MODELING PLANT VIRUS PROPAGATION AND AN OPTIMAL CONTROL

by

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Abstract

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Plants are a food source for man and many species. They also are sources of medicines, fibers for clothes, and are essential for a healthy environment. But plants are subject to diseases many of which are caused by viruses. These viruses often kill the plant. As a result, billions of dollars are lost every year because of virus related crop loss. Most of the time, virus propagation is done by a vector, usually insects that bite infected plants, get themselves infected and then bite susceptible plants. To combat the vectors, and ultimately the viruses, pesticides are often used as a control. Unfortunately, chemicals in pesticides can have a harmful effect on their environment. An alternative method to control the insect population is to introduce a natural predator of the insect. These predators may be more expensive than insecticides, but they are more environmentally friendly. To understand the dynamics, a system of ordinary and delay differential equations modeling interactions between insects and plants is considered and analyzed. To analyze the system, the basic reproductive number is used along with numerical simulations to find bifurcations. Then, a predator is introduced to the model, and the dynamics are studied in a similar fashion. Because of the seasonality of insects, active in the warm months and almost dormant in the cooler ones, the model is then analyzed with periodic coefficients. To study this model, the basic reproductive number is used, but calculated in a couple of different ways: a time average approach and a linear operator one. Finally an optimal control problem is

studied. In this problem, the goal is to minimize the cost of the insecticide, predator, and cost of an infected plant. To solve this problem, two approaches are taken: an indirect approach using Pontryagin maximum principle and a direct approach used in the BOCOP software package.

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Chapter 1
Introduction

Plants are a food source for man and many species. They also are sources of medicines, fibers for clothes, and are essential for a healthy environment. But plants are subject to diseases many of which are caused by viruses. These viruses often kill the plant. As a result, billions of dollars are lost every year because of virus related crop loss. [1] The most common way a plant virus is spread is by an insect vector. The main goal of this dissertation is to model the plant virus propagation in order to understand the dynamics, introduce a predator of the insects as a biological control, and determine the amount of insecticides and predators needed to minimize the cost of vector control.

The following chapters are a compilation of four papers all of which are related to the modeling of plant viruses or their control and a conclusion. The first three papers are published and the last is in preparation for publication. The published papers were submitted to and accepted by the Journal of Computational and Applied Mathematics-Elsevier. Elsevier gives full permission to use the works in this dissertation. Also, all works were co-authored by Benito Chen. He gives permission to use the works in this dissertation as well. In all papers, Mark Jackson was the main author: writing the literature reviews, developing the mathematical models, developing the code for the numerical simulations, and interpreting the results, Benito Chen supervised the work by helping in the development of the models by providing insight, assisting with the development and troubleshooting of code of the numerical simulations, especially with the direct methods in Chapter 5, providing insight of the numerical results and proofreading the papers before submission.

The first paper [2] discusses models of plant viruses with both ordinary differential equations and delay differential equations . The ODEs are studied first with the basic reproductive number used to determine stability of disease free steady states. Because it takes time for a virus to spread

within a plant and vector, a time delay is considered. By incorporating the delay, a system of DDEs are formed. Analyzing the disease free equilibria of the system is not possible analytically. Thus, a stability and bifurcation analysis is done numerically. The results are summerized and conclusions are given.

The second paper [3] discusses a biological control of the virus-transmitting vectors. Because insecticides can be toxic to the environment, a natural predator can be used as a safer alternative to the toxins. The model from the previous chapter is updated to include a predator equation. A stability analysis and bifurcation analysis is performed. Due to the nature of the system, the analysis is again done numerically. The results are summerized and conclusions are given.

The next chapter and paper [4] explores a model of plant viruses when seasonality is considered. Depending on the season, insects have different behaviors. To account for this biological fact, periodic coefficients are introduced to the model. To study the dynamics this model, the basic reproductive number R_0 is used. There are two different approaches to calculating R_0. The first is using a time average approach and the second uses a linear operator approach. Both methods are applied to the model and results are compared.

The following chapter discusses an optimal control problem in which the goal is to determine the minimum cost of using an insecticide and a natural predator to combat plant viruses. Two different approaches are used, a direct and an indirect approach. Direct methods have the advantage over indirect methods in that they are more straightforward to apply and more robust with respect to the initialization. The cost, however, is that some precision is lost. Both methods are applied to the optimal control problem and results are compared. The final chapter discusses the conclusions.

References

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Chapter 2
Modeling Plant Virus Propagation with Delays

Mark Jackson[1], Benito M. Chen-Charpentier[1,*]

Abstract

Plants play a vital role in the everyday life of all organisms on earth. Sometimes, however, plants become infected with a virus. This can have a devastating effect on the ecosystem that depends on it. An insect vector can cause the transmission of the virus from plant to plant. In this paper, a system of ordinary differential equations was first used to model the interaction between the insects and the plants. We found the equilibria of the model, and we analyzed the stability using the Reproductive number, derived by the next generation matrix. Afterwards, we introduced a delay to the model and a system of delay differential equations (DDE) was obtained. Analysis of the DDEs dynamics was performed including equilibria, stability, and bifurcations. Then, numerical solutions of the ODE and the DDE were compared and conclusions are given.

Keywords: delay differential equations, virus propagation, mathematical modeling

1. Introduction

Plants are essential to not only man's existence, but to every species on Earth. Sometimes, plants become infected with a disease. There are many different ways that a plant may contract a disease. One of which is bacterial. For example, *Magnaporthe oryzae* is a bacteria that causes rice blast which can cause rice production to decrease up to 90 percent [5]. Also, a plant may become infected with a fungal disease. One such fungus is *Botrytis*

*M. Jackson, B. Chen, Modeling plant virus propagation with delays, Journal of Computational and Applied Mathematics. 309 611–621.(2016) Eleavier grants permission to use paper in dissertation.

cinerea, and it destroys the fruits the plant produces [11]. In this paper, we will be interested in modeling the interaction between plants, a plant virus, and the insect vector that transfers the virus from one plant to another.

In order to work with the viruses, we must first understand how they replicate. For a virus to replicate, it must invade a healthy cell and use the cell's DNA or RNA to reproduce. The infected cell bursts and several copies of the virus exit. The new virus particles infect other cells. The viruses continue this process until there are no more healthy cells to invade. Different virus processes have been widely studied. See for example [2], [13], [12], and [19].

Plant viruses cause many diseases some of which that affect many plants all over the world. For instance, the *Citrus tristeza* virus once wiped out millions of trees in Brazil [8]. But in order for an infected plant to infect another, a virus from an infected plant must come in contact with a healthy plant. This may happen in several different ways. For example, a field worker might contact the juices of an infected plant and contact a healthy plant. Or maybe the interaction is more organic in that an infected plant might have its juices fall directly onto a plant underneath it. Another, and the most common, way for the transference is by an insect vector. Insect vectors transmit more than 70 percent of all known plant viruses [8]. Many vectors that transport these viruses include aphids, whiteflies, leafhoppers, etc.

There are many ways that plant viruses interact with the vectors, but in this paper we are concerned with circulative, persistent transmission. This transmission works in the following way. The vectors consume sap from an infected host through their stylets. The viruses in the sap enter the salivary glands, circulates within the vector, and then causes infection. This process can take a few hours or up to a day depending on the insect-vector interaction. The vector will hold the infection for the rest of its life. When the infected vector contacts a healthy plant, some virus particles leave the vector and invade the plant [16, 18, 14]. Once the virus has circulated and propagated throughout the plant, the plant may use defense

mechanisms to combat the virus. On example is by antiviral RNA silencing, a process by which slicing or translation repression of viruses occurs [9].

Many physical and biological processes (gestation, maturation, reproduction, infection) take time to complete. In the case of a viral infection, it takes time for a virus to invade a cell, reproduce, and spread in order throughout the host. This process time is a delay time. Processes with delay times can be modeled using delay differential equations (DDE). Delay times can change the dynamics of the model. For example, delays may change the solutions, cause discontinuities in the derivative, introduce oscillations, affect uniqueness, or change the stability. Despite the complications and numerical difficulties, results are more realistic from the biological and physical points of view. The effects of the delay times are highly coupled with the parameters of the model. See for example [7, 10].

Although there are many models that describe the interaction between vectors and humans, there are not as many that describe the relationship between plants and vectors. In this paper, we construct a model assuming that the virus gets transmitted by plant and insect contact, and is modeled using Holing type II [1], since insects can only bite a limited number of plants. In [16] a similar model is presented that also consider that infection can be transmitted from plant to plant. We do not consider such transmission because there is empirical evidence it is not common [18]. In Section 2, we will further develop the assumptions of the model and introduce the system of ordinary differential equations. Then equilibria and stability will be analyzed with the basic reproduction number, using the next generation matrix approach. In Section 3, we will introduce a delay to the model. We perform a stability analysis in this section as well. In Section 4, we compare the numerical solutions to the systems of ODEs and DDEs. Also, some numerical bifurcation analysis will be presented. Finally in Section 5, some conclusions are given.

2. Model Assumptions and System of ODEs

We assume a general model, since there has been no data collected for a specific plant, virus, and insect vector.

There are three populations of plants: Susceptible, S, healthy but subject to be infected by the virus, Infective, I, already infected by the virus, and Recovered, R. Each of these variables describe their respective population at time, t. The total number of plants will be denoted by the fixed positive constant K, $K = S + I + R$. It is reasonable to assume K is fixed, because when a plant dies by the virus or natural death in farms, it is replaced with a new healthy plant. The new plant shares the same characteristics of the plant it replaced, before it was infected.

For the insect vectors, there will be two populations : Susceptible, X, and Infective, Y. Each of which describe the populations at time, t, as well. The total number of insects will be denoted by the constant, N, N=X+Y. Also, the rate at which the insects enter the system, by birth or immigration, is constant. There is no vertical transmission of the virus, and vectors cannot transmit the virus to another vector. In addition, vectors do not get killed by the virus nor do they defend against it. The vector will keep the virus for its lifespan and does not recover. The infective insects do not get sick from the virus, they are just carriers.

As far as the interaction between the insects and the plants, an infected insect vector can only infect a susceptible plant. The only way for the vector to become infected is through coming in contact with an infected plant. The interaction between vector and plant is of predator-prey Holling type 2 [1].

The following table lists the parameters of the model.

Table 1 The following table gives the parameter values

Parameter	Description	Value
K	Total plant host population	50-1000
N	Total insect vector population	50-100
β	infection rate of plants due to vectors	0.01-0.02
β_1	infection rate of vectors due to plants	0.01-0.02
α	saturation constant of plants due to vectors	0.01
α_1	saturation constant of vectors due to plants	0.02
μ	natural death rate of plants	0-0.1
m	natural death rate of vectors	0-0.5
γ	recovery rate of plants	0-0.25
Λ	replenishing rate of vectors (birth and/or immigration)	5
d	death rate of infected plants due to the disease	0.1

Figure 1 is a flow diagram for the interactions.

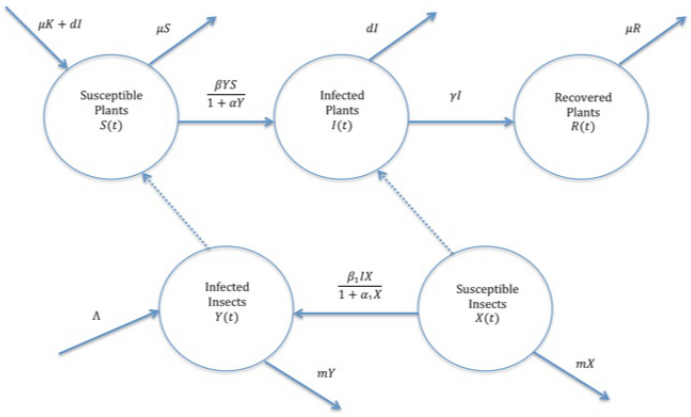

Figure 1: Flow diagram of the interactions

In the flow diagram, the solid lines represents an individual moving from one class to the next. Whereas the dashed line indicates contact between the two classes.

The differential equations describing the interactions between the three plant populations and the two vector ones as given in Figure 1 and considering Holling type II interactions between the plants and the vectors are

$$\begin{aligned}
\frac{dS}{dt} &= \mu K + dI - \mu S - \frac{\beta Y}{1+\alpha Y} S \\
\frac{dI}{dt} &= \frac{\beta Y}{1+\alpha Y} S - (d+\mu+\gamma) I \\
\frac{dR}{dt} &= \gamma I - \mu R \\
\frac{dX}{dt} &= \Lambda - \frac{\beta_1 I X}{1+\alpha_1 I} - mX \\
\frac{dY}{dt} &= \frac{\beta_1 I X}{1+\alpha_1 I} - mY.
\end{aligned}$$

Notice that adding $\frac{dX}{dt}$ and $\frac{dY}{dt}$ yields

$$\frac{dN}{dt} = \Lambda - mN,$$

where $N = X + Y$ and as $t \to \infty$, $N \to \frac{\Lambda}{m}$.

And since the plant and vector populations are constant, we can consider a reduced system of differential equations

$$\begin{aligned}
\frac{dS}{dt} &= \mu(K - S) - \frac{\beta Y}{1 + \alpha Y} S + dI \\
\frac{dI}{dt} &= \frac{\beta Y}{1 + \alpha Y} S - \omega I \\
\frac{dY}{dt} &= \frac{\beta_1 I}{1 + \alpha_1 I} (\frac{\Lambda}{m} - Y) - mY,
\end{aligned} \tag{1}$$

where $\omega = d + \mu + \gamma$

2.1. Analysis of Model

First we study the equilibrium solutions of the subsystem of equations (2). Consider

$$\frac{dx}{dt} = f(x(t), \eta), \tag{2}$$

where $x(t) \in \mathbb{R}^n, f : \mathbb{R}^n \times \mathbb{R}^p \mapsto \mathbb{R}^n$ is a nonlinear smooth function depending on a number of parameters $\eta \in \mathbb{R}^p$. The point $x^* \in \mathbb{R}^n$ is an equilibrium point for the ODE if

$$f(x^*, \eta) = 0,$$

for all t.

By setting the system of equations equal to zero and solving for S, I, and Y we get 2 sets of equilibrium points:

$$S^* = K, I^* = 0, R^* = 0, X^* = N, Y^* = 0$$

and

$$
\begin{aligned}
S^* &= \frac{\omega(\alpha\beta_1 K\Lambda\mu + m(m\omega + K\mu(\beta_1 + \alpha_1 m) - dm))}{\beta_1\beta\Lambda(\mu+\gamma) + \alpha\beta_1\Lambda\mu\omega + \beta_1 m\mu\omega + \alpha_1 m^2\mu\omega} \\
I^* &= \frac{\beta_1\beta K\Lambda\mu - m^2\mu\omega}{\beta_1\beta\Lambda(\mu+\gamma) + \alpha\beta_1\Lambda\mu\omega + \beta_1 m\mu\omega + \alpha_1 m^2\mu\omega} \\
R^* &= K - S^* - I^* \\
X^* &= N - Y^* \\
Y^* &= \frac{\mu(\beta\beta_1 K\Lambda - \omega m^2)}{m(\alpha m\mu\omega + \beta(m\omega + K\mu(\beta_1 + \alpha_1 m) - dm))}.
\end{aligned}
$$

To study the stability of the equilibrium points we will use the Basic Reproduction Number, R_0 [4, 17].

$$
R_0 \sim \left(\frac{infection}{contact}\right) \times \left(\frac{contact}{time}\right) \times \left(\frac{time}{infection}\right).
$$

More specifically:

$$
R_0 = \Gamma \times \overline{c} \times D,
$$

where Γ is the transmissibility (i.e., probability of infection given contact between a susceptible and infected individual), $\overline{c}$ is the average rate of contact between susceptible and infected individuals, and D is the duration of infectiousness.

If $R_0 > 1$ then the disease will propagate, otherwise the disease will eventually die and a fraction of the population will escape infection.

2.2. Next Generation Matrix Method

A next generation matrix G consists of two parts: F and V^{-1}, where

$$
F = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right]
$$

and

$$V = \left[\frac{\partial V_i(x_0)}{\partial x_j} \right].$$

The F_i are the new infections, while the V_i transfers of infections from one compartment to another. x_0 is the disease-free equilibrium state.

