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Evaluation of control strategies for the spread of citrus greening

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EVALUATION OF CONTROL STRATEGIES FOR THE SPREAD OF CITRUS GREENING

A Thesis

by

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ABSTRACT

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Huanglongbing, also known as citrus greening, is a vector-based disease in citrus (with no cure known to date) that has drastically affected the citrus production in Florida in less than a decade and has been recently detected in Texas and California. In this paper, an epidemic model of the spatial spread of the disease is implemented among commercial and residential groves by taking into consideration the diffusion patterns of the psyllid vectors. A system of differential equations resembling one for malaria infection in humans is derived to evaluate different control methods such as quarantine, treatment, removal, foliar treatment, and pest control, among others. Using numerical techniques to analyze location data we determine the optimal techniques for limiting the spread of the disease. Finally, we measure the effect non-commercial trees have in carrying the infection over longer distances even if control measures have been established in commercial groves.

DEDICATION

To my parents, Heriberto Valle Aguilar and Lilia Martinez Galindo, whose unwavering support and love have enhanced my life, my awareness, and my career.

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I would to thank first of all the GAANN committee for allowing me to prioritize my education and giving me the opportunity to further explore the beauty and vastness of mathematics.

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CHAPTER I

INTRODUCTION

Citrus greening is the most important, severe, and destructive disease of citrus[13]. Also known as Huanglongbing (HLB), it is caused by the presence of the bacteria *Candidatus Liberibacter* inside the plant tissue and is carried between trees by the sap-sucking insect *Diaphorina Citri* upon feeding. Without control measures in effect, infection spread in an orchard will reach epidemic levels in an average of eight years, potentially rendering a citrus plantation unprofitable[4, 28]. There are three species of the bacteria: Asian, African, and American. The heat-tolerant species of bacteria Ca. L. asiaticus and the psyllid *D. Citri* are the most prevalent in the United States [23] and thus are the main focus of this work. Huanglongbing is a silent disease that results in death of the infected tree. It is characterized by a long latent period in which no symptoms are expressed but during which bacteria move within the tree and continually reproduce within the phloem, a part of the vascular system that delivers nutrients to the tree[10]. The bacteria affects all commercially cultivated citrus and no cure or resistant citrus varieties have been successfully developed to date, mainly because so far it has been impossible to isolate from the host for study with standard laboratory techniques.

1.1 Symptoms

A tree can be infected by HLB and only show symptoms after two years or more. However, an infected tree can be spotted because of the development of yellow shoots unevenly distributed across the tree with yellow and green blotches distributed asymmetrically throughout the leaves. It is common that most other branches of the tree may appear healthy even with high levels of infection.

Figure 1.1: Characteristic symptoms of Huanglongbing resulting in bitter lopsided fruit as well as leaf chlorosis and blotchy mottle. (Photos by USDAgov and by Monte Nesbitt)

Citrus greening results in eventual death of the tree due to dieback of tree sections and is normally preceded by symptoms such as yellowing of branches, blotchy mottle, production of bitter asymmetric fruit, and early fruit drop. However, the symptoms vary widely and are not a good indicator of pathogen concentration[16].

Other discernible symptoms are thicker leaves and production of asymmetric, discolored and bitter fruit. At later stages of the disease, often one to five years of first symptom expression [13], the nutrient restriction caused by the bacteria cause the infected part of the tree to spread and experience dieback eventually which ultimately results in tree death.

1.2 Impact

Groves of citrus begin to be profitable after 10 years [28]. However, an infected tree will be severely affected within 5 to 8 years of planting. Taking into consideration that the average lifespan of a citrus tree is 50 years throughout which they are continually productive, epidemic levels can rapidly render a plantation economically infeasible within seven to ten years after planting [1, 17, 14, 28]. Usually, due to the long incubation or latency period during which the disease may go unnoticed, the disease is already at high levels when symptoms are expressed. The spread of the infection in a grove is estimated to reach above 95% incidence in 3 to 13 years from first infection [13].

Once a tree acquires the disease, yield is reduced by 30 % to 100 % depending on the severity of the infection. In Florida alone, Hodges and Rahmani estimated the economic impact to be 8.9 billion dollars only for the 2007-08 season [30].

1.3 Insect Vector

Asian citrus psyllids (*D. Citri*), about 3.5 mm in length, feed on the nutrient-rich phloem by piercing the outer layer of the plants. The development of the psyllid is illustrated in figure 1.2 *Diaphorina Citri* has seven life stages: egg, five nymph phases or instars, and adult. The adult phase is reached in average from 13 to 19 days whereas the egg stage lasts between 2 to 4 days [19, 22].

Figure 1.2: Major stages of psyllid development in young tree flush. All nymph phases are not shown. A group of eggs will reach adulthood in roughly three weeks. (Photos from the University of California ANR Division, R. H. Brlansky, and USDA-ARS)

After mating, female psyllids lay eggs on undeveloped leaves and buds. The white eggs are .3 mm in length and turn yellow/orange in about three days (on average, depending on temperature) when they go into the first nymph stage. Adult psyllids will tend to continue feeding on mature leaves and migrate to new areas suitable for oviposition where new flush is available. A female psyllid produces from 800 to 1000 eggs over her lifespan[33]. Psyllids that become infected with the bacteria are able to carry it throughout their life.

Psyllids tend to move to other trees when disturbed by human movement or wind. Often this movement is to closer trees, but longer distances of 25 to 30 meters have been reported through mark and recapture techniques such as spraying a milk protein marker and then measuring its relative

density at different times in different locations[3]. It is still unclear to what extent this dispersal is mainly due to wind or other mechanism such as crowding effects.

Temperature affects the development, reproduction, and lifespan of psyllids. Liu and Tsay [26] studied developmental and survival rates as well as average number of eggs per female across a range of temperatures finding that longevity increased with reduced temperature (117 days at 15 °C to 51 at 30 °C) whereas female oviposition rate increased with temperature (up to a maximum of 748 eggs 28 °C).

1.4 Transmission

The bacteria resides in the nutrient-rich part of the vascular system of the tree, the phloem, which transports sap down the tree through different mechanisms. Chiyaka studied a model of dynamics of Huanglongbing within a citrus tree showing that it could influence the transmission rates of the disease at larger scales. However, the movement and multiplication of the pathogen are still not completely understood, as is pointed out in their paper [10]. Dynamics in play include bacterial growth which deprives cells of their nutrients or transport within the tree that varies with environmental conditions.

Transmission rates from infected tree to psyllids has also been hard to estimate because of bacterial growth occuring in both the host and the vector with intensity dependent on both the life stages of the psyllid, and other factors such as inconsistent feeding periods, temperature, varying disease levels within a tree, and nonhomogeneous population densities of both psyllids and citrus trees.

Because the severe effects of HLB, there are few studies in which the disease was studied for long periods of time without intervention. This makes it harder to measure the rates of infection for epidemic levels [17]. However, by comparing data across different studies, Gottwald found the most significant factors affecting spread were the amount of infectious areas nearby, vector population density and movement, and age of the grove at first infection [13].

There is a possibility for pathogen transmission across cohorts of psyllids. Chiyaka also found that this transmission is low, variable, and dependent on environmental conditions. The

factors influencing this type of infection are still being studied.

Regarding the movement of the vector, there is still speculation on the frequency and distance of psyllid dispersal. *D. Citri* were found to move 100 m within 3 days and up to 400 m within 4 days [18]. A report of the Florida Research and Education Center 2006 report has distance estimates from 50 to 270 miles and suggests the ability of psyllids migration that follows seasonal wind patterns. Furthermore, psyllids can also be transported through long distances by human movement through unprocessed fruit shipments or potted plants[22, 31]. In the Rio Grande Valley of Texas, reportedly, it was accidentally introduced in 2001, carried on *Murraya* plants from Florida [4].

The spatial distribution of the citrus psyllids seems to indicate a higher than expected aggregation level at grove edges as well as geographical features within citrus plantations such as roads, canals, ponds, etc[13, 29]. Furthermore, the spread pattern has been studied dispersion indices and spatial autocorrelation techniques, finding more aggregation of the disease in adjacent trees than those separated by rows[14].

When studying the spatial distribution of HLB, the continuity of the spread seems to depend on the scale. For larger scales, the spread is less continuous, perhaps due to the fact that citrus plantings are often discontinuous between regions[13, 20].

1.5 Alternate Hosts

Alternate routes of transmission can help mantain or increase levels of infection even when control procedures are in place for the species of interest. In particular, several citrus species have been confirmed as hosts of HLB and are known to contribute to the spreading of the disease.

The Florida Department of Agriculture and Consumer Services mantains a list of the known host plants of HLB and *D. Citri* that are common in the U.S.. The list contains 70 plant varieties, of which 31 carried HLB and 40 were vectored by *D. Citri*, where 21 of them belonged in both categories.

