation gives

$$\frac{E[I^3]}{E[I^2]}=\frac{(\phi(1-\alpha)-\phi\alpha E[I])\frac{E[I](\phi-\mu-\phi\alpha E[I])}{\phi(1-\alpha)}+\phi\alpha(E[I])^2}{\phi(1-\alpha)\frac{E[I](\phi-\mu-\phi\alpha E[I])}{\phi(1-\alpha)}+\mu E[I]}.$$

Some algebra reduces this to

$$\frac{E[I^3]}{E[I^2]}=\frac{(1-\alpha-\alpha E[I])(\phi-\mu-\phi\alpha E[I])+\phi\alpha(1-\alpha)E[I]}{\phi(1-\alpha)(1-\alpha E[I])}. \tag{18}$$

Using inequality (17), we know that

$$\frac{E[I^3]}{E[I^2]}-\frac{E[I^2]}{E[I]}\geq 0.$$

Using equations (15) and (18) to substitute into the above inequality gives

$$\begin{aligned}\frac{E[I^3]}{E[I^2]}-\frac{E[I^2]}{E[I]} &= \frac{(1-\alpha-\alpha E[I])(\phi-\mu-\phi\alpha E[I])+\phi\alpha(1-\alpha)E[I]}{\phi(1-\alpha)(1-\alpha E[I])}-\frac{\phi-\mu-\phi\alpha E[I]}{\phi(1-\alpha)}\\ &= \frac{(1-\alpha-\alpha E[I])(\phi-\mu-\phi\alpha E[I])+\phi\alpha(1-\alpha)E[I]-(\phi-\mu-\phi\alpha E[I])(1-\alpha E[I])}{\phi(1-\alpha)(1-\alpha E[I])}\\ &= \frac{\alpha(\phi E[I]-\phi+\mu)}{\phi(1-\alpha)(1-\alpha E[I])}\geq 0.\end{aligned}$$

Observe that the denominator in the last expression above is positive, assuming $0<\alpha<1$ and $\phi>0$, so the above inequality will be satisfied precisely when $\phi E[I]-\phi+\mu\geq 0$. This occurs when

$$E[I]\geq 1-\frac{\mu}{\phi}. \tag{19}$$

Together, inequalities (16) and (19) imply that $E[I] = 1 - \mu/\phi$.

This, together with equation (15) then implies that $E[I^2]/E[I] = 1-\mu/\phi$, i.e. $E[I^2] = (1-\mu/\phi)^2$.

Now assume that

$$E[I^w]/E[I^{w-1}] = 1 - \mu/\phi \tag{20}$$

for some particular w. Setting the equation for $dE[I^w]/dt$ equal to zero and rearranging shows that

$$\frac{E[I^{w+1}]}{E[I^w]} = \frac{\phi(1-\alpha) - \phi\alpha E[I] + \phi\alpha E[I]\frac{E[I^{w-1}]}{E[I^w]}}{\phi(1-\alpha) + \frac{\mu}{Q}},$$

Substituting in equation (20) and reducing then implies that

$$\frac{E[I^{w+1}]}{E[I^w]} = 1 - \frac{\mu}{\phi},$$

which by induction then shows that

$$E[I^w] = \left(1 - \frac{\mu}{\phi}\right)^w$$

for all $w = 1, 2, 3, \ldots$.

D Differences Between Original and Targeted Treatment Models

We know from (11) that:

$$\frac{(E[I^{w-1}])^2}{E[I^{w-2}]} \leq E[I^w],$$

which we can rewrite as

$$\frac{E[I^w]}{E[I^{w-1}]} \geq \frac{E[I^{w-1}]}{E[I^{w-2}]}.$$

When applied repeatedly, we get

$$\frac{E[I^w]}{E[I^{w-1}]} \geq \frac{E[I^y]}{E[I^{y-1}]} \quad \text{for} \quad w > y. \tag{21}$$

We already know that the equation for $dE[I]/dt$ is the same in the old and new models.

Next, in the original model, the equation for $dE[I^w]/dt$ has a recovery/treatment term of the form $-\mu E[I^w]$. In the new model, this term is now $-\mu E[I^{w+1}]E[I]/E[I^2]$ (assuming this is not the last equation, which has had the conditional-closure approximation applied to it). But because

$$\frac{E[I^{w+1}]}{E[I^w]} \geq \frac{E[I^2]}{E[I]}$$

by inequality (21), then

$$\frac{E[I^{w+1}]E[I]}{E[I^2]} \geq E[I^w],$$

which means we are subtracting a larger term from the equation for $dE[I^w]/dt$ in the targeted model than the original model. So if all moments start out equal, $E[I^w]$ will start to become smaller in the targeted model than the original model. In particular, $E[I^2]$ will become smaller in the targeted model, giving smaller variance between households.

Finally, in the original model, if we approximate $E[I^{w+1}]$ and truncate at the w^{th} moment, then the original equation for $dE[I^w]/dt$ has a treatment term of the form $-\mu E[I^w]$. In the new model, this term is now $-\mu E[I^{w+1}]E[I]/E[I^2]$ which we then approximate by $-\mu(E[I^w])^2E[I]/(E[I^2]E[I^{w-1}])$. We can rewrite this factor multiplying $-\mu$ as

$$\left(\frac{E[I^w]E[I]}{E[I^{w-1}]E[I^2]}\right)E[I^w]. \tag{22}$$

The factor inside of parentheses can then be rewritten as

$$\frac{E[I^w]/E[I^{w-1}]}{E[I^2]/E[I]},$$

which by inequality (21) we know is ≥ 1. This means that the coefficient of $-\mu$ in the treatment term of the targeted model, given by expression (22), is $\geq E[I^w]$, the coefficient in the original model. So even including the last equation which has the conditional closure included, all moments $E[I^w]$ for $w \geq 2$ are initially decreasing more quickly in the targeted model than in the original model, assuming the two models start out with the same initial conditions (and therefore the same moments). However, the moments of the two models begin to differ quickly and the above argument

no longer applies.