R_0 is the dominant eigenvalue of the matrix $G = FV^{-1}$. Calculations give

$$F = \begin{bmatrix} 0 & \beta K \\ \beta\Lambda/m & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} \omega & 0 \\ 0 & m \end{bmatrix}.$$

Thus the Next Generation Matrix is

$$FV^{-1} = \begin{bmatrix} \frac{\beta\beta_1\Lambda K}{m^2\omega} & \frac{\beta K}{m} \\ \frac{\beta_1\Lambda}{m} & 0 \end{bmatrix}.$$

Therefore, the basic reproduction number is

$$R_0 = \sqrt{\frac{\beta\beta_1\Lambda K}{m^2\omega}}$$

The linearized stability of an equilibrium point of a systems of ordinary differential equations (2) can be found by considering the Jacobian matrix J of f, which is an $n \times n$ matrix,

and is defined as follows:

$$J = \begin{pmatrix} \frac{\partial f_1(x^*)}{\partial x_1} & \frac{\partial f_1(x^*)}{\partial x_2} & \cdots & \frac{\partial f_1(x^*)}{\partial x_n} \\ \frac{\partial f_2(x^*)}{\partial x_1} & \frac{\partial f_2(x^*)}{\partial x_2} & \cdots & \frac{\partial f_2(x^*)}{\partial x_n} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\partial f_n(x^*)}{\partial x_1} & \frac{\partial f_n(x^*)}{\partial x_2} & \cdots & \frac{\partial f_n(x^*)}{\partial x_n} \end{pmatrix}$$

If all of the eigenvalues of the Jacobian have a negative real part, then the equilibrium point is locally stable. However if an eigenvalue has a positive part, then the point is unstable.

For (2) the Jacobian is :

$$J = \begin{bmatrix} -\mu - \frac{\beta Y^*}{1+\alpha Y^*} & d & -\frac{\beta S^*}{(1+\alpha Y^*)^2} \\ \frac{\beta Y^*}{1+\alpha Y^*} & -\omega & \frac{\beta S^*}{(1+\alpha Y^*)^2} \\ 0 & (\frac{\beta_1}{(1+\alpha_1 I^*)^2}(\frac{\Lambda}{m} - Y^*)) & \frac{-\beta_1 I^*}{1+\alpha_1 I^*} - m \end{bmatrix}.$$

The eigenvalues for the disease free equilibrium point

$$S^* = K, I^* = 0, Y^* = 0$$

are

$$\lambda = -\mu$$

$$\frac{-m^2 - m\omega \pm \sqrt{4m\beta_1\beta K\Lambda + m^4 - 2m^3\omega + m^2\omega^2}}{2m}.$$

As expected, if $R_0 < 1$ then all eigenvalues are negative thus the equilibrium is stable, if $R_0 > 1$ then at least one eigenvalue is positive thus the equilibrium is unstable.

The eigenvalues for the endemic equilibrium are much harder to calculate and have very

long expressions. However, it can be shown numerically that for specific parameter values, that when $R_0 > 1$ the system is stable according to the eigenvalues but unstable when $R_0 < 1$.

Notice that this ODE model does not take into consideration the time it takes for the virus to spread throughout the plant or insect vector. So in order to account for this biological process, we introduce a couple of delays to the model.

3. Delay Differential Equations

Since it takes time for the virus to enter the plant cells and to spread in the plant, and it takes time for the virus to infect the insect, we introduce delays to the system. We consider two discrete delays,

τ_1, which is time it takes a plant to become infected after contagion and τ_2, the time it takes a vector to become infected after contagion.

The model with the two discrete delays is

$$\begin{aligned}
\frac{dS}{dt} &= \mu(K - S) - \frac{\beta Y(t-\tau_1)}{1+\alpha Y(t-\tau_1)} S(t-\tau_1) + dI \\
\frac{dI}{dt} &= \frac{\beta Y(t-\tau_1)}{1+\alpha Y(t-\tau_1)} S(t-\tau_1) - \omega I \\
\frac{dY}{dt} &= \frac{\beta_1 I(t-\tau_2)}{1+\alpha_1 I(t-\tau_2)} (\frac{\Lambda}{m} - Y(t-\tau_2)) - mY
\end{aligned} \tag{3}$$

Consider a general system of delay differential equations

$$\frac{dx}{dt} = f(x(t), x(t-\tau_1), \ldots, x(t-\tau_m), \eta),$$

where $x(t) \in \mathbb{R}^n, f : \mathbb{R}^{n(m+1)} \times \mathbb{R}^p \mapsto \mathbb{R}^n$ is a nonlinear smooth function depending on a

number of parameters $\eta \in \mathbb{R}^p$, and delays $\tau_i > 0, i = 1, \ldots, m$.

The linearization around a solution $x^*(t)$ gives the *variational equation*

$$\frac{dy}{dt} = A_0(t)y(t) + \sum_{i=1}^{m} A_i(t)y(t - \tau_i),$$

with

$$A_i(t) = \frac{\partial f}{\partial x_i}|_{(x^*(t),x^*(t-\tau_1),\ldots,x^*(t-\tau_m))}$$

and $f \equiv f(x^0, x_1, \ldots, x_m, \eta)$

If $x^*(t)$ is a steady state solution:

$$x^*(t) \equiv x^* \in \mathbb{R}^n \text{with} f(x^*, x^*, \ldots, x^*, \eta) = 0,$$

then $A_i(t) = A_i$ are constant, and we have the characteristic equation [10]

$$\det(\Delta(\lambda)) = 0,$$

with

$$\Delta(\lambda) = \lambda I - A_0 - \Sigma_{i=1}^{m} A_i e^{-\lambda\tau_i}.$$

The characteristic equation has an infinite number of roots $\lambda \in \mathbb{C}$, which determine the stability of the steady state solution. The steady state solution is (asymptotically) stable if all roots have negative real part. It is unstable if there exists a root with positive real part

The Jacobian of the system of the system of DDEs (3) is

$$J = \begin{bmatrix} -\mu - \frac{\beta Y^*}{1+\alpha Y^*} e^{-\lambda\tau_1} & d & -\frac{\beta S^*}{(1+\alpha Y^*)^2} e^{-\lambda\tau_1} \\ \frac{\beta Y^*}{1+\alpha Y^*} e^{-\lambda\tau_1} & -\omega & \frac{\beta S^*}{(1+\alpha Y^*)^2} e^{-\lambda\tau_1} \\ 0 & (\frac{\beta_1}{(1+\alpha_1 I^*)^2}(\frac{\Lambda}{m} - Y^*))e^{-\lambda\tau_2} & \frac{-\beta_1 I^*}{1+\alpha_1 I^*} e^{-\lambda\tau_2} - m \end{bmatrix}. \tag{4}$$

The characteristic equation is

$$\Delta(\lambda) = \frac{1}{m(1+\alpha)^2(1+\alpha_1 I^*)^2} e^{-\lambda(\tau_1+\tau_2)} (\beta_1\beta(\mu+\lambda)S^*(\Lambda - mY^*)$$

$$-(1+\alpha_1 I^*)m(-\beta I^* + e^{\lambda\tau_2}(1+\alpha_1 I^*)(m+\lambda)(1+\alpha Y^*)(-\beta_p d Y^*$$

$$+(\lambda+\omega)(\beta_p Y^* + e^{\lambda\tau_1}(\mu+p)(1+\alpha Y^*)))))$$

Solving $\Delta(\lambda) = 0$ for λ is not possible analytically, but it is possible to study the stability for particular values of the parameters using numerical methods.

4. Numerical Methods

Most methods for ordinary differential equations can be modified for delay differential equations [3, 15]. A very good numerical solver based on Runge-Kutta methods is given in [15].

Figure 2 shows the susceptible and infective plant populations when $R_0 < 1$. Figure 3 shows the recovered plant population. The calculations were done using the following values of the parameters: $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01, \beta_1 = .01, \mu = .01, \Lambda = 5, m = .5, d = .2, \gamma = .01$. The delay for the plants was taken larger than for the insects since the plants fight the infection and it takes time for the infection to spread over the plant as opposed to the vectors where the virus has a smaller area to cover.

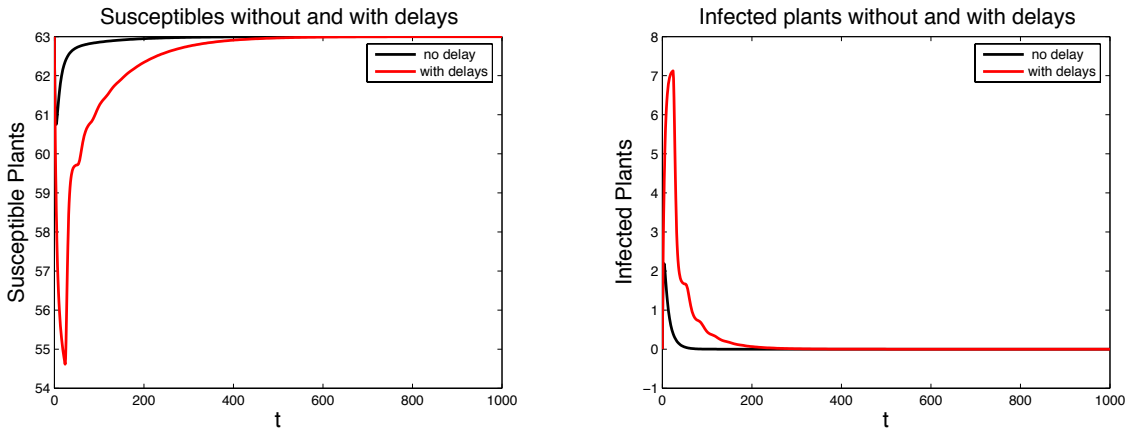

Figure 2: ODE and DDE solutions of susceptible (left) and infected(right) plants when $R_0 < 1$. $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01,\ \beta_1 = .01, \mu = .01, \Lambda = 5, m = .5, d = .2, \gamma = .01$

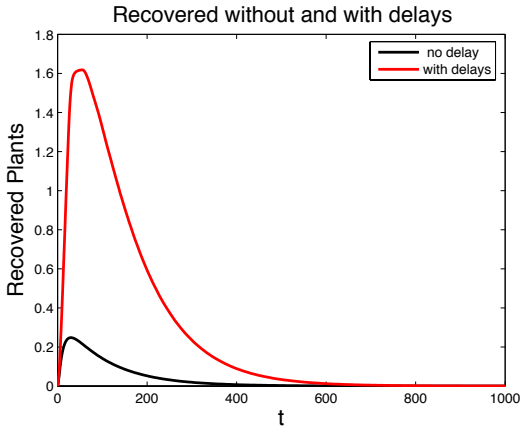

Figure 3: ODE and DDE solutions of recovered plants when $R_0 < 1$. $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01,\ \beta_1 = .01, \mu = .01, \Lambda = 5, m = .5, d = .2, \gamma = .01$

Figure 4 shows the corresponding plots for the susceptible and infective populations of the vectors for the same values of the parameters and $R_0 < 1$.

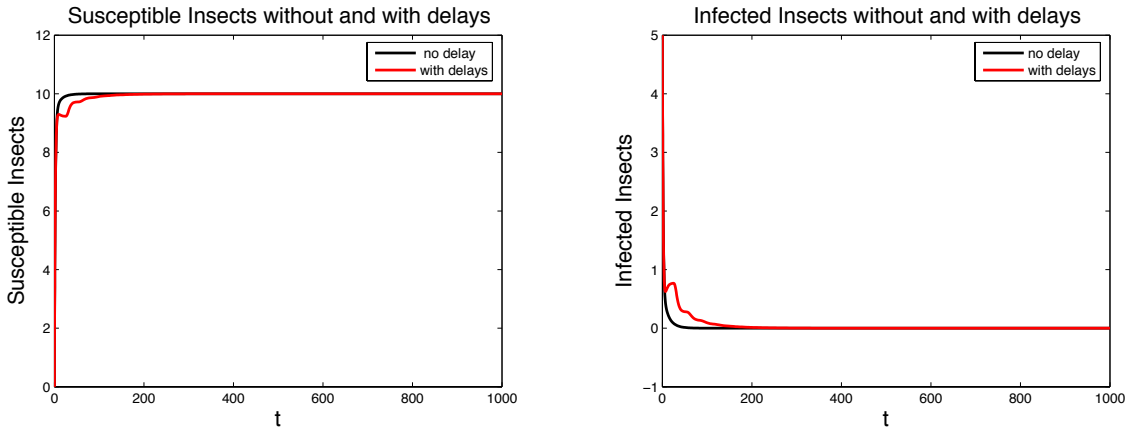

Figure 4: ODE and DDE solutions of susceptible and infective vectors when $R_0 < 1$. $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01,\ \beta_1 = .01, \mu = .01, \Lambda = 5, m = .5, d = .2, \gamma = .01$

For the following values of the parameters, $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01,\ \beta_1 = .01, \mu = .01, \Lambda = 5, m = .32, d = .2, \gamma = .01,\ R_0 > 1$ and therefore the disease free equilibrium point is unstable. Figures 5, 6 and 7 show the susceptible, infective and recovered plant populations and the susceptible and infective vector populations, respectively.

We varied the value of m, the natural death rate of the vector, since in real life that is one rate that can be changed by the use of insecticides of predators. Furthermore, the results show that it is not necessary to eliminate the vector completely, only to increase its death rate to control an epidemic.

The study of the stability of both equilibrium points of the delay differential equation system (3), has to be done numerically. We used the code *dde-biftool* [6], which also allows the variation of some parameters and will approximate the location of bifurcation points. Figure 8 shows the largest eigenvalues of the Jacobian (4) evaluated at the disease free equilibrium point for two values of m, the natural death rate of the vectors. The left figure is for $m = .32$ and the right for $m = .5$. For the lower value of m the point is unstable with one eigenvalue having a positive real part, and stable for the larger value of

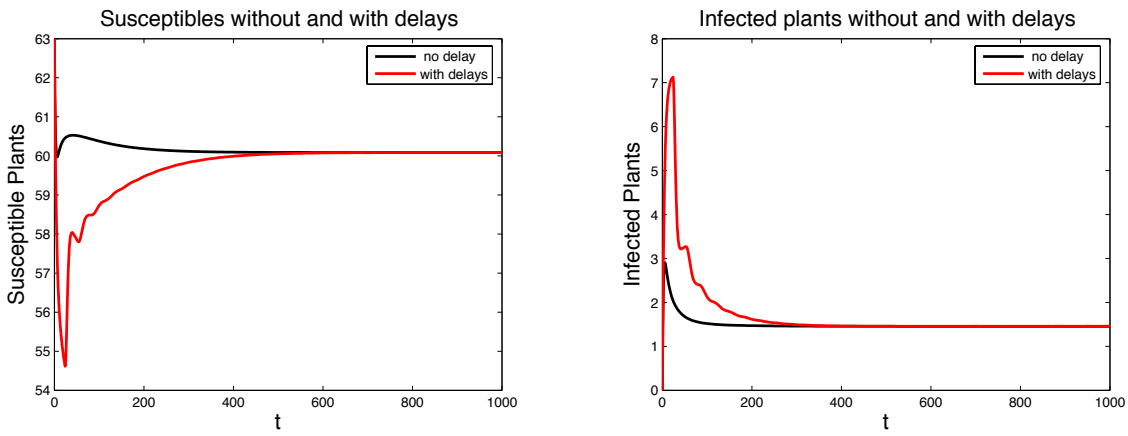

Figure 5: ODE and DDE solutions of susceptible plants (left) and infected plants (right) when $R_0 > 1$. $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01$ $\beta_1 = .01, \mu = .01, \Lambda = 5, m = .32, d = .2, \gamma = .01$