Further, inoculum sources are not only restricted to citrus varieties. Effective transmission between infected citrus trees and other plants such as dodder (Cuscuta campestris), periwinkle (Catbaranthus roseus), and tobacco (Nicotiana tabacum Xanthi) were shown by Bove[4] . However,

it is unclear the extent to which these contribute directly to transmission since they are not as attractive to psyllids.

1.6 Management

The following are techniques in place to control the spread of the disease:

Use of Certified Clean Nursery Stock

Avoiding the unaware introduction of an disease is required to avoid epidemic levels, There are many reasons why new stock may be introduced to a grove. By enforcing measures through which no infected material is introduced unadvertently, new innoculum sources can be avoided. HLB infection can also be transferred by grafting, a technique in which two pieces of plant tissue are joined with some advantages to the grower, such as earlier fruit production.

Chemical Control of Psyllid Population

Periodic insecticide application is the most common method of control for commercial groves that have the equipment, occurring from six to eight times a year[32]. However, it also has to be factored into the total management costs for the grove and may have different effects depending on the mode of action. For example, foliar and systemic insecticides may kill the insect shortly after feeding has occured. Similarly, rotation between insecticide modes of action has been suggested to mitigate the development of resistance by the psyllids[18].

Chemical methods need not be directly harmful to psyllids. Patt and Yotsuda studied the attraction of psyllids to several volatiles released by citrus varieties, showing the potential to create traps or attractors for psyllids that could decrease transmission rate by lowering psyllid population densities in susceptible areas[27, 21].

Chemical control is unlikely to happen in residential citrus groves where there is no management incentive due to profit losses, especially in small groves or for home owners with infected trees. Abandoned groves have been repeatedly shown to be a significant source of dispersal for HLB and *D. Citri* populations[32].

Removal of Symptomatic Trees

The effectiveness of removal for controlling the disease is dependent on the latency period of the infection, which is significantly long when compared to symptom expression. Regardless of actual duration, however, studies have shown that eradication of symptomatic trees alone will not eliminate the disease [30].

Regional Scale Management

Even when taking every measure to limit the spread of greening, disease prevalence in the surrounding areas can continue to be innoculum sources. Control is expected to be more effective when there is cooperation for controlling the disease at a regional level. However, there is still no consensus as to what quantifiable extent these measures need to be implemented in order to contain the disease. In fact, the application of these strategies vary widely between growers who evaluate how to apply them according to their own cases.

Quarantines and Regional Management

Quarantine procedures at regional scales have been lifted by the United States Department of Agriculture prohibiting the sale and transport of trees with the potential of infection.

Even with proper procedures in place within a grove, a neighboring grove that is not properly managed can continue to serve as a source of inoculum [30]. This suggests that management of the infection at larger scales will be more effective through a combined effort of growers.

High-resolution aerial sensing techniques have been shown to be a good prospect for identification of HLB-infected trees at larger scales, however, both techniques had false negatives of near 30% [12]. Furthermore, laboratory tests such as DNA assays for HLB infection are cumbersome at a large scale when no symptoms are expressed[23].

Less Successful Approaches

There are enhanced nutritional programs that ignore the disease progression and simply focus on repairing impaired production of fruit. These were shown by Gottwald to be ineffective in

sustaining tree health, yield, or fruit quality in some trials[15]. Research is ongoing to determine if these practices have promoted the increase of disease spread by large scale buildup of the inoculum in the hopes to maximize short-term profit.

Other methods of control have been tried with limited success. For instance, biological control through introduction of parasites [8, 9, 11] or predators of the vector. Introduction of T. Radiatus in Florida resulted in disease reduction ranging from 4% to as high as 70% in some cases. Introduction of other populations is also in conflict with application of insecticides, reducing the practical effectiveness of these measures. Finally, novel methods that involve influencing the movement of the vectors are being investigated. These include interplanting citrus with guava which apparently inhibits the vector, windbreaks [23], as well as sticky traps that release volatiles that attract the psyllids[27, 2].

CHAPTER II

MODEL FORMULATION

2.1 Epidemic Base ModeL

The epidemic dynamics of HLB transmission in a grove without replanting can be modeled in a simplified form as a compartmental model using a system of differential equations. This model will be adapted to incorporate explicitly the spatial effects and control measures of interest.

We consider the dynamics within a grove of citrus trees where there is a portion of susceptible (S_T) as well as infected (I_T) trees. Within the grove, we assume a population of psyllids that can be susceptible (S_P) or infected (I_P) exists in the area of interest. For the purpose of this section, the variables can represent total population density with respect to the plantation. Similarly, we assume infection occurs regardless of the degree of intensity of infection within the individual. We assume a well-mixed environment to show the epidemic nature of this simple model.

Susceptible individuals become infected at contact rates β_1 and β_2 respectively which represent the infection caused from an infected psyllid feeding on a susceptible tree and a susceptible psyllid feeding on an infected tree, respectively. The variable $\mathscr{R}$ is for now assumed to be zero, but will be a useful representation of a replanting control method in a later implementation.

All psyllids are assumed to have the same mortality rate μ_2 and growth rate e, regardless of infection status. Similarly, μ_1 represents the mortality rate of infected trees, which is assumed to be part of the infection.

$$\frac{dS_T}{dt} = -\beta S_T I_P + \mathscr{R} \tag{2.1}$$
$$\frac{dI_T}{dt} = \beta S_T I_P - \mu_1 I_T \tag{2.2}$$
$$\frac{dS_P}{dt} = -\beta S_T I_P - \mu_2 S_P + e S_P \tag{2.3}$$
$$\frac{dI_P}{dt} = \beta S_T I_P - \mu_2 I_P + e I_P \tag{2.4}$$

Assuming positivity of the parameters and variables for biological relevance, the behaviour of the system is shown in Figure . Table summarizes the parameters involved in the model as well as estimated values for the parameter as well as their sources.

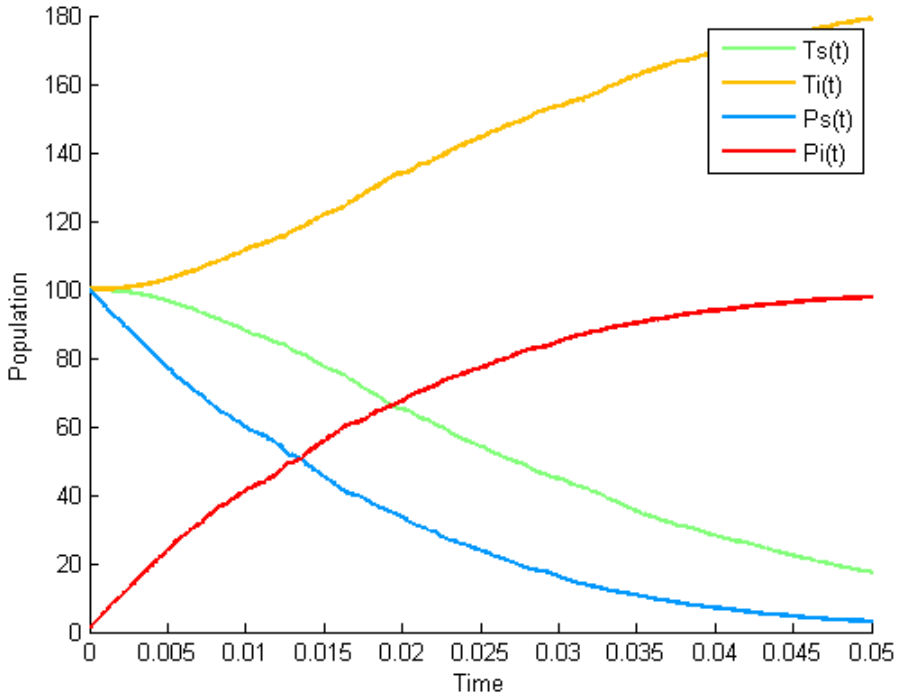

Figure 2.1: Short term dynamics of system (2.1)-(2.4)

The model seems to accurately represent the behaviour of an untreated grove with initial infectious trees. A careful choice of parameters ensures that the model is constrained to realistic values. It is worth mentioning at this point that, due to the biological interactions at play and relatively low understanding of the disease, citrus greening presents a challenge in traditional

parameter estimation. For instance, infection and mortality rates reported in the literature vary widely depending on many factors, including environmental variables such as temperature and wind, or the age structure of both populations.We now introduce a spatial component in the previous system.