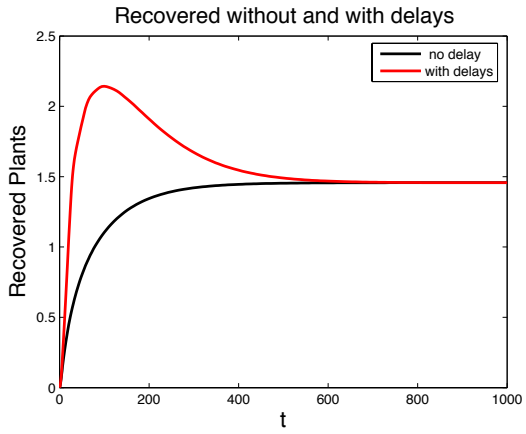

Figure 6: ODE and DDE solutions of recovered plants when $R_0 < 1$. $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01$ $\beta_1 = .01, \mu = .01, \Lambda = 5, m = .32, d = .2, \gamma = .01$

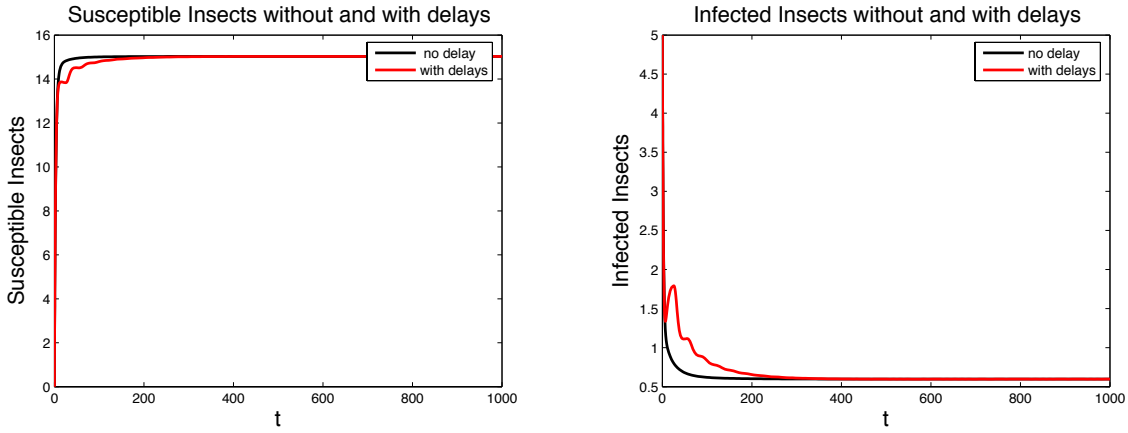

Figure 7: ODE and DDE solutions of susceptible (left) and infected(right) insects when $R_0 < 1$. $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01$ $\beta_1 = .01, \mu = .01, \Lambda = 5, m = .32, d = .2, \gamma = .01$

m. The other parameter values are $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01,$ $\beta_1 = .01, \mu = .01, \Lambda = 5, d = .2, \gamma = .01$. For $m = .32$ $R_0 = 1.40$ and for $m = .5$ $R_0 = .573$.

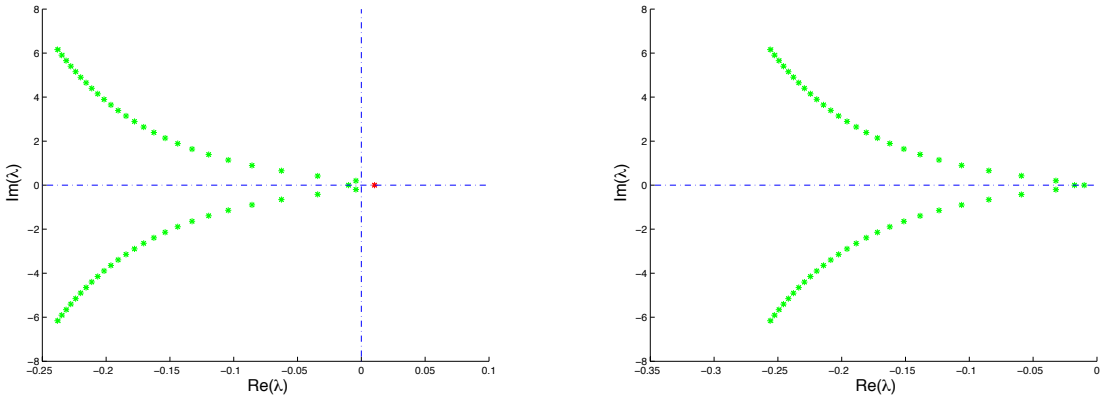

Figure 8: Eigenvalues of the Jacobian evaluated at the disease free equilibrium for two different values of m: $m = .32$ (left), $m = .5$ (right) $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01,$ $\beta_1 = .01, \mu = .01, \Lambda = 5, d = .2, \gamma = .01$

Figure 9 shows the real part of the eigenvalues of the Jacobian evaluated at the disease free branch when m is used as the continuation parameter. The other parameters are as before. Note that for $m < .379$ there is at least one eigenvalue with positive real part so the disease free equilibrium is unstable. For $m > .379$ all the eigenvalues have negative real parts and thus the equilibrium point is stable.

Figure 10 shows the eigenvalues of the Jacobian evaluated at the disease free point, with $m = .2974, \tau_1 = 24$, and $\tau_2 = 1$. There is a pair of eigenvalues with zero real part which corresponds to a Hopf bifurcation, but there is also an eigenvalue with positive real part which makes the point unstable. Also calculations of the Hopf branch gives solutions with negative values for the populations. Disease free branch Hopf bifurcation at $m = .2974, \tau_1 = 24, \tau_2 = 1$

Looking now at the endemic equilibrium point, Figure 11 shows the eigenvalues of the Jacobian matrix at the endemic equilibrium point for the parameter values $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01,$ $\beta_1 = .01, \mu = .01, \Lambda = 5, m = .32, d = .2, \gamma = .01$. All

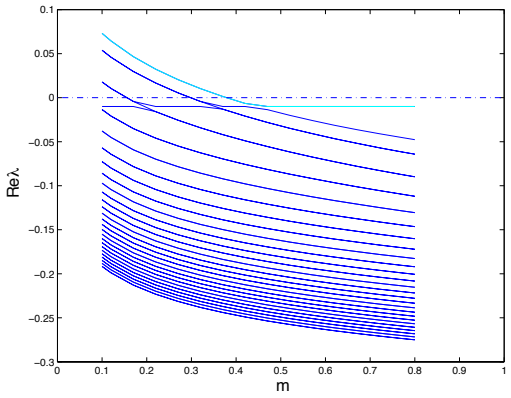

Figure 9: The real part of the eigenvalues of the Jacobian at the disease free branch versus m for $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01$ $\beta_1 = .01, \mu = .01, \Lambda = 5, d = .2, \gamma = .01$

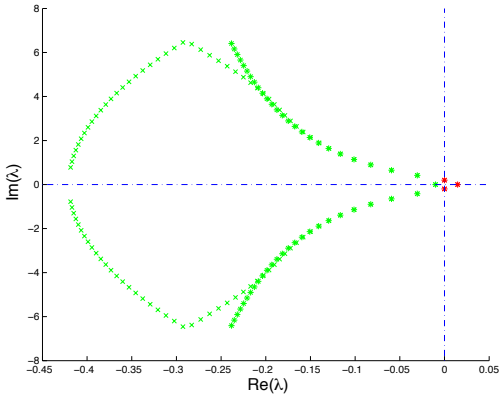

Figure 10: The eigenvalues of the Jacobian evaluated at the disease free equilibrium for $m = .2974$, showing a pair of pure imaginary eigenvalues. The other parameters are and $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01$ $\beta_1 = .01, \mu = .01, \Lambda = 5, d = .2, \gamma = .01$

the eigenvalues have negative real parts so the point is stable. Note that $m = .32$ and the disease free equilibrium point is unstable there.

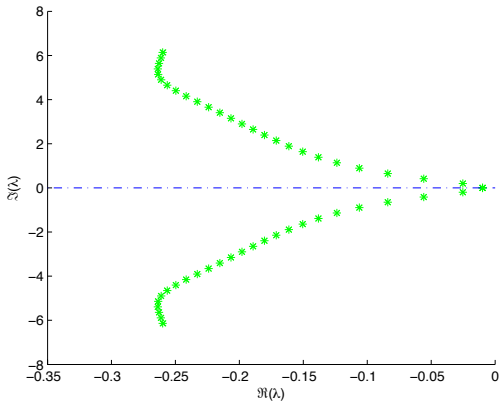

Figure 11: Eigenvalues of the Jacobian at an endemic equilibrium point for $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01$ $\beta_1 = .01, \mu = .01, \Lambda = 5, m = .32, d = .2, \gamma = .01$

Figure 12 shows the real parts of the larger eigenvalues of the Jacobian evaluated at the endemic equilibrium point. As can be seen for $m < .379$ there is at least one eigenvalue with positive real part and thus the solution is unstable. But for $m > .379$ all the real parts of the eigenvalues are negative and thus the solution is stable. Also for $m < .379$, some of the populations of the endemic equilibrium branch are negative, so the branch is not only unstable there but also nonphysical. There is also a Hopf bifurcation point on this branch at $m = .474$ but it also is an unstable point.

We ran simulations for different values of τ_1 and τ_2, and similar results were obtained. In particular, we increased τ_2, the delay time for the virus-insect interaction. The solutions were slightly different but the disease free equilibrium was stable for $R_0 < 1$ and unstable when $R_0 > 1$. Likewise, the endemic equilibrium was stable for $R_0 > 1$ and unstable when $R_0 < 1$.

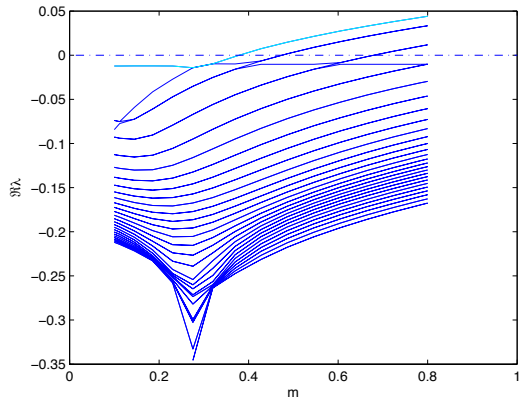

Figure 12: The real part of the eigenvalues of the Jacobian at the endemic branch versus m for $\tau_1 = 24, \tau_2 = 1, K = 63, \alpha = .2, \alpha_1 = .1, \beta = .01\ \beta_1 = .01, \mu = .01, \Lambda = 5, d = .2, \gamma = .01$

5. Conclusion

We presented two plant virus propagation models, one with no delays and the other with two delays. In the case where $R_0 < 1$, we notice that the introduction of delays introduces significant changes in the solution for the susceptible, infected, and recovered plants, including a longer time to approach approach the disease free equilibrium point and oscillations. For the insect populations, there is a smaller change in the solutions. When $R_0 > 1$, however, the endemic steady state becomes stable and the disease free becomes unstable and this also corroborated by the numerical calculations. The natural death rate of the vector m was chosen as the continuation parameter, since it is one value that can be modified by the use of pesticides, predators or other vector control means.

In addition, there is a Hopf bifurcation of the disease free branch at $m = .2974$ when $\tau_1 = 24, \tau_2 = 1$, but the some populations at the bifurcated branch are negative, so the branch is unrealistic. The same is true for the Hopf bifurcation point on the endemic equilibrium branch. So there are no periodic solutions.

Delayed model is more realistic because it takes into account the time between the release of a factor and absorption and its effect.

Although a specific plant, insect vector, and virus was not considered, the parameters can be modified to fit a particular situation.

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Chapter 3
A Model of Biological Control of Plant Virus Propagation with Delays

Mark Jackson[1], Benito M. Chen-Charpentier[1,*]

Abstract

Plants are a food source for man and many species. They also are sources of medicines, fibers for clothes and are essential for a healthy environment. But plants are subject to diseases. Many of these are caused by viruses. In plants most the virus propagation is done by a vector, usually insects that bite infected plants and the susceptible plants. Chemical insecticides are commonly used to control the insects, but unfortunately these chemicals have toxic effects on humans, animals and the environment in general. An alternative is to introduce a new species, or just increase the number of a naturally present one, to prey on the insects and thus control their number. The different steps in the virus propagation process take take. In this paper we add a predator to a plant-virus propagation model with delays. The model is based on delay differential equations. There are five populations: susceptible and infected plants, susceptible and infected vectors, and predators. The infected vectors have no ill effects from the virus so they do not fight the virus and therefore they do not recover. The predators do not get sick from the virus. The total number of plants is assumed to be constant since for crops the framers will replace the death plants and one of the plant populations can be determined using the total constant plant population. Therefore we have a system of five delay ordinary differential equations in five unknowns. We assume that predators are introduced at the initial time and after their growth rate has

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a Holling 2 type dependence on the number of vectors. We consider two delays: the time it takes from the moment a plant is bitten by an infected vector to the moment the plant is infected; and a second smaller delay for the time from the moment a vector bites an infected plant to the moment the vector becomes infected. We determine the non-negative steady state solutions of the system of delay differential equations. We analyze their stability by calculating the eigenvalues of the linearized system about each equilibrium point. We do numerical simulations for certain values of the parameters. Finally we search for bifurcation points numerically using the software package biftool. Conditions are presented for which the disease is controlled or even eliminated. From the practical side, the model can be used to determine the amount of introduced predators necessary to eliminate the epidemic under different conditions.
Keywords: delay differential equations, plant virus propagation, mathematical modeling

1. Introduction

Plants play a vital role in almost every ecosystem on the planet. Sometimes plants may become infected with a virus. These infections can be devastating to not only the plants themselves but also the ecosystem that depends on them. Also, plant virus infections can have a negative impact on the crops necessary for human survival. For example, the cassava plant, which is a staple in many underdeveloped African countries, is susceptible to the cassava mosaic virus. This virus has ravaged plants in Kenya, Uganda and Tanzania [6]. Another serious example of plants that have been infected by viruses are tomato plants in India. These viruses cause tomato leaf curling disease (TLCD). This disease causes the leaves of the plants to curl and possibly become sterile [6].

Viruses need a method of transportation to move from one plant to another. Typically an insect vector this is the mode of transportation. In fact, insects are responsible for 70 percent of all plant virus transmissions [3]. The insect must come in contact with an infected

plant, usually by feeding on it, obtain the virus, and transmit the virus to another healthy plant. *Bemisia tabaci* is the vector that transmits both the cassava mosaic virus and TLCD.

Mathematical models can be used to understand the dynamics of a particular situation. Ordinary differential equations (ODEs) have been used to model plants infected with viruses. In [8], the authors develop a model to combat plant viruses by continuously removing infected plants and replacing them with healthy plants. In [13] a system of ODEs is considered that explicitly models the interaction between plants and insects. Although ODE models can help one understand the interaction between plants, viruses, and vectors, they do not consider the time it takes for a virus to spread within a plant or insect, unless additional equations accounting for the latent class are introduced. Introducing latent equations present some difficulties, however. From a biological point of view, it can be difficult to determine how many plants and insects are in the latent stage since they are not showing symptoms. Mathematically, introducing more equations can make the system harder to analyze. By introducing a delay to the system, we can account for this biological fact without introducting more equations for the model. For example, the authors in [14] introduced a delay to the model in [8] to account for the incubation period of the plants and noticed a change in the dynamics of the model. In [5], the authors modified the model in [13] by including multiple delays to account for the incubation periods of the virus in both the plants and insects. They too noticed changes in the dynamics in the system, specifically changes in the solutions. From a mathematical perspective, delays change the solution, may change the stability of steady solutions and may introduce discontinuities in the derivatives [1].

There are many different ways to combat the disease. One could try to breed a plant that is resistant to the virus and replant infected plants with the resistant ones. Another way is the use of pesticides. However, there are some drawbacks to the two methods. To breed a plant that is resistant to the virus, such a plant must first be discovered. For pesticides, too much can be harmful to the plants and to the environment as well. Another method is

to introduce a predator to the environment to feed upon the insects. This alternative can be more environmentally friendly compared to the pesticides. Predators may not be present naturally or their number may not be enough to control the vectors. We will explore the effects of introducing a predator to the system or increasing its number.

In this paper, we first discuss our modeling assumptions. Then we construct a system of ordinary differential equations modeling the interaction between the plant hosts, the insect vectors, and the predators of the vectors. Afterwards, a stability analysis is performed on the system. A couple of delays accounting for the time it takes for a plant and vector to become infected by the virus are introduced to the system. We use a programming software biftool to numerically approximate eigenvalues and bifurcation points of the delay differential equations (DDE's). Afterwards, we introduce a predator at a constant rate to the model and we analyze the dynamics. Results and conclusions are presented .

2. Model Assumptions and System of ODEs

In this paper, we extend the model in [5] to include a predator We consider 6 populations: susceptible plants $S(t)$, infected plants $I(t)$, recovered plants $R(t)$, susceptible insect vectors $X(t)$, infected insect vectors $Y(t)$, and predators $P(t)$. Each variable describes it's respective population at time t. Susceptible plants do not have the disease but could contract the disease if infected with the virus. The infected plants have the virus but cannot directly transmit the virus to susceptible plants. Additionally, since the infected plants can die from the viral infection their death rate is higher than that of plants that do not have the virus. We also assume that as soon as a plant dies either from the infection or from a natural death, it is immediately replaced with a new susceptible plant by a farm worker. Thus it is reasonable to assume that the plant population remains fixed and the total plant population will be denoted by K. This assumption has the modeling advantage that $K = S(t) + I(t) + R(t)$ can be used to eliminate the recovered population from the system of equations. The susceptible

insects do not have the virus but can obtain the virus if they come in contact with a infected plant. Infected insects can transmit the virus to susceptible plants upon contact. We assume no vertical transmission of the virus with neither plants nor vectors. Moreover, we assume that the virus does not harm the vector and thus the vector does not defend against the virus and it retains the virus for the rest of its life. We assume that the predators consume both infected and healthy insects at the same rate. The predators use this energy from feeding on the vectors to grow the predator population. We also assume that even if a predator consumes an infected insect, it will not become infected with the virus. We will also include competition between predators for the insects. Moreover, predators can feed on the infected insects and susceptible insects at different rates, but since we are assuming that the vectors are asymptomatic in the calculations we will use the same rate. The interaction between vector and plant as well as that of predator and vector are assumed to have a limitation of the form of predator-prey Holling type 2.

Table 1 gives a list of the parameters used in the model, their description and their value or range. The parameters were used from the ranges given in [13].

Parameter	Description	Value
K	Total plant host population	63
β	infection rate of plants due to vectors	0.01
β_1	infection rate of vectors due to plants	0.01
α	saturation constant of plants due to vectors	0.2
α_1	saturation constant of vectors due to plants	0.1
μ	natural death rate of plants	.01
m	natural death rate of vectors	.2974
γ	recovery rate of plants	0.01
Λ	replenishing rate of vectors	10
d	death rate of infected plants due to the disease	0.2
c_1	contact rate between predators and healthy insects	0.05
c_2	contact rate between predators and infected insects	0.05
δ	natural death rate of predators	0.05
ϵ	competition constant between predators	0.01
α_3	saturation of predators due to insects	0.1
α_4	conversion rate of predators due to insects	0.1

$$
\begin{aligned}
\frac{dS}{dt} &= \mu(K - S) + dI - \frac{\beta Y}{1 + \alpha Y} S \\
\frac{dI}{dt} &= \frac{\beta Y}{1 + \alpha Y} S - \omega I \\
\frac{dX}{dt} &= \Lambda - \frac{\beta_1 I}{1 + \alpha_1 I} X - \frac{c_1 X}{1 + \alpha_3 X} P - mX \\
\frac{dY}{dt} &= \frac{\beta_1 I}{1 + \alpha_1 I} X - \frac{c_2 Y}{1 + \alpha_3 Y} P - mY \\
\frac{dP}{dt} &= \frac{\alpha_4 c_1 X}{1 + \alpha_3 X} P + \frac{\alpha_4 c_2 Y}{1 + \alpha_3 Y} P - \delta P - \epsilon P^2
\end{aligned} \tag{1}
$$

where $\omega = d + \mu + \gamma$

2.1. Analysis of Model

The system of equations 1 can be written as

$$\frac{dx}{dt} = f(x(t), \eta),$$

where $x(t) \in \mathbb{R}^5$ is the vector of the 5 populations, $f : \mathbb{R}^5 \times \mathbb{R}^p \mapsto \mathbb{R}^n$ is the nonlinear smooth right-hand side function depending on a number of parameters $\eta \in \mathbb{R}^p$. The point $x^* \in \mathbb{R}^n$ is an equilibrium point for the ODE if

$$f(x^*, \eta) = 0,$$

for all t.

By setting the system of equations equal to zero and solving for S, I, X, Y, and P we get one of equilibrium point that is easy to analyze:

$$S^* = K, I^* = 0, R^* = 0, X^* = \frac{\Lambda}{m}, Y^* = 0, P^* = 0.$$

This point is usually referred as the disease-free equilibrium. There are other equilibrium points but due to the complexity of their formulas it is not possible to determine whether they make physical sense.

The local stability of the equilibrium points of the system of ODE's is established by linearizing the system about the equilibrium point and determining whether the eigenvalues of the Jacobian matrix have positive real parts. For the disease-free equilibrium we found that it is stable when $\frac{m^2\omega}{K\Lambda} > 1$ (the real part of all the eigenvalues is negative) and unstable when $\frac{m^2\omega}{K\Lambda} < 1$.

The other equilibrium points and their stability needs to be studied numerically for given values of the parameters and results are presented later.

3. Delay Differential Equations

After an infected vector bites a susceptible plant, it takes time for the virus to enter the plant cells, to replicate and to spread in the plant, and since it also takes time for the virus to infect a susceptible insect after it bites an infected plant, we introduce delays to the system. In particular, we consider two discrete delay,

τ_1, which is time it takes a plant to become infected after contagion and τ_2, the time it takes a vector to become infected after contagion. τ_1 is much larger than τ_2 since the virus needs to penetrate the plant cells, replicate and spread throughout the plant. In the vector the virus does not replicate and usually stays only around the jaws of the insect.

The model with the two discrete delays is

$$
\begin{aligned}
\frac{dS}{dt} &= \mu(K-S) + dI - \frac{\beta Y(t-\tau_1)}{1+\alpha Y(t-\tau_1)} S(t-\tau_1) \\
\frac{dI}{dt} &= \frac{\beta Y(t-\tau_1)}{1+\alpha Y(t-\tau_1)} S - (d+\mu+\gamma) I \\
\frac{dX}{dt} &= \Lambda - \frac{\beta_1 I(t-\tau_2)}{1+\alpha_1 I(t-\tau_2)} X(t-\tau_2) - \frac{c_1 X}{1+\alpha_3 X} P - mX \\
\frac{dY}{dt} &= \frac{\beta_1 I(t-\tau_2)}{1+\alpha_1 I(t-\tau_2)} X(t-\tau_2) - \frac{c_2 Y}{1+\alpha_3 Y} P - mY \\
\frac{dP}{dt} &= \frac{\alpha_4 c_1 X}{1+\alpha_3 X} P + \frac{\alpha_4 c_2 Y}{1+\alpha_3 Y} P - \delta P - \epsilon P^2
\end{aligned}
\tag{2}
$$

System 2 can also be written as a general system of delay differential equations

$$
\frac{dx}{dt} = f(x(t), x(t-\tau_1), \ldots, x(t-\tau_m), \eta),
$$

where $x(t) \in \mathbb{R}^n, f : \mathbb{R}^{n(m+1)} \times \mathbb{R}^p \mapsto \mathbb{R}^n$ is a nonlinear smooth function depending on a number of parameters $\eta \in \mathbb{R}^p$, and delays $\tau_i > 0, i = 1, \ldots, m$. For system 2 $n = 5$ and $m = 2$.

The linearization around a solution $x^*(t)$ gives the *variational equation*

$$
\frac{dy}{dt} = A_0(t)y(t) + \sum_{i=1}^{m} A_i(t)y(t-\tau_i),
$$

with

$$
A_i(t) = \frac{\partial f}{\partial x_i}|_{(x^*(t), x^*(t-\tau_1), \ldots, x^*(t-\tau_m))}
$$

and $f \equiv f(x^0, x_1, \ldots, x_m, \eta)$

If $x^*(t)$ is a steady state solution:

$$
x^*(t) \equiv x^* \in \mathbb{R}^n \text{with} f(x^*, x^*, \ldots, x^*, \eta) = 0,
$$

then $A_i(t) = A_i$ are constant, and we have the characteristic equation [7]

$$\det(\Delta(\lambda)) = 0,$$

with

$$\Delta(\lambda) = \lambda I - A_0 - \Sigma_{i=1}^{m} A_i e^{-\lambda\tau_i}.$$

The characteristic equation has an infinite number of roots $\lambda \in \mathbb{C}$, which determine the stability of the steady state solution. The steady state solution is (asymptotically) stable if all roots have negative real part. It is unstable if there exists a root with positive real part.

We can construct a Jacobian matrix for our system of delay equations and determine a characteristic equation using the disease free equilibria. However, it is very difficult to determine the eigenvalues from the equation. It is also troublesome to determine the endemic equilibria analytically because of the number of parameters in our system. This, in turn, is problematic when performing a stability analysis on the endemic equilibria. Therefore, we will run numerical simulations for particular values to see how the system behaves. We will use them to determine the equilibria values and we will investigate the stability of these equilibria using numerical simulations.

4. Numerical Methods

Most methods for ordinary differential equations can be modified for delay differential equations [1, 12].A very good numerical solver based on Runge-Kutta methods is given in [12]. In our numerical simulations we use the following values for our parameters: K=63, β=0.01, β_1= 0.01, α=0.2, α_1 =0.1, μ=.01, m=.2974, γ=0.01, Λ=10, d=0.2, c_1=0.05, c_2=0.05, δ=0.05, ϵ= 0.01, α_3= 0.1, α_4=0.1. Also, the initial conditions for S, I, X, Y, and P are as follows $S(0) = 59.8478$, $I(0) = 1.57612$, $X(0) = 14.6247478$, $Y(0) = 19.5$, $P(0) = 2$. The values of the delays used are $\tau_1 = 24$ and $\tau_2 = 1$. The history for the delay equations is $S_h = 59.8478$,

$I_h = 1.57612$, $X_h = 14.6247478$, $Y_h = 19.5$, $P_h = 2$. Since we are mostly concerned with the stability of the equilibrium points, we use constant history because it is simplest that can be done. We could use the data on the interval [0,24] (24 being the max delay) but it would only shift the solution. Another possibility would be to start with a few infected insects in the history and make the infected insects 0 at t=0 and see what happens on [0,24].

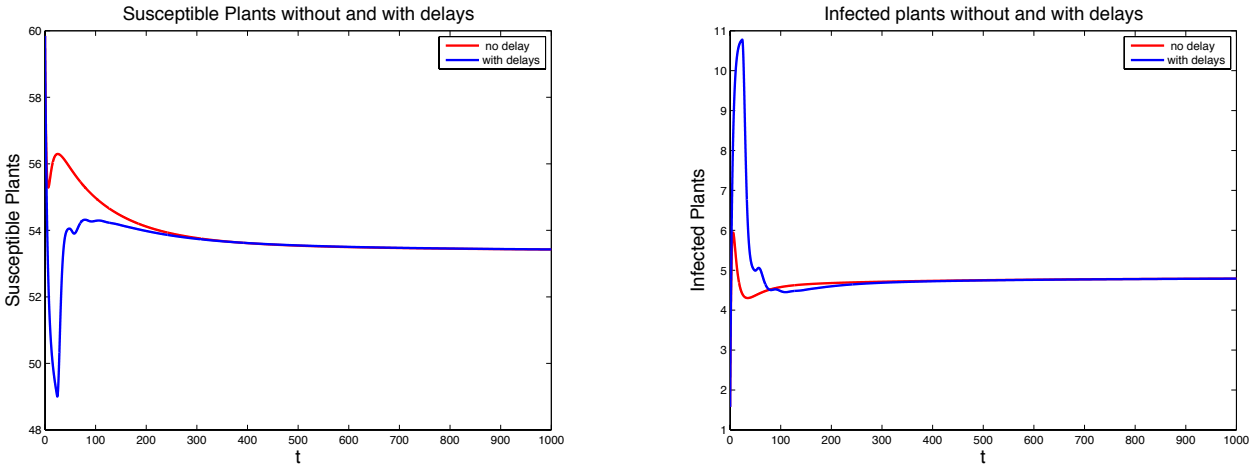


Figure 1: ODE and DDE solutions of susceptible (left) and infected (right) plants

Figure 1 shows the numerical solutions for susceptible (S) and infected plants (I) with and without delays. We notice that the virus is still prevalent within the plants since $S \to 53.33$ and $I \to 4.83499$ as $t \to \infty$ for both the system of ODEs and DDEs. This implies that for the given parameters we have an endemic equilibrium. Also, the solution of the DDEs yield a a lower minimum of susceptible plants and a a higher maximum of infected plants compared to their ODE counterparts.

Figure 2 displays the numerical solutions for susceptible (X) and infected insects (Y) with and without delays. We observe that the virus is also present in the insect population since $X \to 30.2981$ and $Y \to 3.31825$ as $t \to \infty$. These equilibrium values are the same for both ODE and DDE systems. We also notice that the ODE and DDE solutions for the susceptible and infected insects are almost identical except for when $10 \leq t \leq 80$. In which

case, the values for the susceptible insects are lower in the DDE solution and the infected insects are higher in the DDE solution.

Figure 3 exhibits the numerical solutions for the predators with and without delays. We notice that the solutions for both ODE and DDE yield almost identical solutions. Also, the predators are almost eliminated from the system since $P \to 0.00499883$.

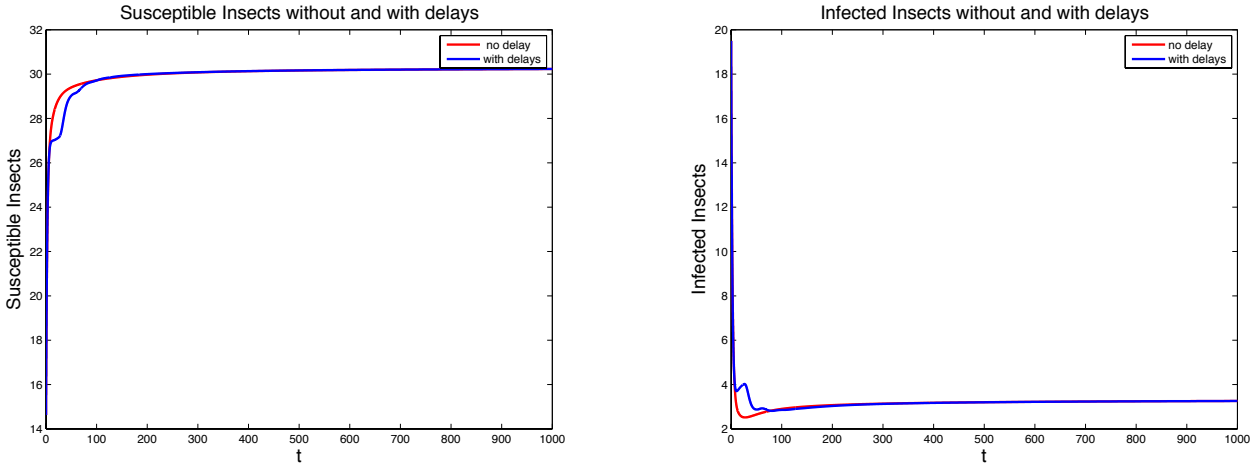


Figure 2: ODE and DDE solutions of susceptible (left) and infected (right) insects.

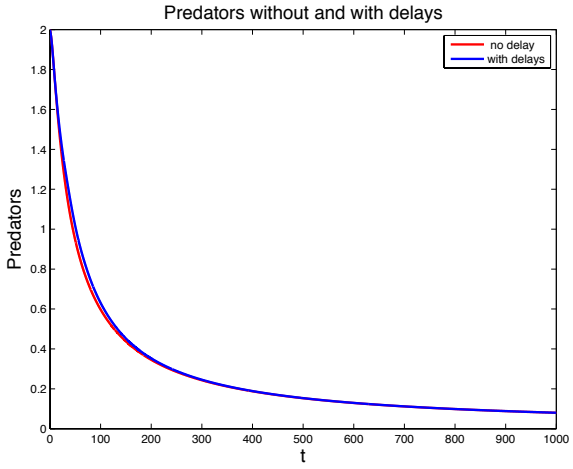


Figure 3: ODE and DDE solutions of predators

For the system delay differential equations, the stability of the equilibrium points (**??**), has to be done numerically. We used the code *dde-biftool* [2]. This program will approximate

the eigenvalues of a system of delay differential equations. It also allows the variation of some parameters and will approximate the location of bifurcation points. The following figures display some results.