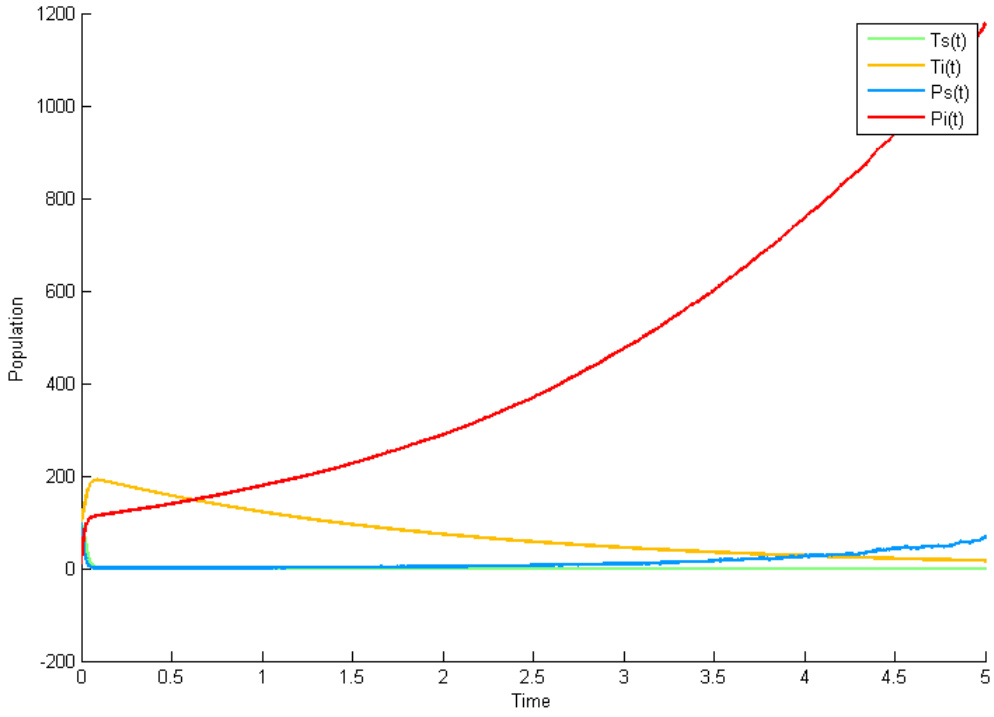

Figure 2.2: Long term dynamics of the system (2.1)-(2.4)

2.2 The Heat Equation

Whenever a function depends on more than one parameter, making it unable to directly calculate rates of change, we make use of partial differential equations. Functions may depend on the quantities located at given x and y coordinates (as is the case with our paper) or some other variables of interest. The heat equation is an example of a partial differential equation that can describe processes with diffusion.

We will use the heat equation model of diffusion to model the spread of psyllids. This will allow for modifications in our simulation to include factors such as wind (or drift) as well as

density-dependent effects.

The choice for the heat diffusion equation as a model for psyllid movement is based on the assumption that psyllids move randomly and move away from areas with high population density. Indeed, psyllids eventually leave infected trees because the infection restricts the nutrients available. Further, it corresponds to the biology of the life stages of the psyllids. Groups of new psyllid eggs tend to be together and disperse when reaching adulthood. A more careful treatment of the life-stage dependency of the dispersal could be of interest. Similarly, the spread may not be always locally continues because of factors such as accidental human transport or natural causes. Lee suggested introducing a probabilistic kernel in the dispersal function for this matter[22] and introduced an agent-based model with great detail. However, we will be working with the average local density of a population for the purposes outlined in this paper.

In what follows, we derive the diffusion or heat equation we used to model the spatial spread of psyllids. A more extensive discussion is outlined in [5]. Consider the motion of particles in a region of space. Our goal is to write an equation for the concentration of particles in a given region.

For that matter, that the results extend to $\mathbb{R}^n$. However, we will work only in two dimensions. A simple bounded region can be chosen to be the rectangle bounded by $x_0 < x < x_0 + h$ and $y_0 < y < y_0 + k$. Let $u(x,y,t)$ represent the density of a population behaving like particles at (x_0, y_0) at time t. From this, we get that the total number of particles in the region is approximately $hku(x,y,t)$ with an error proportional to $h^2 + k^2$.

At any given time and location, two processes can occur in relation to the number of particles: the creation of new particles due to some intrinsic growth, and the transcience of some particles through that location. Let $J(x,y,t) = J_1(x,y,t) + J_2(x,y,t)$ represent the flux in two dimensions as the sum of its corresponding vector components in the x and y directions. Finally, let Q(x,y,t) represent the growth rate of particles mentioned previously.

Then, we should have that the total amount of particles at a given point (x_0, y_0) should be the net flux (in all directions) plus the creation rate of particles at (x_0, y_0). This gives us

$$hku(x,y,t) = -k[J_1(x_0+h, y_0) - J_1(x_0, y_0, t)] - h[J_2(x_0, y_0+k, t) - J_2(x_0, y_0, t)]$$

For our purposes, we can let $Q(x,y,t) = 0$ since there is no spontaneous creation of psyllids other than that accounted for already by the model dynamics. By dividing both sides by *hk* and taking the limit as h and k approach zero, we get:

$$u_t(x,y,t) = -\left[\frac{\partial J_1(x,y,t)}{\partial x} + \frac{\partial J_2(x,y,t)}{\partial y}\right]$$

So far we have a statement relating the change in density with the flux of particles at a given point. However, it would be useful to work only with densities since those are only considered in our model. Here is where the assumption of random movement of psyllids is helpful.

Fick's law states that, as long as the motion is random, the flux is proportional to the rate of change in concentration. In other words, $J_1 = -\mathscr{D}u_x$ and $J_2 = -\mathscr{D}u_y$, where $\mathscr{D}$ is termed the diffusion coefficient. Therefore, we have derived the second order differential equation of interest, where the right-hand-side is in fact the Laplace (or divergence) operator:

$$u_t(x,y,t) = \mathbb{D}\left[u_{xx}(x,y,t) + u_{yy}(x,y,t)\right] = \mathbb{D}\Delta U$$

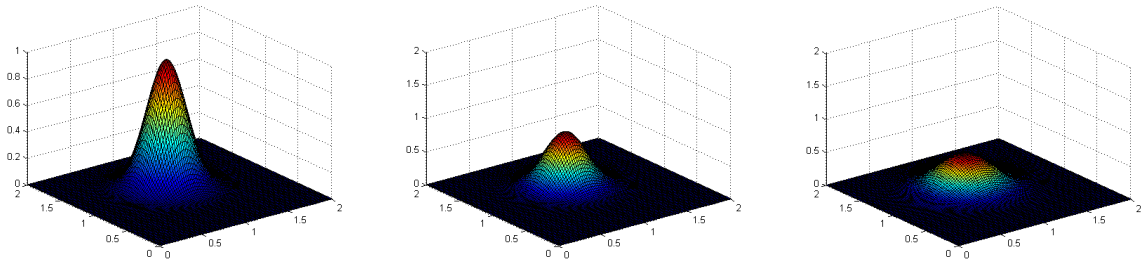

Figure 2.3: Illustration of the diffusion process at different time steps.
Speed of propagation is dependent on the diffusion coefficient.

The diffusion equation above is an example of an initial boundary value problem (IBVP) for which a unique solution exists provided suitable conditions at the boundary are known. The initial conditions can be specified in a multitude of ways. For example, Dirichlet initial conditions specify that particles are absent initially at the boundary. Another possible choice is a Neumann boundary condition, specifying that the partial derivative vanishes at the boundary, thus assuming that particles are confined to the region and never escape.

2.3 Wind and Linear Drift

The general equation for diffusion with a linear drift [5] is given by

$$\frac{\partial u}{\partial t} = a\frac{\partial^2 u}{\partial x^2} + (g - kx)\frac{\partial u}{\partial x}$$

For our purposes not involving altitude, we will not consider spatial dependence of the drift. Thus we can let $k = 0$. Notice $a = \mathscr{D}$ from the heat equation before.

A short derivation follows: Consider the diffusion equation where there is a flux term in one direction $\frac{\partial u}{\partial t} = a\frac{\partial^2 u}{\partial x^2} - [u(x, y-\varepsilon) - u(x,y)]$. We say at any given point, there is a net contribution in the flux from particles at some ε in the y-direction. From the taylor expansion of $u(y-\varepsilon)$ we readily get the approximation $u(x, y-\varepsilon) - u(x,y) = -\varepsilon\frac{\partial u}{\partial y} + \mathscr{O}(\varepsilon^2)$. Then, we will use $\frac{\partial u}{\partial t} = \mathscr{D}\frac{\partial^2 u}{\partial x^2} + \nu\frac{\partial u}{\partial x}$ where ν is a constant relating the magnitude of the drift.