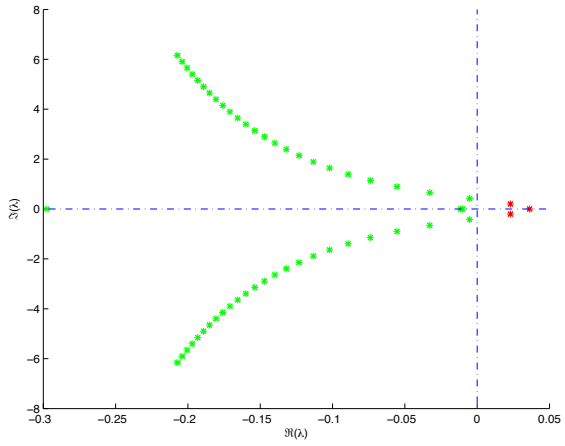


Figure 4: Eigenvalues when equilibria values are $S = K, I = 0, X = \frac{\Lambda}{m}, Y = 0, P = 0$

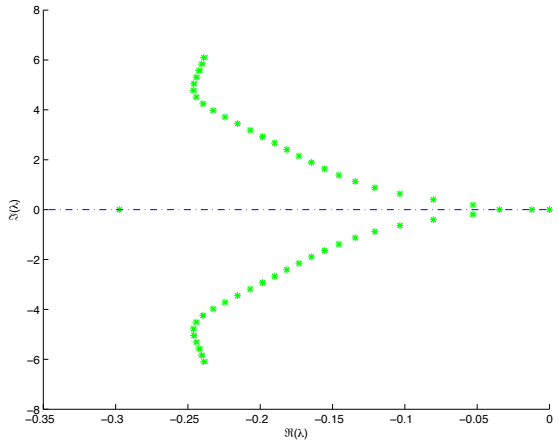


Figure 5: Eigenvalues when equilibria values are $S = 53.330, I = 4.835, X = 30.298, Y = 3.3182, P = 0.0050$

Figure 4 shows the numerical approximations of the eigenvalues for the disease free eqililibria with the given values of the parameters. We notice that there is an eigenvalue with a positive real part. This means that the disease free equilibria is unstable for the given

parameters. Figure 5 displays the eigenvalues for the endemic equilibria. We notice that all of the eignevalues have negative real part, thus the endemic equilibria is stable.

5. Introducing a Predator at Constant Rate

The previous numerical solutions showed that the predator is not very effective in combating the disease if the initial population is small. We would like to see what happens if we introduce a certain amount of the predator every day. We will now define a new parameter, Λ_p. Λ_p is the amount of predator that a farm worker will artificially insert into the system each day. It is a constant rate. The system of differential equations stays the same except for the equation modeling the predator.

$$\frac{dP}{dt} = \Lambda_p + \frac{\alpha_4 c_1 X}{1+\alpha_3 X}P + \frac{\alpha_4 c_2 Y}{1+\alpha_3 Y}P - \delta P - \epsilon P^2$$

We now run some simulations for $\Lambda_p = 0.4$ and $\Lambda_p = 0.8$

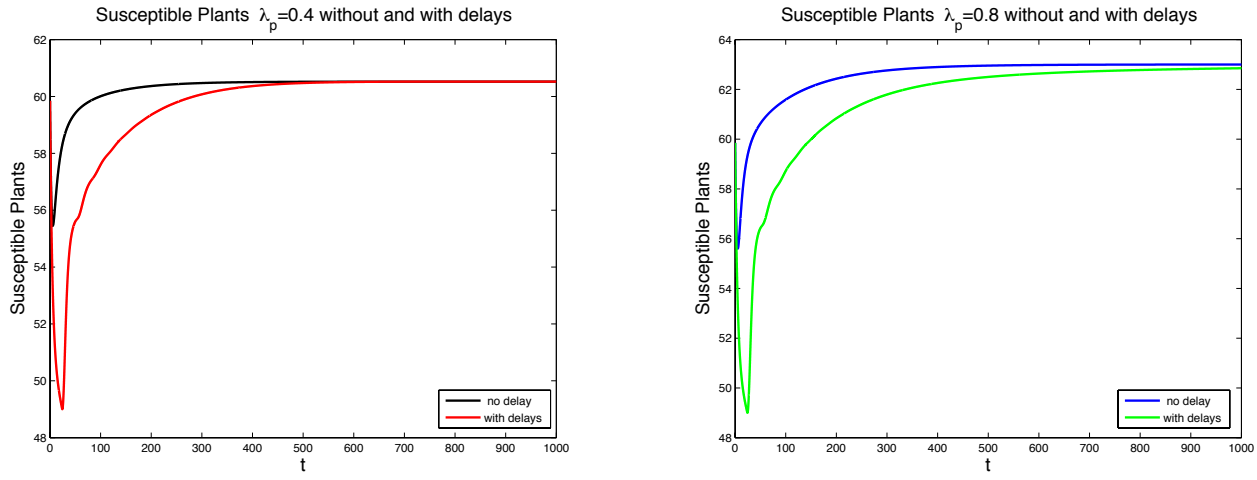
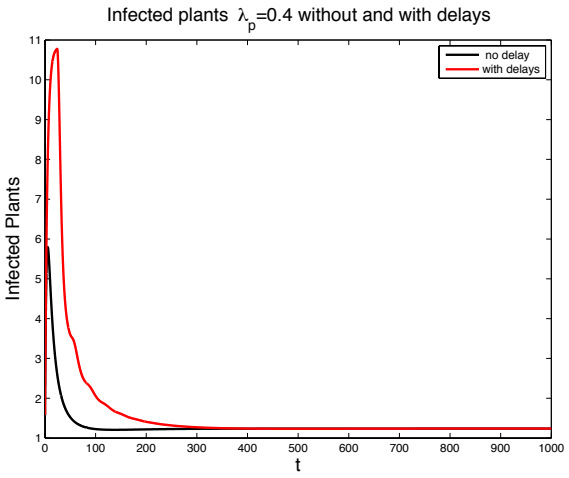


Figure 6: The above figures show the susceptible plant populations for $\Lambda_p = 0.4$ (left) and $\Lambda_p = 0.8$ (right).

Figure 6 displays the numerical solutions for the susceptible plant populations for $\Lambda_p = 0.4$ and $\Lambda_p = 0.8$. We notice that in each case the solutions of the delay equations have lower



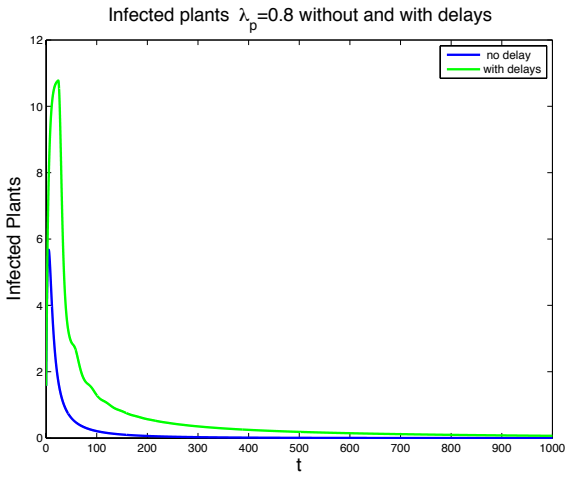
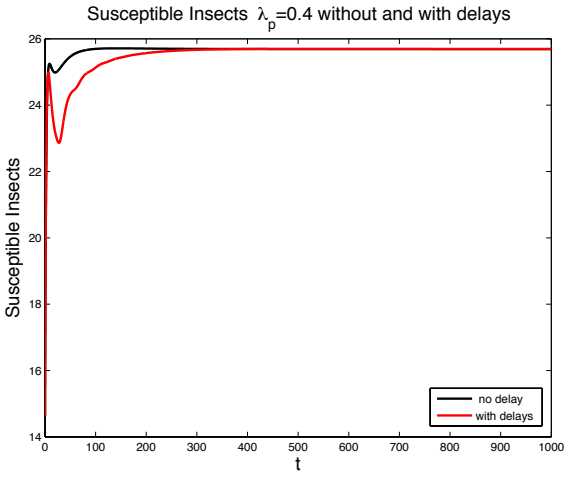


Figure 7: The above figures show the infected plant populations for $\Lambda_p = 0.4$ (left) and $\Lambda_p = 0.8$ (right).



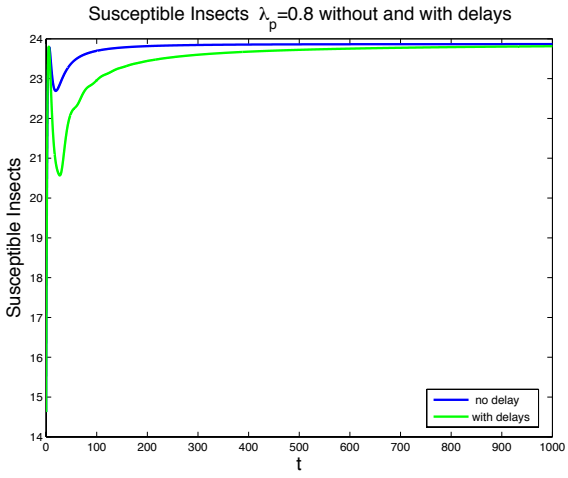
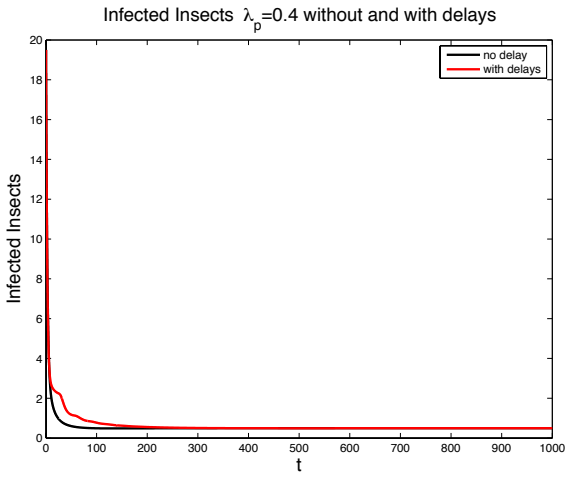


Figure 8: The above figures show the suseptible insect populations for $\Lambda_p = 0.4$ (left) and $\Lambda_p = 0.8$ (right).



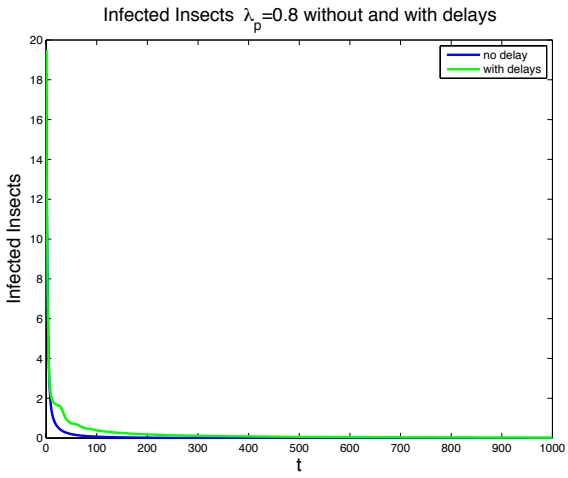
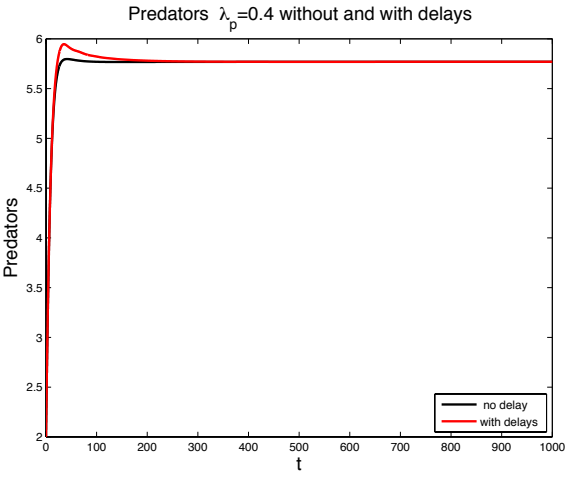


Figure 9: The above figures show the infected insect populations for $\Lambda_p = 0.4$ (left) and $\Lambda_p = 0.8$ (right).



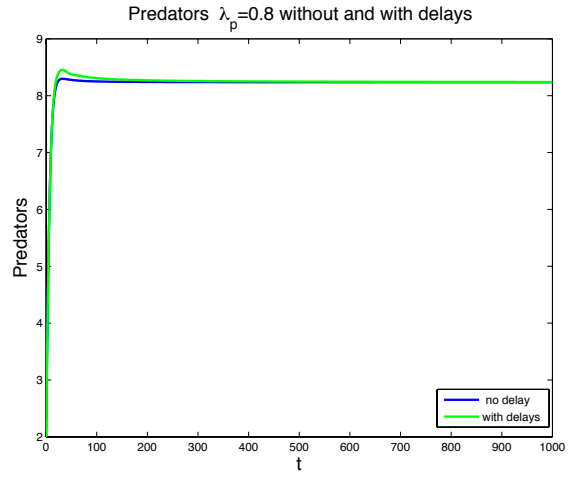


Figure 10: The above figures show the predator populations for $\Lambda_p = 0.4$ (left) and $\Lambda_p = 0.8$ (right).

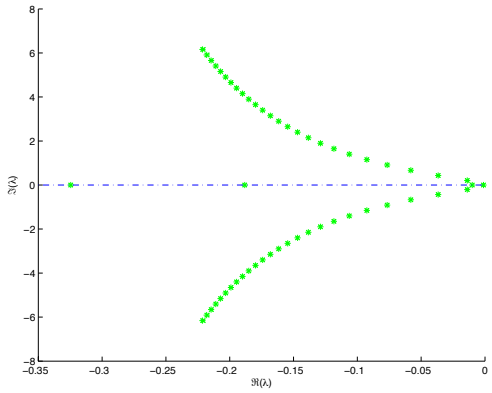


Figure 11: Eigenvalues when equilibrium values are $S = 63, I = 0, X = 23.8662, Y = 0, P = 8.23648$

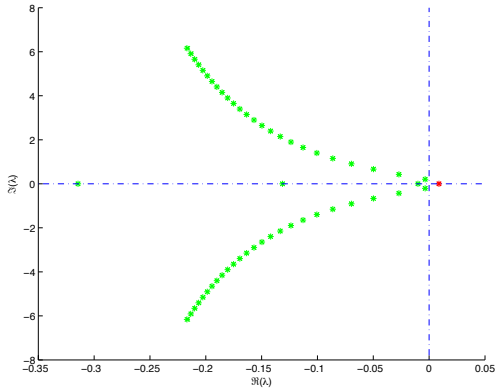


Figure 12: Eigenvalues when equilibrium values are $S = 63, I = 0, X = 26.6793, Y = 0, P = 5.679$

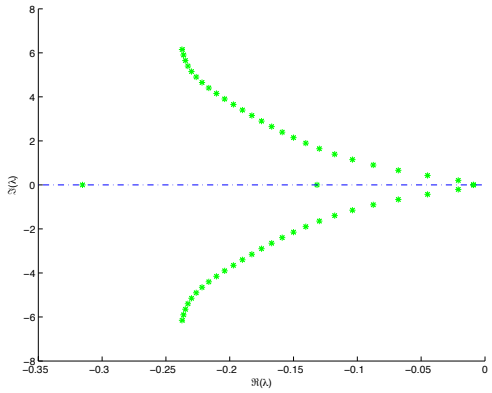


Figure 13: Eigenvalues when equilibrium values are $S = 60.5225, I = 1.23875, X = 25.6913, Y = 0.494852, P = 5.76876$

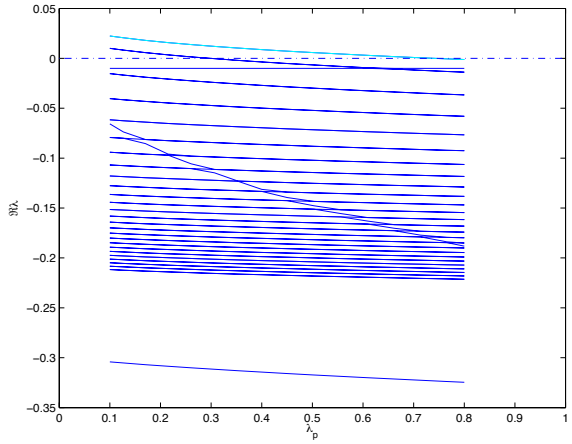


Figure 14: The graph shows the real parts of the larger eigenvalues of the Jacobian evaluated at the disease free equilibrium point.