2.4 Spatial Model with Diffusion of Psyllids

The study of local aspects of a SIR model with diffusion is known to exhibit more spatial patterns as well as nontrivial endemic states as explained in [7]. Reaction diffusion models are often used in chemical dynamics such as particle dynamics or for studying catalysts, often including a stochastic component for processes with a spatial nature[24, 25].

We consider the reaction-diffusion SI model of the interactions between densities of the state variables shown in equations (2.1) -(2.4) where now the state variables will be densities dependent on spatial location and incorporate a spatial diffusion term for random dispersion of psyllids.

Since our spatial domain is $\mathbb{R}^2$, representing a grove or geographical square area of length L, We assume that altitude has no effect in any of the state variables.

We have $u_i(\vec{x};t), x \in \Omega$ where $u_1 = s_T$, $u_2 = i_T$, $u_3 = s_P$, and $u_4 = i_P$ for $t \geq 0$.

The system can be described by the following system in $\Omega \times \mathbb{R}_+$

$$\frac{\partial u}{\partial t} - \mathscr{D}\Delta u(\vec{x};t) \quad = \quad f(u(\vec{x};t)) \tag{2.5}$$

Where $u = (u_1, u_2, u_3, u_4)^\intercal$; $\mathscr{D} = diag(0, 0, \mathscr{D}, \mathscr{D})$; Δ represents the laplace operator, and $f(z)$ is the interaction between densities according to the epidemic model introduced.

Thus, we have formulated the following:

$$\begin{aligned}
\frac{d}{dt}s_T(x,y) &= -\beta_1 s_T(x,y)i_P(x,y) + \mathscr{R}(x,y;t) && (2.6)\\
\frac{d}{dt}i_T(x,y) &= \beta_2 s_T(x,y)i_P(x,y) - \mu_1 i_T(x,y) && (2.7)\\
\frac{d}{dt}s_P(x,y) &= -\beta_1 s_T(x,y)i_P(x,y) - \mu_2 s_P(x,y) + eS_P + \mathscr{D}\Delta s_P && (2.8)\\
\frac{d}{dt}i_P(x,y) &= \beta_1 s_T(x,y)i_P(x,y) - \mu_2 i_P(x,y) + eI_P + \mathscr{D}\Delta i_p && (2.9)
\end{aligned}$$

Note that the totals for the state variables are found by integrating each variable over the domain. Namely,

$$\begin{aligned}
S_T &= \int_\Omega s_T(\vec{x};t)d\vec{x} && (2.10)\\
I_T &= \int_\Omega i_T(\vec{x};t)d\vec{x} && (2.11)\\
S_P &= \int_\Omega s_P(\vec{x};t)d\vec{x} && (2.12)\\
I_P &= \int_\Omega i_P(\vec{x};t)d\vec{x} && (2.13)
\end{aligned}$$

The force of infection at point $\vec{x} \in \Omega$ at a given time calculated using the law of mass action as $\beta i(x;t)s(x;t)$ can be changed in principle to $\mathscr{F}(i(\cdot;t))(\vec{x}) = \int_\Omega \mathscr{I}(\vec{x},\vec{x}\prime)i(\vec{x},\vec{x}\prime)d\vec{x}\prime$ were $\mathscr{I}$ represents the influence of infectives at point $\vec{x}\prime$ on the susceptibles at point $\vec{x}$. Although in this paper we do not use this modification, it could in principle be used where other effects related to the state variables take place in the transmission dynamics, such as distance from infection sites or distinct infectiousness between age cohorts.

CHAPTER III

METHODOLOGY AND FINDINGS

3.1 Numerical Simulation for Diffusion Model

We introduce a discretization of the model using densities with a diffusion term in order to run a numerical simulation of the control methods against citrus greening. We assume the trees are located in a rectangular grid with interactions occuring at each node with respect to the model. We use a forward-in-time central-in-space (FTCS) scheme for approximation of the differential equations in the system. Fourth-order Runge-Kutta method was implemented explicitly for the approximate solution of the system of differential equations of system 2.9-2.9 at each time step and was modified to hardcode changes in the model representing the application of control strategies. The MATLAB codes can be found in the appendix.

The program assigns parameters for infection rate from infected psyllids to healthy trees, infection rate from infected trees to healthy psyllids, death rate of trees due to HLB infection, natural death rate of psyllids, diffusion coefficient of psyllids, and growth rate of psyllids. We will assume that from initial conditions, trees are already susceptible and assumed to be adult trees ready for infection and hence no growth rate assumed for the tree densities. For infected trees, we assume a death rate caused by the deprivation of nutrients due to disease and eventual dieback. A removal procedure of infected trees is implemented at after specified period of time.

Although the program allows for different boundary conditions, Dirichlet boundary conditions will be used. That is, we set all values at the boundary to be zero. It is important to note that this choice does not stem from the model assumptions but it is necessary for the diffusion to have a unique solution. The model predictions are limited to points inside the boundary.

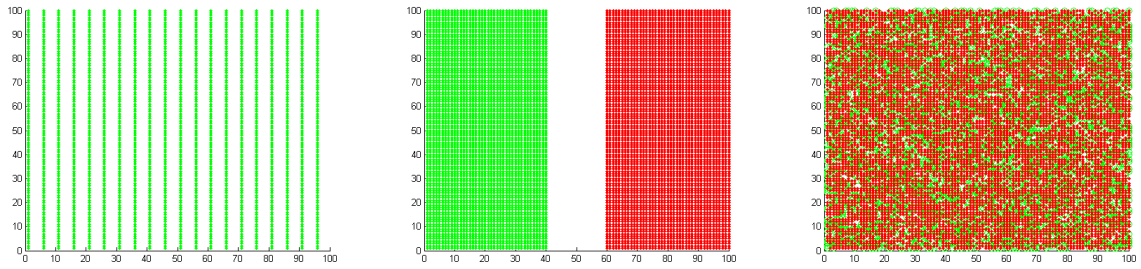

Figure 3.1: Different spatial arrangements of interest. Green marks represent a density of infected trees at that location. Gap lengths and percent concentrations can be adjusted accordingly.

In the program developed, different choices for initial conditions for the state variables can be chosen in order to test different scenarios representing an individual grove with a percent infection, or separate groves separated by a gap. This is exemplified in Figure 3.1. The values for row gap, distance between two groves of different infectious status, and percent of infection in a homogeneous grove can be specified before running the simulation.

In order to determine whether groups of infected psyllids could interact with susceptible psyllids at a different location, we choose a homogeneous initial condition of susceptible trees with no initial infected trees (Figure 3.2). Then, we study the cumulative dynamics of the system when the two groups are in either the same initial location or they start in two separate locations (Figure 3.3). Finally, we choose to test a case in which the concentration of psyllids (both infective and susceptible) are initially random at every location to determine the behavior without replanting (Figure 3.4).

To test the impact of separation between trees, whether it is within the grove or between groves, we consider initial conditions in which there is a separation between rows or groves of distinct infectious levels. We start with two separate groves, one infectious and one susceptible. Then start with an equal ammount of susceptible and infected psyllids with initial conditions at the center. We found that a neighboring infectious grove results in a larger growth of overall infectious psyllids. The results of the simulation are shown in Figure 3.5.

In addition, it is possible to introduce random infectious and susceptible trees in this gap to

represent the effect of homeowner's citrus trees and to determine if these, or alternate hosts, are able to bridge the disease between neighboring groves. A background level of susceptibles that is excluded from control methods can be introduced to represent the bridging caused by alternate hosts that were assumed to be virtually immune to removal procedures.

Active control methods involved are implemented as follows:

If a replanting function $\mathscr{R}$ is chosen, the program will cycle through every position in the grove and increment the susceptible tree density in that area at any given time based on a rule. For our purposes, we tested a constant replanting function against a procedural rule that acted locally. The cumulative dynamics did not present significant differences, however.

Chemical control of the psyllids is a function of time and is modeled as having a net increase in the mortality rate of the psyllids regardless of infectious status. This function was chosen to be a constant, or a periodic treatment corresponding to the frequency of applications found in the literature. For the periodic treatment, we chose a simple function $f(t) = a * (cos((t-20)/(pi)) + 1)/2$; where a is the net effect on mortality and is proportional to the application rate.

Removal of infected trees is also determined according to a rule which determines a location in which removal is necessary based on a tolerance level by taking into account the relative levels infection nearby. That is, at every location, it calculates the infection within a radius of size r_1 and then proceeds to set all infectious and susceptible tree densities at a radius r_2 to zero. Although the determination of infectiousness in the field is not practical yet, we assumed the optimal case in which at every time step we can determine the most infectious places of the grove and remove all adjacent trees before the infectious dynamics in the next time step. Finally, we also considered the case for periodic measurement and removal.