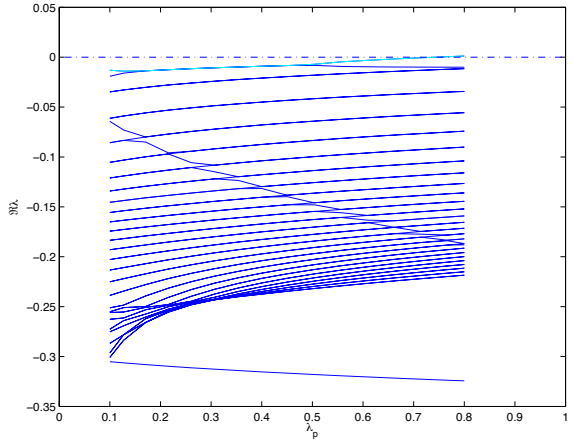


Figure 15: The graph shows the real parts of the larger eigenvalues of the Jacobian evaluated at the endemic equilibrium point.

minimums than the solutions of the ODE equations. However, when $\Lambda_p = 0.4$ the healthy plant population tends to 60.5225 and when $\Lambda_p = 0.8$ it tends to 63 as $t \to \infty$.

Figure 7 compares the infected plant populations when for $\Lambda_p = 0.4$ and $\Lambda_p = 0.8$. Similar to when there was no Λ_p, the delay equations for the infected plant populations yield a higher maximum than the equations with no delay. As $t \to \infty$, the infected population tends to 1.23875 when $\Lambda_p = 0.4$ and 0 when $\Lambda_p = 0.8$. This indicates that when Λ_p is high enough, the disease is elminated from the plants.

Figure 8 show the differences between the susceptible insect populations when $\Lambda_p = 0.4$ and $\Lambda_p = 0.8$. In each case, the solutions increase sharply but then decrease to a local minimum, and then increase again to the equilibrium point. The delay solutions both have a lower relative minimum than their ODE counterparts. When $\Lambda_p = 0.4$, the susceptible insect population tends to 25.6913. However, the healthy plants tend to 26.6793 as $t \to \infty$.

Figure 9 shows the solutions of the ODE and DDE system when $\Lambda_p = 0.4$ and $\Lambda_p = 0.8$. When $\Lambda_p = 0.4$, the infected insects are still present in the system since both solutions tend to 0.494852. In the $\Lambda_p = 0.8$ case, the solutions each tend to 0 as $t \to \infty$. This indicates that the infected insects will be eliminated from the population.

Figure 10 displays the predator population for $\Lambda_p = 0.4$ and $\Lambda_p = 0.8$. In each case the predator population survives. The number of predators in the system when $\Lambda_p = 0.4$ is smaller than the predators when $\Lambda_p = 0.8$ as $t \to \infty$.

Figure 11 shows the eigenvalues for the equilibrium point S = 63, I = 0, X = 23.8662, Y = 0, P = 8.23648 when $\Lambda_p = 0.8$. We notice that the eigenvalues are all negative. This implies that our equilibrium point is stable. If $\Lambda_p = 0.8$, this means that there are enough predators to eliminate the virus from the system.

Figure 12 shows the eigenvalues for the equilibrium point S = 63, I = 0, X = 26.6793, Y = 0, P = 5.679 when $\Lambda_p = 0.4$. Here we notice an eigenvalue with positive real part (in red). This means the equilibrium point is unstable. Biologically, the disease free state does not

occur if $\Lambda_p = 0.4$. This is more evident by the fact that the endemic equilibria S = 60.5225, I = 1.23875, X = 25.6913, Y = 0.494852, P = 5.76876 is stable, since the eigenvalues in figure 13 all have negative real part.

Figure 13 Shows the eigenvalues for the equilibrium point S = 60.5225, I = 1.23875, X = 25.6913, Y = 0.494852, P = 5.76876 when $\Lambda_p = 0.4$. Here we notice that the eigenvalues each have negative real part. Thus the system is stable for our parameters. This means that we have an endemic if $\Lambda_p = 0.4$.

Figure 14 shows the continuation for Λ_p when the jacobian is evaluated at the disease free equilibria. For $\Lambda_p < .744$ the eigenvalues have real negative part which means that the disease free equilibrium is unstable. Also,

Figure 15 shows the continuation for Λ_p when the jacobian is evaluated at the endemic equilibria. For $\Lambda_p > .744$ one of the eigenvalues has real positive part which means that the endemic equilibrium is unstable.

We notice that if the predators are not introduced at a high enough rate the disease will persist.

6. Conclusion

We presented two plant virus propagation models, one with no delays and the other with two delays. Even though we used a general model, we applied it to specific examples to give additional validation and to show the type of results that can be expected if more data was available. Showing that a model can be used for predictions may encourage plant and insect researchers to gather the data necessary for the model. With the data to be collected, the parameters can be modified to fit a particular situation.

The delay model can be useful for agriculture researchers and workers. They can utilize the model to help predict the behavior of the epidemic. For example, suppose an agriculture worker notices a plant endemic occurs, but after a period of time they notice that fewer

plants are becoming infected. The delay model will let the worker know that there will be an increase in the number of infections at a later time, because of the oscillatory behavior of the model. This will force workers to not become relaxed in trying to combat the virus through vector control means.

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Chapter 4
Modeling Plant Virus Propagation with Seasonality

Mark Jackson[1], Benito M. Chen-Charpentier[1,*]

Abstract

Plants are essential to life. They are a source of food, medicine, clothing, and are important to a healthy environment. Unfortunately, plants can become infected with a disease. A viral infection is one way that a plant may become diseased. Often times, plants die from this infection. These viruses hurt the agriculture industry as billions of dollars are lost due to crop loss every year. An insect vector is typically the cause for the virus propagation. The vectors exhibit seasonal behavior as they are active in the warm months, but not as much so in the cooler months. To defend against the vectors, pesticides have been used. While the pesticides might be effective in controlling the vectors, they can have harmful side effects on the environment. An alternative solution is to introduce a predator, or just increase the number of a naturally present one, to prey on the insects. In this paper, we use a mathematical model of ordinary differential equation to model the dynamics of this biological process. We first present an autonomous system, then two nonautonomous systems, accounting for the periodic nature of the insects. To analyze the models, the basic reproductive number is used. We demonstrate a couple of approaches for determining this number: a time average approach and a linear operator approach. Afterwards, numerical simulations are used to demonstrate the results. Finally, comparisons are made between the models and the approaches.
Keywords: periodic coefficients, plant virus propagation, mathematical modeling

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1. Introduction

Plants are a food source for man and many species. They also are sources of medicines, fibers for clothes, and are essential for a healthy environment. But plants are subject to diseases many of which are caused by viruses. These viruses often kill the plant. Specific examples can be seen in [5], [6], [11]. As a result, billions of dollars are lost every year because of virus related crop loss [10]. Most of the time, virus propagation is done by an insect vector. Insect vectors typically have a seasonal behavior. For example, aphids, a vector that transmits plant viruses, are more active in the growing season than other times of the year [7]. In general, vectors are very active in the warm months and not very active, almost dormant, in the cool or wet months. To combat the vectors, chemical insecticides are commonly used as a control. Unfortunately, these chemicals have toxic effects on humans, animals and the environment in general. An alternative is to introduce a predator, or just increase the number of a naturally present one, to prey on the insects. To understand the dynamics, we will use a mathematical model of differential equations.

Mathematical models have been used to model plant virus propagation [3], [4], [5], [9], [12]. However, these models do not take into consideration of the periodic nature of the vectors. In this paper, we extend the work in [4] to include this fact by introducing periodic coefficients to the model. We will consider a Holling Type 1 interaction. To analyze the model, the basic reproductive number R_0 is used. This is the number of secondary infections an infected individual generates. If $R_0 < 1$, the disease will die out as $t \to \infty$, but if $R_0 > 1$, an endemic will occur. For autonomous systems, the authors in [2] developed a method to determine basic reproductive number using the next generation approach. For nonautonomous systems, there have been several approaches to determining R_0. The authors in [8], and [14] used a time average approach by replacing the periodic coefficients with their long time averages. This approach works in some specific cases, but can overestimate or underestimate R_0 in others. In [1], the authors defined R_0 for periodic case and presented some numerical methods to determine R_0. The authors in [13] established the basic reproductive number to a large class of compartmental models using linear operators.

In section 2, we outline our modeling assumptions and introduce a system of nonautonomous ordinary differential equations with periodic contact rates. In section 3, we analyze the autonomous system using the basic reproductive number determined by the next generation matrix. In section 4, we analyze the nonautonomous system. We first attempt to analyze the model using the basic reproductive number obtained by the time average method [8]. Then we use the linear operator method to determine R_0 proposed by [13]. After determining R_0, some numerical simulations will be given to demonstrate the analytical results. In section 5, we then extend the model to include periodic migration rates and periodic natural death rates. We again use the linear operator method to determine R_0 and provide numerical simulations to demonstrate the analytical results. In section 6, we will wrap up the results in the conclusion.

2. Model Assumptions and Model

We consider 6 populations: susceptible plants $S(t)$, infected plants $I(t)$, recovered plants $R(t)$, susceptible insect vectors $X(t)$, infected insect vectors $Y(t)$, and predators $P(t)$. Each variable describes it's respective population at time t. Susceptible plants do not have the disease but could contract the disease if infected with the virus. The infected plants have the virus but cannot directly transmit the virus to susceptible plants. Infected plants can either die from the disease or recover. Additionally, since the infected plants can die from the viral infection their death rate is higher than that of plants that do not have the virus. We also assume that as soon as a plant dies either from the infection or from a natural death, it is immediately replaced with a new susceptible plant by a farm worker. Thus it is reasonable to assume that the plant population remains fixed and the total plant population will be denoted by K. This assumption has the modeling advantage that $K = S(t) + I(t) + R(t)$ can be used to eliminate the recovered population from the system of equations. The susceptible insects do not have the virus but can obtain the virus if they come in contact with a infected plant. Infected insects can transmit the virus to susceptible plants upon contact. We assume no vertical transmission of the virus with neither plants nor vectors. Moreover,

we assume that the virus does not harm the vector and thus the vector does not defend against the virus and it retains the virus for the rest of its life. Additionally, contact rate between insect and plant changes depending on the season. Contact rates are higher in warmer months than cooler ones. Thus, we first assume the contact rates to be yearly periodic. The predators use this energy from feeding on the vectors to grow the predator population. We also assume that even if a predator consumes an infected insect, it will not become infected with the virus. Since the insects do not show signs of being infected, the predators cannot differentiate between healthy and infected insects. Thus, we assume that the predators consume both infected and healthy insects at the same rate. The interaction between vector and plant as well as that of predator and vector are assumed to have a limitation of the form of predator-prey Holling type 1. The following table lists the parameters in the model.

The following is a flow diagram that describes the interactions as described in the model assumptions.

The following system of ordinary differential equations modeling the biological situation with periodic contact rates as outlined in the flow diagram.

$$\begin{aligned}
\frac{dS}{dt} &= \mu(K-S) + dI - \beta(t)YS \\
\frac{dI}{dt} &= \beta(t)YS - \omega I \\
\frac{dX}{dt} &= \Lambda - \beta_1(t)IX - cXP - mX \\
\frac{dY}{dt} &= \beta_1(t)IX - cYP - mY \\
\frac{dP}{dt} &= \alpha_4 cXP + \alpha_4 cYP - \delta P.
\end{aligned} \tag{1}$$

where

$$\beta(t) = \beta(1 + h\cos(\frac{2\pi t}{365})) \qquad \beta_1(t) = \beta_1(1 + h\cos(\frac{2\pi t}{365})). \tag{2}$$

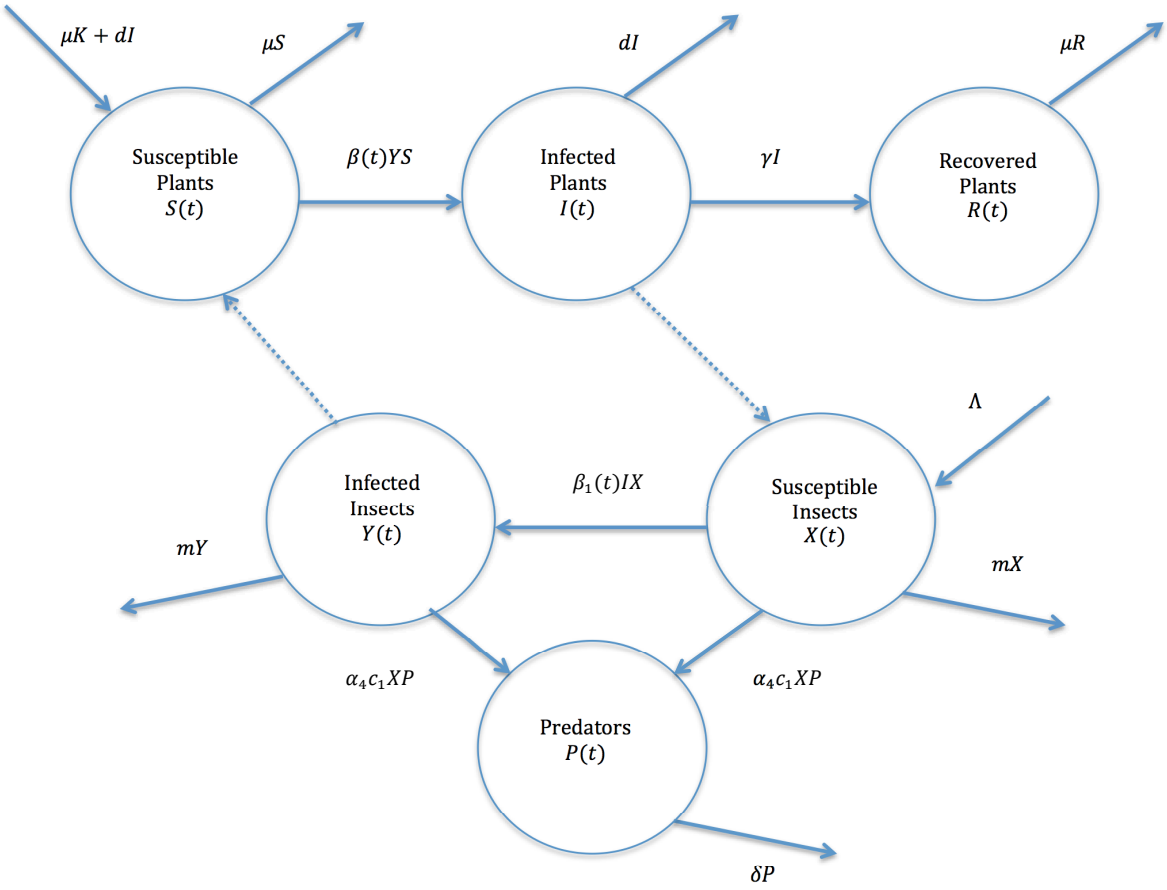


Figure 1: The above figure describes the interations between the plants, insects, and predators. A solid arrow toward a state represents an inflow of the given population. A solid arrow away from a population describes an outflow of the given population. A dotted line from one population to another illustrates a transmission of the virus.

Notice that when h=0, we have an autonomous system.

Table 1 gives a list of the parameters used in the model, their description and their value or range. The parameters were used from the ranges given in [12].

Parameter	Description	Value
K	Total plant host population	63.00
β	biting rate of plants due to vectors	0.01000-0.1000
β_1	biting rate of vectors due to plants	0.01000-0.1000
μ	natural death rate of plants	0.01000
m	natural death rate of vectors	0.2974
γ	recovery rate of plants	0.01000
Λ	replenishing rate of vectors	10.00
d	death rate of infected plants due to the disease	0.2000
c	contact rate between predators and healthy insects	0.05000
δ	natural death rate of predators	0.05000
α_4	conversion rate of predators due to insects	0.01000

3. Analysis of the Autonomous Case

We first analyze the autonomous system when h=0, meaning that all parameters in the model are constant. To study this system, we begin by finding the disease free steady solutions. By setting the right hand side equations equal to 0 and solving for S, I, X, Y, and P we get the following disease free equilibrium (DFE) points

$$S^* = K, \quad I^* = 0, \quad X^* = \frac{\delta}{c\alpha_4}, \quad Y^* = 0, \quad P^* = \frac{-m}{c} + \frac{\Lambda\alpha_4}{\delta}$$

$$S^* = K, \quad I^* = 0, \quad X^* = \frac{\Lambda}{m}, \quad Y^* = 0, \quad P^* = 0.$$

We focus our attention to the first DFE since second disease free equilibrium point is not as meaningful as there are no predators in the system. To determine the conditions for which this equilibrium point is stable or unstable, we could linearize the system about the equilibrium point. Then we can determine stability when the eignevalues of the Jacobian matrix all have negative real parts or instability if one eigenvalue has a real part. This can be challenging, since we have a system of 5 equations. An alternative is to look at the basic reproductive number, R_0. This is defined as the number of secondary infections an infected individual will infect in a completely susceptible population. If $R_0 > 1$ then the disease will propagate, otherwise the disease will eventually die and a fraction of the population will escape infection. Not only does this number have biological meaning, but it has a mathematical meaning pertaining to the disease free equilibrium point. The authors in [2] developed a method for determining R_0 for autonomous compartmental models. Their approach is to examine a reduced system of equations that involves only infected or latent classes.To determine R_0 we need to define the next generation matrix.

A next generation matrix G consists of two parts: F and V^{-1}, where

$$F = \left[\frac{\partial \mathscr{F}_i(x_0)}{\partial x_j} \right]$$

and

$$V = \left[\frac{\partial \mathscr{V}_i(x_0)}{\partial x_j} \right].$$

The $\mathscr{F}_i$ are the new infections, while the $\mathscr{V}_i$ transfers of infections from one compartment to another. x_0 is the disease-free equilibrium state, and R_0 is the dominant eigenvalue of the matrix $G = FV^{-1}$.

To set up the F and V matricies, we use the following 2 equations from our system since they dictate the new or transfers of infections.

$$\begin{aligned} \frac{dI}{dt} &= \beta YS - \omega I \\ \frac{dY}{dt} &= \beta_1 IX - cYP - mY \end{aligned} \tag{3}$$

Moreover, x_1 will correspond to the I variable and x_2 will correspond to the Y variable. and So

$$\mathscr{F}_1 = \beta YS,$$

$$\mathscr{F}_2 = \beta_1 IX,$$

$$\mathscr{V}_1 = \omega I,$$

and

$$\mathscr{V}_2 = cYP + mY,$$

and $x_0 = [0,0,K,\frac{\delta}{c\alpha_4},\frac{-m}{c}+\frac{\Lambda\alpha_4}{\delta}]$

In our case, the F and V matrices are:

$$F = \begin{bmatrix} 0 & \beta K \\ \frac{\beta_1\delta}{c\alpha_4} & 0 \end{bmatrix}, V = \begin{bmatrix} \omega & 0 \\ 0 & \frac{c\alpha_4\Lambda}{\delta} \end{bmatrix}.$$

Thus the Next Generation Matrix is

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\beta K\delta}{c\alpha_4\Lambda} \\ \frac{\delta\beta_1}{c\omega\alpha_4} & 0 \end{bmatrix}.$$

Therefore, the basic reproduction number is

$$R_0 = \frac{\delta\sqrt{\beta\beta_1 K}}{c\alpha_4\sqrt{\omega\Lambda}}.$$

If $R_0 > 1$, the disease free equilibrium point is unstable, but stable if $R_0 < 1$

4. Analysis of Nonautonomous Case

To determine the conditions for which the disease free is stable or unstable is much more difficult for a system of periodic coefficients. One way of determining the conditions is finding the eigenvalues of the linearized system about the DFE, then finding when the eigenvalues have negative real part. Unfortunately, these eignvalues are periodic in nature. Thus making the task nearly impossible. Alternatively, we can calculate R_0. As we saw in the previous sections for autonomous systems, R_0 is calculated using next generation matrix method. Calculating R_0 for nonautonomous systems is more difficult due to the periodic coefficients. One way we can calculate R_0 is to approximate it numerically by fixing time and using the next generation matrix from the autonomous case for several values of time and take an average. The problem with this method is that if R_0 is near 1, we risk overapproximating or underapproximating it. Fortunately, there have been two approaches that have been used in the literature: time average method [8],[14] and linear operator method [13]. The authors in [8] considered periodic contact rates in some SIR and SEIR models and were able to calculate the basic reproductive number using long time averages for the periodic coefficients. In [14], the authors extended the work in [8] by considering SIRS models with seasonal births, deaths, transitions, seasonal isolation, and with multiple strains and cross-immunity. We attempt the time average method to see if it works in our case. For this method, we replace time varying parameters by long term averages, and use next generation approach to determine R_0. Define

$$< \cdot >= \lim_{t\to\infty} \int_0^t \cdot \ d\tau$$

to be the time average of $\cdot$ so

$$< \beta(1+h\cos(\frac{2\pi t}{365})) >= \beta \qquad < \beta_1(1+h\cos(\frac{2\pi t}{365})) >= \beta_1$$

Thus R_0 is same for autonomous case. $R_0 = \frac{\delta\sqrt{\beta\beta_1 K}}{c\alpha_4\sqrt{\omega\Lambda}}$

Using the values of the parameters in Table 1 with $\beta = .0184, \beta_1 = .0184$ and $h = 0.5$, we obtain $R_0 = .9846$.

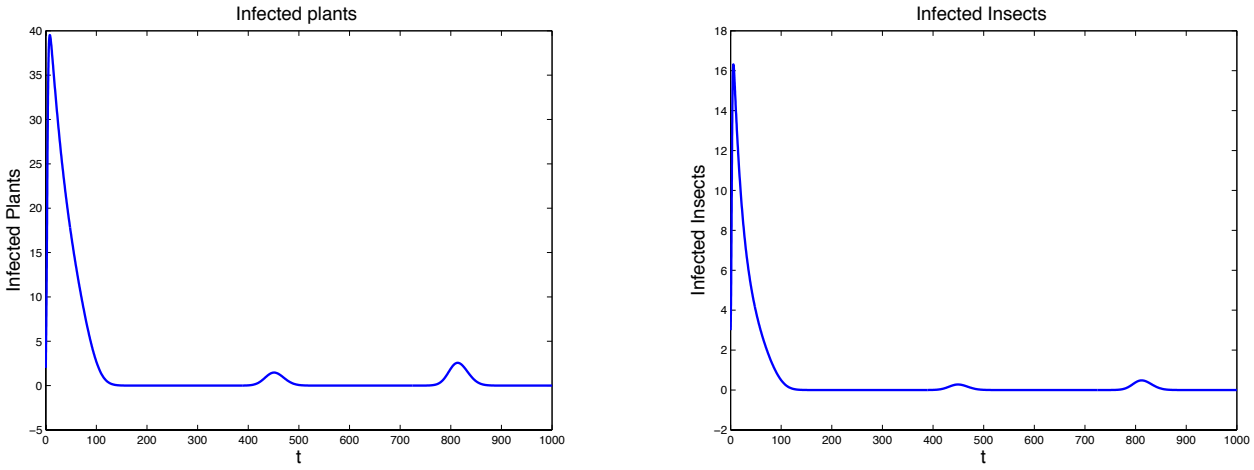


Figure 2: Figure 2a (left) shows the infected plant population and Figure 2b (right) shows infected insects when $R_0 = .9846$.

Notice that when $R_0 < 1$, Figures 2a and 2b both show that infected plants and insects do not go to disease free state. This indicates that the time average method is not useful for the this system.

We now set up the conditions for the linear operator method developed by the authors in [13]. Let $x = (x_1, x_2, ..., x_n)$ be the state of individuals in each compartment, $x_i \geq 0$. Assume compartments can be broken into two types infected and uninfected compartments with indices $i = 1, .., m$ and $i = m+1, ..., n$. Let X_s be the set of disease free states, $\mathscr{F}_i(t,x)$ be input rate of newly infected individuals, $\mathscr{V}_i^-(t,x)$ be the rate of transfer out of compartment i, and be the input rate of individuals by other means $\mathscr{V}_i^+(t,x)$. The model is given by

$$\frac{dx_i}{dt} = \mathscr{F}_i(t,x) - \mathscr{V}_i(t,x) = f_i(t,x), i = 1, .., n$$

where $\mathscr{V}_i(t,x) = \mathscr{V}_i^-(t,x) - \mathscr{V}_i^+(t,x)$.

In order to use the linear operator method, some conditions must be satisfied:

- (A1) For each $1 \leq i \leq n$ the functions $\mathscr{F}_i(t,x)$, $\mathscr{V}_i^+(t,x)$, and $\mathscr{V}_i^-(t,x)$ are nonnegative and continuous on $\mathbb{R} \times \mathbb{R}^n$ and continuously differentiable wrt x
- (A2) There is a real $\Omega > 0$ such that $\mathscr{F}_i(t,x)$, $\mathscr{V}_i^+(t,x)$, and $\mathscr{V}_i^-(t,x)$ are Ω periodic for all i
- (A3) If $x_i = 0$ then $\mathscr{V}_i^-(t,x) = 0$
- (A4) $\mathscr{F}_i(t,x) = 0$ for $i > m$
- (A5) If $x \in X_s$ then $\mathscr{F}_i(t,x) = \mathscr{V}_i^+(t,x) = 0$ for $i = 1, ..m$
- (A6) Define

$$M(t) = \left[\frac{\partial f_i(x_0)}{\partial x_j} \right]_{m+1 \leq i,j \leq n}$$

  and $\Phi_M(t)$ to be the monodromy matrix of $\frac{dz}{dt} = M(t)z$. The spectral radius of $\Phi_M(\Omega)$ is needs to be less than 1.

- (A7) Define

$$F = \left[\frac{\partial \mathscr{F}_i(x_0)}{\partial x_j} \right]_{1 \leq i,j \leq m} \quad V = \left[\frac{\partial \mathscr{V}_i(x_0)}{\partial x_j} \right]_{1 \leq i,j \leq m}$$

The spectral radius of $\Phi_{-V}(\Omega)$ is to be less than 1, where $\Phi_{-V}(t)$ is monodromy matrix of $\frac{dy}{dt} = -V(t)y$

Note: the monodromy matrix is the fundamental matrix of a system of ordinary differential equations evaluated at the period of the coefficients of the system.

We organize the system to be

$$\begin{aligned}
\frac{dI}{dt} &= \beta YS - \omega I \\
\frac{dY}{dt} &= \beta_1 IX - cYP - mY \\
\frac{dS}{dt} &= \mu(K-S) + dI - \beta(t)YS \\
\frac{dX}{dt} &= \Lambda - \beta_1(t)IX - cXP - mX \\
\frac{dP}{dt} &= \alpha_4 cXP + \alpha_4 cYP - \delta P.
\end{aligned} \tag{4}$$

with variables $x_1 = I$, $x_2 = Y$, $x_3 = S$, $x_4 = X$, $x_5 = P$ and the disease free state $x_0 = [0,0,K,\frac{\delta}{c\alpha_4},\frac{-m}{c} + \frac{\Lambda\alpha_4}{\delta}]$.

Thus we have that

$$\mathscr{F} = \begin{bmatrix} \beta(t)YS \\ \beta_1 IX \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \mathscr{V}^- = \begin{bmatrix} \omega I \\ cYP + mY \\ \beta(t)YS \\ \beta_1(t)IX + cXP + mX \\ \delta P \end{bmatrix} \quad \mathscr{V}^+ = \begin{bmatrix} 0 \\ 0 \\ \mu(K-S) + dI \\ \Lambda \\ \alpha_4 cXP + \alpha_4 cYP \end{bmatrix}$$

Note that (A1)-(A5) are satisfied.

To verify (A6), we must find the spectral radius of $\Phi_M(t)$. To do this we must solve

$$\frac{dz}{dt} = \begin{bmatrix} -\mu & 0 & 0 \\ 0 & \frac{-c\alpha_4\Lambda}{\delta} & \frac{-\delta}{\alpha_4} \\ 0 & \frac{\alpha_4(c\alpha_4\Lambda - m\delta)}{\delta} & 0 \end{bmatrix} z$$

However, this is a difficult task because determining the eigenvectors associated with the eignvalues is not easy. This is due to the fact there are many parameters to consider. So, it is verified numerically with the spectral radius of $\Phi_M(t)$ is equal to .026

For (A7) we need the spectral radius of $\Phi_{-V}(\Omega)$, which is the monodromy matrix of the system

$$\frac{dy}{dt} = -\begin{bmatrix} \omega & 0 \\ 0 & \frac{c\alpha_4}{\delta} \end{bmatrix} y$$

thus

$$\Phi_{-V}(t) = \begin{bmatrix} e^{-\omega t} & 0 \\ 0 & e^{-\frac{c\alpha_4 \Lambda t}{\delta}} \end{bmatrix}$$

and spectral radius is $\max\{e^{-365\omega}, e^{-\frac{365c\alpha_4\Lambda}{\delta}}\}$ which is less than 1 as needed

Let $Y(t,s), t \geq s$ be the evolution operator of the ω-periodic system

$$\frac{dy}{dt} = -V(t)y.$$

For every $s \in \mathbf{R}$, the m by m matrix $Y(t,s)$ satisfies

$$\frac{dY}{dt} = -V(t)Y(t,s).$$

Suppose that $\phi(s)$ is the initial distribution of infections individuals. Given $t \geq s$, $Y(t,s)F(s)\phi(s)$ gives the distribution of infected individuals who were newly infected at time s and remain in the infected compartments at time t. Let C_Ω be the ordered Banach space of all Ω-periodic functions from $\mathbf{R} \to \mathbf{R}^n$, which is equiped with the maximum norm and the positive cone $C_\Omega^+ := \{\phi \in C_\Omega : \phi(t) \geq 0 \forall t \in \mathbf{R}\}$. Define a linear operator $L : C_\Omega \to C_\Omega$ by

$$L(\phi)(t) = \int_0^\infty Y(t, t-a)F(t-a)\phi(t-a)da$$

for all $t \in \mathbf{R}$ and $\phi \in C_\Omega$. Define R_0 to be the spectral radius of L. This spectral radius is difficult to

determine in our case. However, authors in [13] proved the following theorem.

Theorem 1. Assume that (A1)-(A7) hold. Then the following statements are valid:

- 1 $R_0 = 1$ iff $\rho(\Phi_{F-V}(\Omega)) = 1$
- 2 $R_0 > 1$ iff $\rho(\Phi_{F-V}(\Omega)) > 1$
- 3 $R_0 < 1$ iff $\rho(\Phi_{F-V}(\Omega)) < 1$

$\rho(\Phi_{F-V}(t))$ is spectral radius of the monodromy matrix of the system $\frac{dz}{dt} = (F-V)z$

This theorem is beneficial since we do not need to detemine the spectral radius of L and that we only need to determine the spectral radius of $\Phi_{F-V}(t)$ at the period. The draw back is that we are not able to determine R_0 explicitly. We now determine $\rho(\Phi_{F-V}(\Omega))$.