3.2 Results

By far, the most effective control technique of those tested in our model was the chemical control of the psyllid population. In fact, the overall dynamics of the system seem to suggest that psyllid movement is the driving process through which the disease spreads. This agrees with results in the literature [13] for the main factors influencing spread.

When comparing the dynamics of an initial condition of two clouds of psyllids (one infectious, one susceptible) at different locations in a homogeneous grove where no initial infectious trees were present, there was no significant difference in overall behavior of the system as in the case where both clouds of psyllids are centered at the same location. However, when both were at the same location, the growth of psyllids occurred slightly faster. Notice that our original model included no vector-vector infection. One possible explanation is that a higher concentration of infected psyllids is likely to infect trees rapidly while susceptible psyllids feed of infected trees.

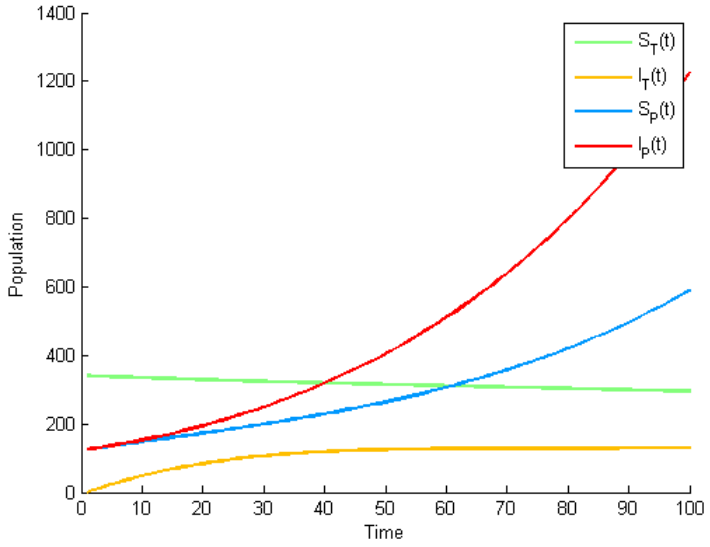

Figure 3.2: Cummulative dynamics of an initially susceptible homogeneous grove with psyllid clouds at the same location.

After this, still in a homogeneous susceptible grove, we study the changes in concentration of psyllids when at every location the levels of infectious and susceptible psyllids are random. After the simulation, we compare the final states of psyllids concentrations for both populations, as is shown in the following figure.We find that the aggregation patterns of both populations are distinct.

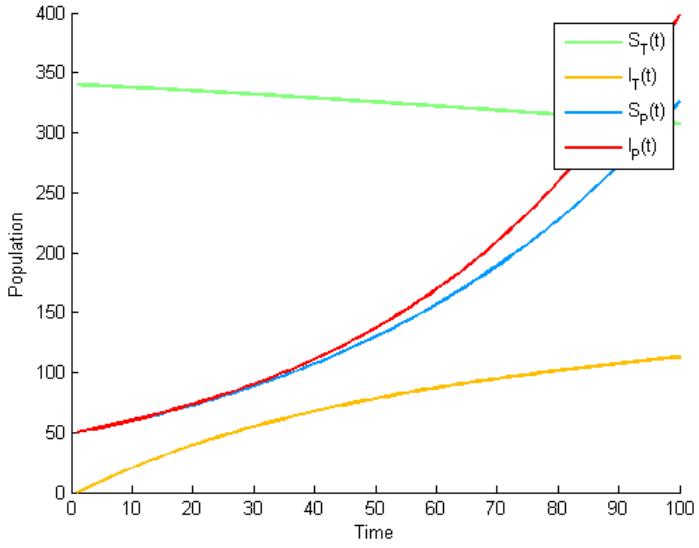

Figure 3.3: Cummulative dynamics of an initially susceptible homogeneous grove with psyllid clouds at separated locations.

Perhaps due to the high death rate of infectious trees used (estimates from the literature listed in the appendix) and the fact that our model does not include an explicit latent period of infection, we found that the infection was not significantly due to the spacing of the groves when compared to the movement of psyllids. This suggests that if the death rate of trees is high enough (either by natural or artificial means), isolated cases may be able to die off before bridging the infection between groves of citrus.

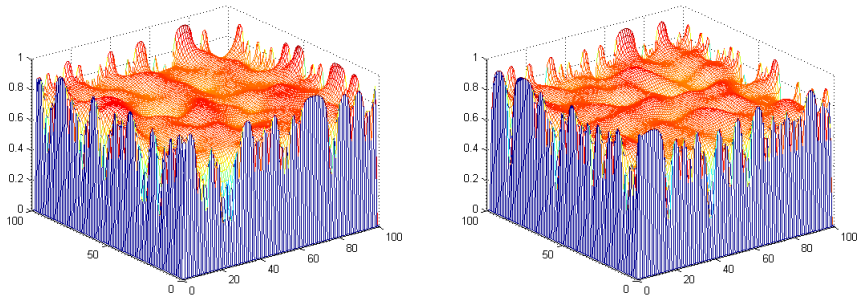

Figure 3.4: Final density distribution of susceptible (left) and infected (right) psyllids initially placed randomly in an initially homogeneously susceptible grove. No vector preference was coded but distinct aggregation patterns are found.

Similarly, no significant changes in the overall dynamics were found through the introduction of a constant replanting function or a procedure which replanted periodically. The cumulative dynamics did not present significant differences other than the expected increase in concentrations.

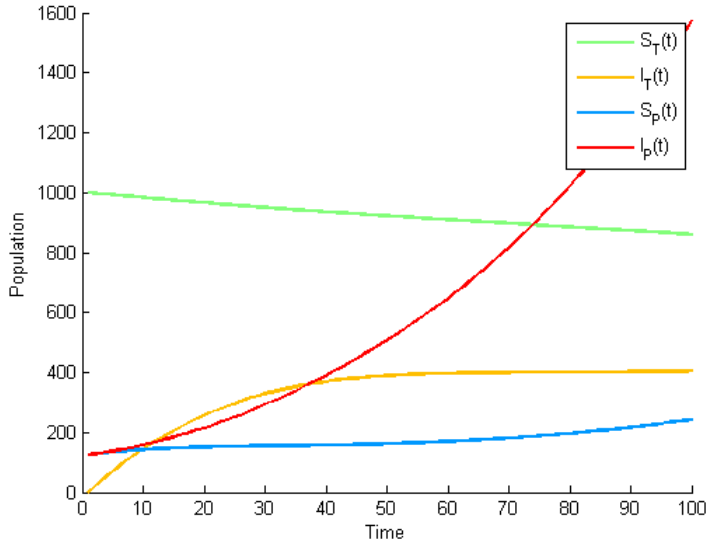

Figure 3.5: Effects of grove separation. Infectious sources of inoculum result in higher populations of infective psyllids.

Finally, the chemical control of psyllids was modelled as an increase of mortality rate for both groups of psyllids. The net contribution is then found to be directly proportional to the

concentration of the chemical at any given moment. Because this concentration corresponds to the cycles of insecticide application in place, we chose a simple periodic function.

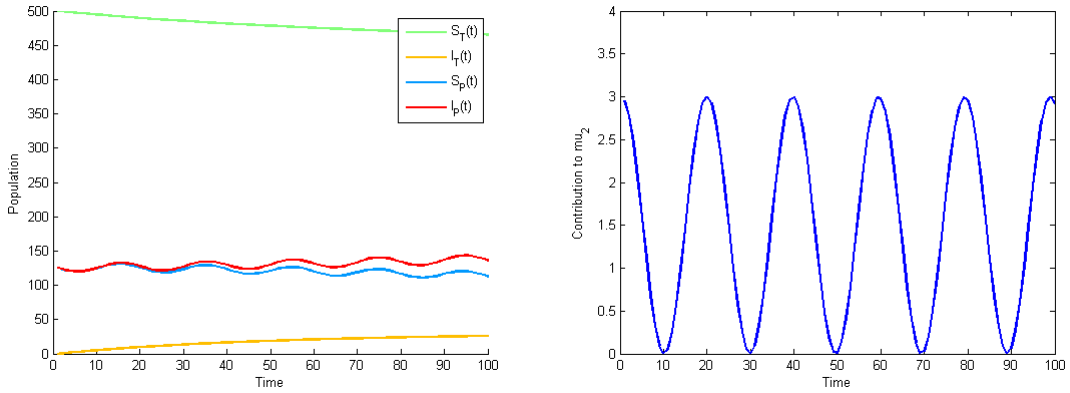

Figure 3.6: (a) System dynamics when including a contribution from chemical control. (b) The net effect of the insecticide application on mortality rate of psyllids.

One could argue that the function we choose determines the dynamics of the system. However, using a wider amplitude (greater net effect on mortality rate) for our insecticide application function seems to indicate that it is the magnitude of this effect drives the asymptotic dynamics in the long term by controlling the psyllid population.

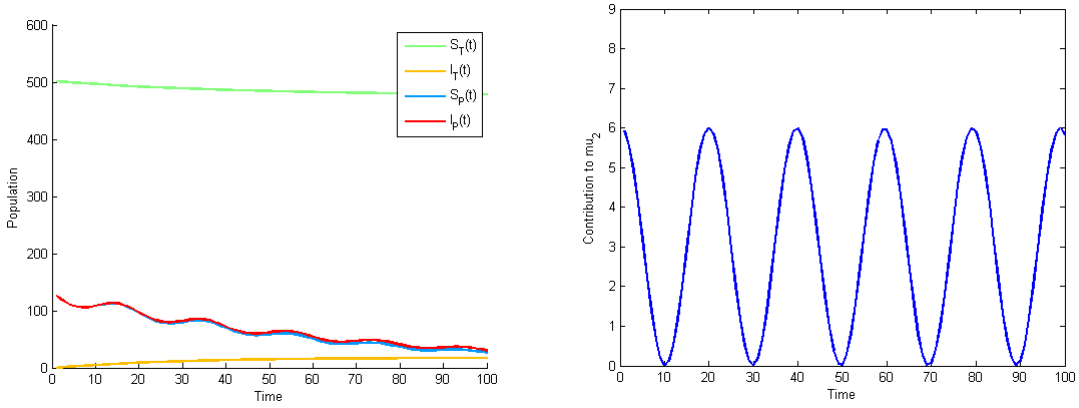

Figure 3.7: Effects of significanlty increasing the contributions of chemical control to psyllid mortality rate.

Other functions were used finding the same behavior driven by the magnitude of the

contribution. This function thus can be adapted in order to capture the insecticide decay behavior more realistically. For example, if the decay of the fungicide effect on psyllid death rate is linear, a sawtooth wave might be appropriate.

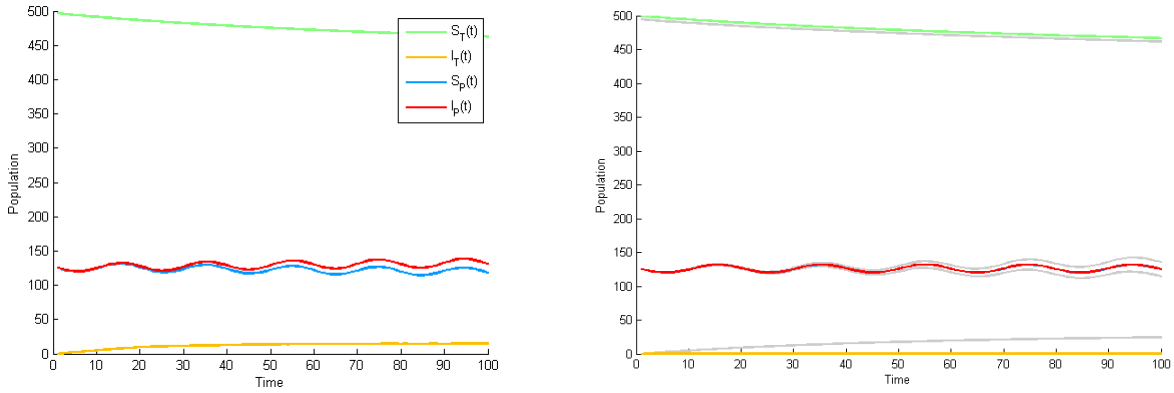

Figure 3.8: (a) shows the effects of removal of infectious trees. In (b), we compare the effects of no removal (in gray) with the cummulative behavior when removal is present.

Finally, we included a rule at every time step determining the removal of all infectious trees in a neighborhood at every cell if the total infection levels within the neighborhood were greater than a tolerance level.The removal of infected trees did not drive the overall asymptotic dynamics. Note that in this case we are assuming concurrent insecticide application.

CHAPTER IV

CONCLUSIONS

We were able to implement a numerical approximation algorithm to solve a system of differential equations with the inclusion of a nonlinear diffusion term representing the random movement of psyllids. Furthermore, we investigated the behaviour of the system when considering different cases of interest to a region.

The current program can be readily extended to incorporate actual spatial data by specifying the relative density of infectious and susceptible trees at any given location that could be gathered at large scale by GIS techniques. Psyllid movement patterns and locations of susceptible and alternate hosts could help determine the best strategies to apply in a region.

The model proposed can also be extended to include cohort dynamics or environmental variables in play such as wind, temperature and humidity provided their effects are accounted for in all of the affected parts of the algorithm.

Primarily, we found that the major driving mechanism in the cummulative dynamics of the system is psyllid concentration and movement. In addition, we tested a variety of control methods to determine the relative effectiveness for controlling the epidemic, finding that chemical control is the most promising. We also found that if the death rate of isolated infectious trees is high enough, the spread of the disease can be contained.

These results are of interest to growers seeking to maximize the ratio of yield to maintenance costs. In the future, we would like to apply the model to real world data such as from the United States Department of Agriculture. Regardless of the results found, we have not yet considered costs related to the application of control procedures that could help growers minimize costs and yield

loss. An approach such as exemplified in [6] for yield maximization and control expenditures can be implemented on these results.

A current limitation of these models is the difficulty to estimate parameters without laboratory data. Further work in the study of HLB should help refine these results.

APPENDIX A

APPENDIX A

1.1 PARAMETER VALUES

The following table indicates sources for the ranges of values used in simulations. The reader should keep in mind that these estimates vary depending on environmental factors and there is a relatively low understanding of HLB.

An asterisk in the citation indicates that the source arrived at the value through a listed review of literature from which values vary widely.

Obtaining more accurate estimates based on ground data would help optimize the predictions of the model which could in turn help determine optimality of control strategies and their costs.

Item			
Parameter	Description	Value	Source
β_1	Infection rate: infected psyllid to susceptible tree	$.88 - .3\ \frac{tr.u.}{day}$	[22, 13] *
β_2	Infection rate: infected tree to susceptible psyllid	$.95\ \frac{ps.u.}{day}$	[22, 13] *
$\mathscr{R}$	Replanting rate for new healthy trees	$Specified$	N/A
μ_1	Death rate of infected citrus tree	$\frac{1}{(5-8)}\ \frac{tr.u}{day}$	[28]
μ_2	Death rate of all psyllids	$\frac{1}{(51-117)}\ \frac{ps.u}{day}$	[22]
e	Reproduction rate of all psyllids	$7424.2\ \frac{eggs}{period}$	[26, 22] *
$\mathscr{D}$	Diffusion coefficient of psyllid movement	$7.23\ m - 4\ km$	[22] *

Table 1.1: Parameters chosen from literature.

1.2 MATLAB CODE: NONSPATIAL SYSTEM

```
function dy = dydtsys(t, y)
a=rand+rand; b=rand+rand;
```

```
c=rand+rand; d=rand+rand;
e=rand+rand; g=rand+rand;

dy=[
    -a*y(1)*y(4);                                % y(1)= ST
    a*y(1)*y(4) - b*y(2);                        % y(2)= IT
    -c*y(2)*y(3)-d*y(3)+e*(y(3)+y(4))+g*0;       % y(3)= SP
    c*y(2)*y(3)-d*y(4)+g*0;                      % y(4)= IP
    ];

%in the command line, run something like:
%[t y] = rk4sys(@dydtsys,[0 10],[0 0],2);
%disp([t' y(:,1) y(:,2)])
```

1.3 MATLAB CODE: NONSPATIAL SOLVER

```
clear all
tspan = [0 .05];
x0 = [100; 100; 100; 1;]; % A vector with I.C.'s
hold on
[t,x] = ode45('dydtsys',tspan,x0(:,1));

figure(1)
hold on
    plot(t,x(:,1),'Color',[132/256 255/256 123/256],'Linewidth',2);
    plot(t,x(:,2),'Color',[255/256 191/256 000/256],'Linewidth',2);
    plot(t,x(:,3),'Color',[000/256 153/256 255/256],'Linewidth',2);
    plot(t,x(:,4),'Color',[255/256 000/256 000/256],'Linewidth',2);
    legend('S_T(t)','I_T(t)','S_P(t)','I_P(t)');
xlabel('Time'); ylabel('Population');
```

1.4 MATLAB CODE: IMPLEMENTATION OF CONTROL METHODS MODEL

```
clear all

beta1=10; % Infection rate from infected psyllid to healthy trees
beta2=5; % Infection rate from infected tree to healthy psyllid
mu1=0.5; % Death rate of tree due to HLB infection
mu2=1; % Death rate of psyllids
ee=2.5;  % Growth rate of psyllids
g=.1; % Diffusion coefficient
gw=.1;%wind

grovegap=20; % this will be used for the gap between two groves (S vs I)
rowgap=3; % this is the gap between rows in a grove
wind=1;
replanting=0;

homes=1; % if 1, noncommercial areas included in model
howmany=.30; % percentage of noncommercial area infected

removal=0; % if this option is =1 there will be a removal procedure
R1=10; % radius of detection at a particular location
R2=5; % radius of elimination

xmax=10; %bounds of grid
ymax=10;

nx=100; % Number of grid steps in x and y directions
ny=100;
nt=100; % Number of time steps to run

insecticide=zeros(1,nt);
for i=1:nt
```

```
insecticide(i)=3*(cos((i-20)/(pi))+1)/2;
end

dx=xmax/nx; %lenghts of steps
dy=ymax/ny;
dt=.01;

x=0:dy:xmax; %note X(1)=0.
y=0:dy:ymax;

U1=zeros(nt,nx,ny); % solution matrices for state variables
U2=zeros(nt,nx,ny);
U3=zeros(nt,nx,ny);
U4=zeros(nt,nx,ny);

W1=zeros(nx,ny); % approximation matrices at a given time step
W2=zeros(nx,ny);
W3=zeros(nx,ny);
W4=zeros(nx,ny);

% Dirichlet Boundary conditions
UW=0;                              %x=0 Dirichlet B.C
UE=0;                              %x=L Dirichlet B.C
US=0;                              %y=0 Dirichlet B.C
UN=0;                              %y=L Dirichlet B.C
% Neumann Boundary Conditions
% UnW=0;                            %x=0 Neumann B.C (du/dn=UnW)
% UnE=0;                            %x=L Neumann B.C (du/dn=UnE)
% UnS=0;                            %y=0 Neumann B.C (du/dn=UnS)
% UnN=0;                            %y=L Neumann B.C (du/dn=UnN)

% Initial Conditions for-loops for each variable
```

```
%--- Susceptible Trees
%- Homogeneously distributed
%    U1(1,:,:)=1;
%- Square Groves distribution (left)
U1(1,:,:)=(rand(size(U1,2),size(U1,3))<.5)*.1; % random

%U1(1,:,:)=.025; % used as a baseline, otherwise no dynamics
% for j=2:nx
%     for k=2:ny
%         if (j<=(xmax/2-grovegap/2))
%             U1(1,j,k)=2;
%         else
%             %    U1(1,j,k)=0;
%         end
%         if (homes==1)
%             if (j<=(xmax/2+grovegap/2)&&j>=(xmax/2-grovegap/2))
%                 %if in the gap, create some noisy bridges
%                 U1(1,j,k)=U1(1,j,k)+(rand<.1)*.2;
%             end
%         end
%     end
% end
%    - Row Distribution
%     for j=1:rowgap:nx
%         for k=1:ny
%                 U1(1,j,k)=2;
%         end
%     end

% Initial conditions for SUSCEPTIBLE TREES ended here
```

```
%--- Infected Trees
%- No Initial Infection
%  U2(1,:,:)=0;
%- Homogeneously distributed
%     U2(1,:,:)=1;
%- Random with density percent
%     U2(1,:,:)=(rand(size(U2,2),size(U2,3))<.010);
%- Square Groves distribution (right)

%U2(1,:,:)=(rand(size(U2,2),size(U2,3))<.5)*.1; % random

%U2(1,:,:)=.025; % used as a baseline, otherwise no dynamics
% for j=2:nx
%     for k=2:ny
%         if (j>=(xmax/2+grovegap/2))
%             U2(1,j,k)=1;
%         else
%             %    U2(1,j,k)=0;
%         end
%
%         if (homes==1)
%             if (j<=(xmax/2+grovegap/2)&&j>=(xmax/2-grovegap/2))
%                 %if in the gap, create some noisy bridges
%                 U2(1,j,k)=U2(1,j,k)+(rand<.1)*.2;
%             end
%         end
%
%     end
% end
%- Row Distribution
%     for j=2:rowgap:nx
%         for k=2:ny
```

```
%                 U2(1,j,k)=2;
%             end
%         end
%     end
% Initial conditions for INFECTED TREES ended here

for j=1:nx
    for k=1:ny
%--- Susceptible psyllids
%- Center
U3(1,j,k)=(1/(pi*2)^(1/2))*exp(-(( (x(j)-x(nx/2) )^2)+ ...
    ( y(k)-y(ny/2) )^2 )*1);
%- Side (right)
%U3(1,j,k)=(1/(2*pi))*exp(-(( (x(j)-x(2*nx/5) )^2)+ ...
%   ( y(k)-y(ny/2) )^2 )*1);
%- Random with density percent
%U3(1,j,k)=(rand>.25);
%--- Infected psyllids
%- No Infection
%
%- Center
U4(1,j,k)=(1/(pi*2)^(1/2))*exp(-(( (x(j)-x(nx/2) )^2)+ ...
    ( y(k)-y(ny/2) )^2 )*1);
%- Side (left)
%U4(1,j,k)=(1/(2*pi))*exp(-(( (x(j)-x(3*nx/5) )^2)+ ...
%   ( y(k)-y(ny/2) )^2 )*1);
%- Random with density percent
%U4(1,j,k)=(rand>.25);
    end
end
% Initial conditions for BOTH PSYLLIDS trees ended here
```

```
% plot initial conditions here



ready=1;
if ready==1
    for i=1:nt-1
        W1(:,:)=U1(i,:,:);
        W2(:,:)=U2(i,:,:);
        W3(:,:)=U3(i,:,:);
        W4(:,:)=U4(i,:,:);

        U3(i,1,:)=US; % every time the loop runs update boundary conditions
        U3(i,nx,:)=UE;
        U3(i,:,1)=UN;
        U3(i,:,ny)=UW;

        U4(i,1,:)=US; % these apply only to the diffusion processes
        U4(i,nx,:)=UE;
        U4(i,:,1)=UN;
        U4(i,:,ny)=UW;

        W1(:,:)=W1(:,:)+replanting*(W1(:,:)==0);

        k1W1=dt*(-beta1*W1.*W4);
        k1W2=dt*(beta1*W1.*W4-mu1*W2);

        k1W3=zeros(nx,ny);
        k1W4=zeros(nx,ny);
```

```
        for j=2:nx-1
            for k=2:ny-1
        k1W3(j,k)=dt*(-beta2*W2(j,k)*W3(j,k)-(mu2+...
            insecticide(i))*W3(j,k)+ee*W3(j,k)+g*...
            ((W3(j+1,k)-2*W3(j,k)+W3(j-1,k))/(dx^2) + (W3(j,k+1)-...
            2*W3(j,k)+W3(j,k-1))/(dy^2)));
        k1W4(j,k)=dt*(beta2*W2(j,k)*W3(j,k)-(mu2+...
            insecticide(i))*W4(j,k)+ee*W4(j,k)+g*...
            ((W4(j+1,k)-2*W4(j,k)+W4(j-1,k))/(dx^2) + (W4(j,k+1)-...
            2*W4(j,k)+W4(j,k-1))/(dy^2)));
            end
        end



        k2W1=dt*(-beta1*(W1+k1W1/2).*(W4+k1W4/2));
        k2W2=dt*(beta1*(W1+k1W1/2).*(W4+k1W4/2)-mu1*(W2+k1W2/2));

        k2W3=zeros(nx,ny);
        k2W4=zeros(nx,ny);

        for j=2:nx-1
            for k=2:ny-1
k2W3(j,k)=dt*(-beta2*(W2(j,k)+k1W2(j,k)/2)*(W3(j,k)+k1W3(j,k)/2)...
    -(mu2+insecticide(i))*(W3(j,k)+k1W3(j,k)/2)+ee*(W3(j,k)+...
    k1W3(j,k)/2)+g*(((W3(j+1,k)+k1W3(j+1,k)/2)-2*(W3(j,k)+k1W3(j,k)/2)+...
    (W3(j-1,k)+k1W3(j-1,k)/2))/(dx^2) + ...
    ((W3(j,k+1)+k1W3(j,k+1)/2)-2*(W3(j,k)+k1W3(j,k)/2)+...
    (W3(j,k-1)+k1W3(j,k-1)/2))/(dy^2)));

k2W4(j,k)=dt*(beta2*(W2(j,k)+k1W2(j,k)/2)*(W3(j,k)+...
    k1W3(j,k)/2)-(mu2+insecticide(i))*(W4(j,k)+k1W4(j,k)/2)+ee*(W4(j,k)+...
```

```
    k1W4(j,k)/2)+g*(((W4(j+1,k)+k1W4(j+1,k)/2)-2*(W4(j,k)+...
    k1W4(j,k)/2)+(W4(j-1,k)+k1W4(j-1,k)/2))/(dx^2) + ...
    ((W4(j,k+1)+k1W4(j,k+1)/2)-2*(W4(j,k)+k1W4(j,k)/2)+...
    (W4(j,k-1)+k1W4(j,k-1)/2))/(dy^2)));
            end
        end

        k3W1=dt*(-beta1*(W1+k2W1/2).*(W4+k2W4/2));
        k3W2=dt*(beta1*(W1+k2W1/2).*(W4+k2W4/2)-mu1*(W2+k2W2/2));

        k3W3=zeros(nx,ny);
        k3W4=zeros(nx,ny);

        for j=2:nx-1
            for k=2:ny-1
k3W3(j,k)=dt*(-beta2*(W2(j,k)+k2W2(j,k)/2)*(W3(j,k)+...
    k2W3(j,k)/2)-(mu2+insecticide(i))*(W3(j,k)+k2W3(j,k)/2)+ee*(W3(j,k)+...
    k2W3(j,k)/2)+g*(((W3(j+1,k)+k2W3(j+1,k)/2)-2*(W3(j,k)+...
    k2W3(j,k)/2)+(W3(j-1,k)+k2W3(j-1,k)/2))/(dx^2) + ...
    ((W3(j,k+1)+k2W3(j,k+1)/2)-2*(W3(j,k)+k2W3(j,k)/2)+...
    (W3(j,k-1)+k2W3(j,k-1)/2))/(dy^2)));

k3W4(j,k)=dt*(beta2*(W2(j,k)+k2W2(j,k)/2)*(W3(j,k)+...
    k2W3(j,k)/2)-(mu2+insecticide(i))*(W4(j,k)+k2W4(j,k)/2)+ee*(W4(j,k)+...
    k2W4(j,k)/2)+g*(((W4(j+1,k)+k2W4(j+1,k)/2)-2*(W4(j,k)+...
    k2W4(j,k)/2)+(W4(j-1,k)+k2W4(j-1,k)/2))/(dx^2)+...
    ((W4(j,k+1)+k2W4(j,k+1)/2)-2*(W4(j,k)+k2W4(j,k)/2)+...
    (W4(j,k-1)+k2W4(j,k-1)/2))/(dy^2)));
            end
        end

        k4W1=dt*(-beta1*(W1+k3W1).*(W4+k3W4));
```

```
        k4W2=dt*(beta1*(W1+k3W1).*(W4+k3W4)-mu1*(W2+k3W2));

        k4W3=zeros(nx,ny);
        k4W4=zeros(nx,ny);

        for j=2:nx-1
            for k=2:ny-1
k4W3(j,k)=dt*(-beta2*(W2(j,k)+k3W2(j,k))*(W3(j,k)+k3W3(j,k))-...
    (mu2+insecticide(i))*(W3(j,k)+k3W3(j,k))+ee*(W3(j,k)+k3W3(j,k))+g*...
    (((W3(j+1,k)+k3W3(j+1,k))-2*(W3(j,k)+k3W3(j,k))+(W3(j-1,k)+...
    k3W3(j-1,k)))/(dx^2) + ((W3(j,k+1)+k3W3(j,k+1))-...
    2*(W3(j,k)+k3W3(j,k))+(W3(j,k-1)+k3W3(j,k-1)))/(dy^2)));

k4W4(j,k)=dt*(beta2*(W2(j,k)+k3W2(j,k))*(W3(j,k)+k3W3(j,k))-...
    (mu2+insecticide(i))*(W4(j,k)+k3W4(j,k))+ee*(W4(j,k)+k3W4(j,k))+g*...
    (((W4(j+1,k)+k3W4(j+1,k))-2*(W4(j,k)+k3W4(j,k))+...
    (W4(j-1,k)+k3W4(j-1,k)))/(dx^2) + ((W4(j,k+1)+k3W4(j,k+1))-...
    2*(W4(j,k)+k3W4(j,k))+(W4(j,k-1)+k3W4(j,k-1)))/(dy^2)));
            end
        end

        U1(i+1,:,:)=W1+(1/6)*(k1W1 + 2*k2W1 + 2*k3W1 + k4W1);
        U2(i+1,:,:)=W2+(1/6)*(k1W2 + 2*k2W2 + 2*k3W2 + k4W2);
        U3(i+1,:,:)=W3+(1/6)*(k1W3 + 2*k2W3 + 2*k3W3 + k4W3);
        U4(i+1,:,:)=W4+(1/6)*(k1W4 + 2*k2W4 + 2*k3W4 + k4W4);

        if (removal==1) && (mod(i,10)==0)
       for j=3:nx-1
           for k=3:ny-1
               %behold the square circle of radius 1
               %maxnorm ball
            totalinfective=U2(i,j-1,k+1)+ U2(i,j,k+1)+ U2(i,j+1,k+1)+...
```

```
                        U2(i,j-1,k)   + U2(i,j,k)   + U2(i,j+1,k)+...
                        U2(i,j-1,k-1)+ U2(i,j,k-1)+ U2(i,j+1,k-1);
            totalsusceptible=U1(i,j-1,k+1)+ U1(i,j,k+1)+ U1(i,j+1,k+1)+...
                        U1(i,j-1,k)   + U1(i,j,k)   + U1(i,j+1,k)+...
                        U1(i,j-1,k-1)+ U1(i,j,k-1)+ U1(i,j+1,k-1);
            if ((totalsusceptible-totalinfective)/2-.5)<0
                U1(i+1,j,k)=0;
                U2(i+1,j,k)=0;
            end

          end
       end
        end

    end
end

%% Plot totals

Utotals=zeros(4,nt);
for time=1:nt
    for xdir=1:nx
        for ydir=1:ny
            Utotals(1,time)=Utotals(1,time)+U1(time,xdir,ydir);
            Utotals(2,time)=Utotals(2,time)+U2(time,xdir,ydir);
            Utotals(3,time)=Utotals(3,time)+U3(time,xdir,ydir);
            Utotals(4,time)=Utotals(4,time)+U4(time,xdir,ydir);
        end
    end
end

hold on
```

```
%figure(1)

plot(1:nt,Utotals(1,:),'Color',[132/256 255/256 123/256],'Linewidth',2);
plot(1:nt,Utotals(2,:),'Color',[255/256 191/256 000/256],'Linewidth',2);
plot(1:nt,Utotals(3,:),'Color',[000/256 153/256 255/256],'Linewidth',2);
plot(1:nt,Utotals(4,:),'Color',[255/256 000/256 000/256],'Linewidth',2);
legend('S_T(t)','I_T(t)','S_P(t)','I_P(t)');
xlabel('Time'); ylabel('Population');

%% Below are all of the mesh plotting functions

%figure(1)
%axis([0 xmax 0 ymax 0 100])

% %for repeat=1:20
%     for count=1:nt-1
%     %hold on
%     %A1(:,:) = U1(count,:,:);
%     %mesh(1:nx,1:ny,A1)
%     %A2(:,:) = U2(count,:,:);
%     %mesh(1:nx,1:ny,A2)
%     figure(1)
%     A3(:,:) = U3(count,:,:);
%     mesh(1:nx,1:ny,A3)
%
%     figure(2)
%     A4(:,:) = U4(count,:,:);
%     mesh(1:nx,1:ny,A4)
%     pause(0.001)
%     %hold off
%     end
%end
```

```
%xlabel('y')
%ylabel('x')

%% plotting initial conditions

% U1reduced=squeeze(U1);
% hold on
%     for j=1:nx
%         for k=1:ny
%             if U1reduced(j,k)~=0
%             plot3(j,k,U1reduced(j,k),'g.');
%             end
%         end
%     end
```

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BIOGRAPHICAL SKETCH

Vicente Valle Martinez was born in Jalisco, Mexico. He moved to the United States to continue his studies and graduated and earned a Bachelor of Science in Applied Mathematics with a Chemistry minor from The University of Texas - Pan American (soon to be University of Rio Grande Valley) in 2013. He currently resides at 1604 N San Antonio St, Alton, TX 78573 and is reachable at vvalle1@broncs.utpa.edu.

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Vicente was a research assistant at Rensselaer Polytechnic Institute's Computational Science Training for Undergraduates in the Mathematical Sciences (CSUMS) as well as at Arizona State University's Mathematical and Theoretical Biology Institute (MTBI) summer research programs. He is also involved with the development of local technology through Tech Tuesdays and Code#RGV events.

He received a Master of Science in Mathematical Sciences from UTPA in July 2015 after receiving GAANN Fellowship support for two years. He will soon continue doctoral studies at Iowa State University. Vicente aims to continue using mathematics and computing in order to develop meaningful research projects and models in order to one day become a professor at a prestigious university or research group and work to inspire and guide other students to further their education.