We have that

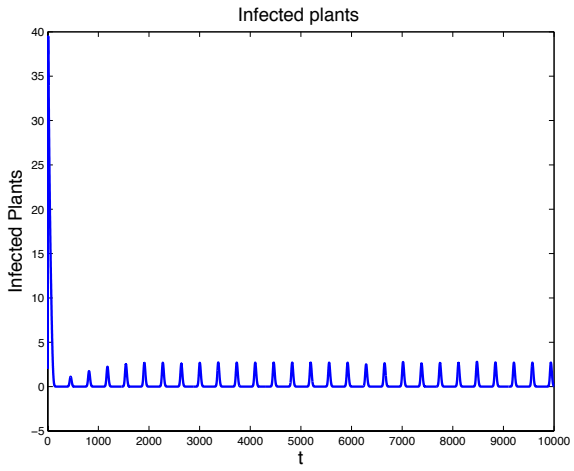
$$F = \begin{bmatrix} 0 & \beta(t)K \\ \frac{\beta_1(t)\delta}{c\alpha_4} & 0 \end{bmatrix}, V = \begin{bmatrix} \omega & 0 \\ 0 & \frac{c\Lambda\alpha_4}{\delta} \end{bmatrix}$$

Thus the system we must solve is

$$\frac{dz}{dt} = \begin{bmatrix} -\omega & \beta(t)K \\ \frac{\beta_1(t)\delta}{c\alpha_4} & -\frac{c\alpha_4\Lambda}{\delta} \end{bmatrix} z$$

However $\rho(\Phi_{F-V}(\Omega))$ is difficult to determine from this system, as detemining the fundamental matrix of a system of periodic equations is not feasible with this many parameters, so it is done numerically. With $h = 0.5, \beta = \beta_1 = .018186$, and values from Table 1.1 we get that $\rho(\Phi_{F-V}(\Omega))$=1. Which means $R_0 = 1$. We run some numerical simulations to validate the results. The following Figure 3a and Figure 3b show the infected plants and insects when $R_0 > 1$. Figure 4a and 4b show infected plants and insects when $R_0 < 1$.

Notice that Figures 3a and 3b demonstrate that infected plants and insects do not tend to 0 when $R_0 > 1$, However, both infected populations do when $R_0 < 1$ as seen in Figures 4a and 4b.



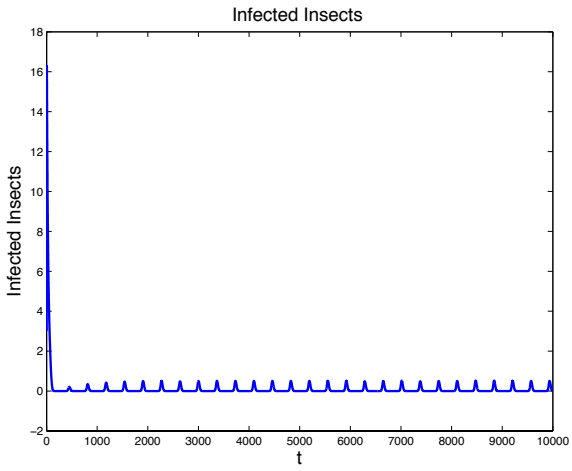
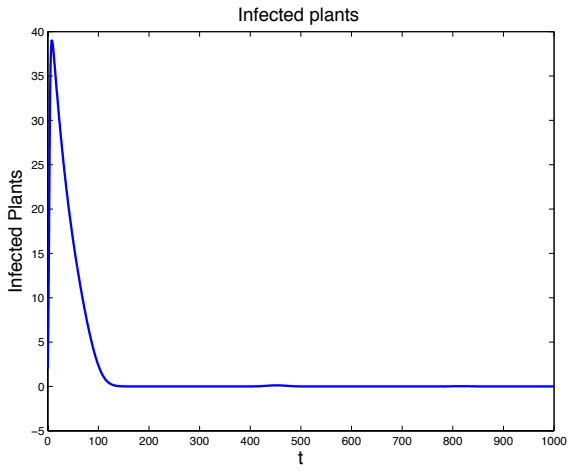


Figure 3: Figure 3a (left) shows the infected plants and Figure 3b (right) shows the infected insects when $R_0 > 1$



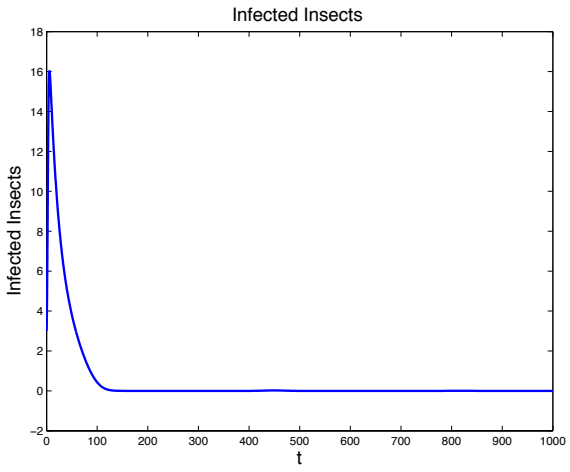


Figure 4: Figure 4a (left) shows the infected plants and Figure 4b (right) shows the infected insects when $R_0 < 1$

5. Periodic Immigration and Death Rates

Immigration rates and death rates change due to the season. This is because insects more active in warmer months, less active in cooler months. Therefore, we are now going to consider Λ and m, the immigration and natural death rates respectively, to be periodic. We also assume that the contact rates remain periodic as in equation 1. The following is the updated system.

$$\begin{aligned}
\frac{dS}{dt} &= \mu(K-S) + dI - \beta(t)YS \\
\frac{dI}{dt} &= \beta(t)YS - \omega I \\
\frac{dX}{dt} &= \Lambda(t) - \beta_1(t)IX - cXP - m(t)X \\
\frac{dY}{dt} &= \beta_1(t)IX - cYP - m(t)Y \\
\frac{dP}{dt} &= \alpha_4 cXP + \alpha_4 cYP - \delta P
\end{aligned} \tag{5}$$

$$m(t) = m(1 + A\cos(\frac{2\pi t}{365})) \qquad \Lambda(t) = \Lambda(1 + A\cos(\frac{2\pi t}{365})) \tag{6}$$

By setting the right hand side equations equal to 0 and solving for S, I, X, Y, and P we get the following disease free equilibrium points:

$$S^* = K, \quad I^* = 0, \quad X^* = \frac{\delta}{c\alpha_4}, \quad Y^* = 0, \quad P^* = \frac{-m(t)}{c} + \frac{\Lambda(t)\alpha_4}{\delta}$$

$$S^* = K, \quad I^* = 0, \quad X^* = \frac{\Lambda(t)}{m(t)}, \quad Y^* = 0, \quad P^* = 0$$

The second disease free equilibrium point is not as meaningful as the first as there are no predators in the system.

To use linear operator method, we must verify conditions (A1)-(A7). We construct $\mathscr{F}, \mathscr{V}^-$, and $\mathscr{V}^+$ in a similar way when only the contact rates were periodic.

$$\mathscr{F} = \begin{bmatrix} \beta(t)YS \\ \beta_1 IX \\ 0 \\ 0 \\ 0 \end{bmatrix} \mathscr{V}^- = \begin{bmatrix} \omega I \\ cYP + m(t)Y \\ \beta(t)YS \\ \beta_1(t)IX + cXP + m(t)X \\ \delta P \end{bmatrix} \mathscr{V}^+ = \begin{bmatrix} 0 \\ 0 \\ \mu(K-S) + dI \\ \Lambda(t) \\ \alpha_4 cXP + \alpha_4 cYP \end{bmatrix}$$

As before, it is easy to verify that (A1)-(A5) are satisfied.

To verify (A6), we must find the spectral radius of $\Phi_M(t)$. To do this we must solve

$$\frac{dz}{dt} = \begin{bmatrix} -\mu & 0 & 0 \\ 0 & \frac{-c\alpha_4\Lambda(t)}{\delta} & \frac{-\delta}{\alpha_4} \\ 0 & \frac{\alpha_4(c\alpha_4\Lambda(t) - m(t)\delta)}{\delta} & 0 \end{bmatrix} z$$

However, this is a difficult task, since this system is a system of periodic coefficients, so it is verified numerically with the spectral radius of $\Phi_M(t)$ is equal to .026

For (A7) we need the spectral radius of $\Phi_{-V}(\Omega)$, which is the monodromy matrix of the system

$$\frac{dy}{dt} = -\begin{bmatrix} \omega & 0 \\ 0 & \frac{c\Lambda(t)\alpha_4}{\delta} \end{bmatrix} y$$

thus

$$\Phi_{-V}(t) = \begin{bmatrix} e^{-\omega t} & 0 \\ 0 & e^{-\frac{c\alpha_4\Lambda t}{\delta}} \end{bmatrix}$$

and spectral radius is $\max\{e^{-365\omega}, e^{-\frac{365c\alpha_4\Lambda}{\delta}}\}$ which is less than 1 as needed

We have that

$$F = \begin{bmatrix} 0 & \beta(t)K \\ \frac{\beta_1(t)\delta}{c\alpha_4} & 0 \end{bmatrix}, V = \begin{bmatrix} \omega & 0 \\ 0 & \frac{c\Lambda(t)\alpha_4}{\delta} \end{bmatrix}$$

Thus the system we must solve is

$$\frac{dz}{dt} = \begin{bmatrix} -\omega & \beta(t)K \\ \frac{\beta_1(t)\delta}{c\alpha_4} & -\frac{c\alpha_4\Lambda(t)}{\delta} \end{bmatrix} z$$

However $\rho(\Phi_{F-V}(\Omega))$ is difficult to determine from this system, since determining the fundamental matrix of a periodic system is almost impossible. So, it is done numerically. With $h = 0.5, A = 0.5\beta = \beta_1 = .0186371$, and values from Table 1.1 we get that $\rho(\Phi_{F-V}(\Omega))$=1. Which means $R_0 = 1$. We run some numerical simulations to validate the results. The following figures show the infected plants and insects when $R_0 > 1$ and $R_0 < 1$.

Infected Insects and Infected Plants $R_0 > 1$

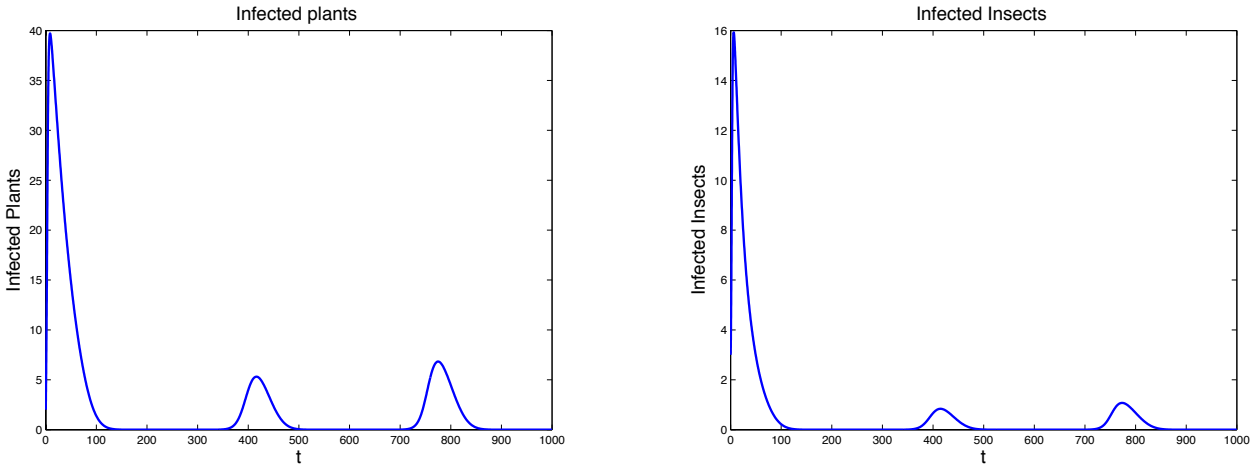


Figure 5: Figure 5a (left) shows the infected plants and Figure 5b (right) shows the infected insects when $R_0 > 1$

Notice that in Figures 5a and 5b infected plants and insects do not tend to 0 when $R_0 > 1$, in fact, they tend to a nonzero periodic solution. However, Figures 6a and 6b illustrate that both infected classes tend to 0 when $R_0 < 1$.

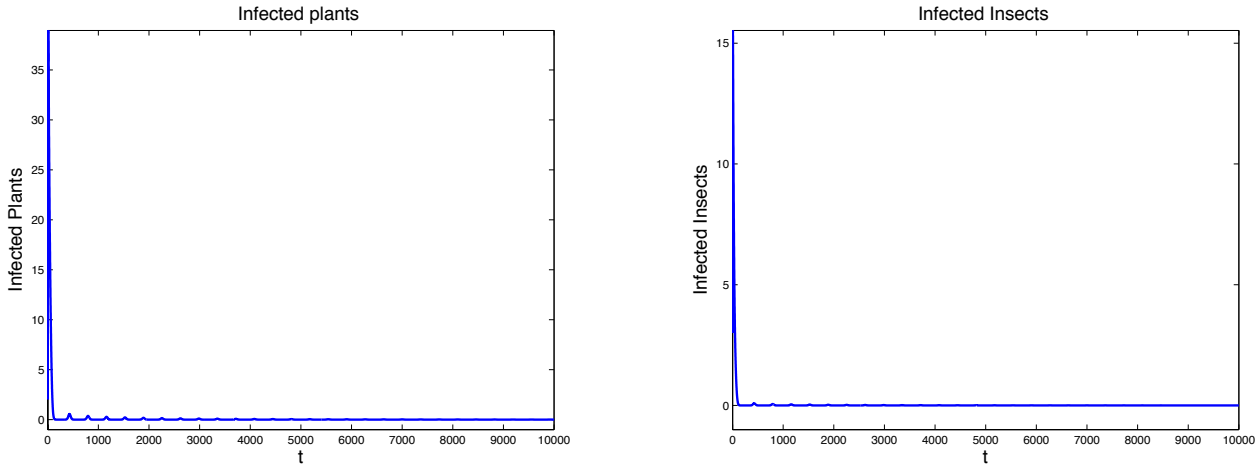


Figure 6: Figure 6a (left) shows the infected plants and Figure 6b (right) shows the infected insects when $R_0 < 1$

6. Conclusions

Making contact, death and immigration rates periodic makes model more realistic, with the tradeoff being that R_0 is more difficult to calculate. R_0 calculated by taking average value periodic coefficients using methods in [8] and [14] is not as accurate as linear operator method [13]. R_0 calculated by linear operator method is more difficult to implement, but we only need to know if $R_0 > 1$ or not. Having a more accurate R_0 is crucial in epiodimiological models. If R_0 is underestimated, then enough predators to control the vectors will not be introduced meaning an epidemic is likely to occur. If R_0 is overestimated, the number of predators needed will be higher than necessary according to the model. This means a higher cost to farmers who are purchasing the predators for control.

When immigration and death rates are made periodic in addition to the periodic contact rates , the system becomes more difficult to analyze, however it is more realistic. Additionally, R_0 changes for when the immigration and death rates are periodic. In the first model, when $\beta = \beta_1 = .018186$, $R_0 = 1$ but in second model when $\beta = \beta_1 = 0.0186371$, $R_0 = 1$. This means the contact rates from the first model yield a lower R_0 in the second, more realistic model. This means that we would underestimate R_0 using the contact rates from the first model.

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Chapter 5
An Optimal Control of Plant Virus Propagation with Seasonality and Delays

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Abstract

Plants are a food source for man and many species. They also are sources of medicines, fibers for clothes, and are essential for a healthy environment. But plants are subject to diseases many of which are caused by viruses. These viruses often kill the plant. As a result, billions of dollars are lost every year because of virus related crop loss [8]. Most of the time, virus propagation is done by a vector, usually insects that bite infected plants, get themselves infected and then bite susceptible plants. Insect vectors typically have a seasonal behavior. They are very active in the warm months and not very active, almost dormant, in the cool months. To combat the vectors, chemical insecticides are commonly used as a control. Unfortunately, these chemicals not only are expensive but also have toxic effects on humans, animals and the environment in general. An alternative is to introduce a predator species, or just increase the number of a naturally present one, to prey on the insects and limit the spread of the virus. A combination of insecticide and predators can be used to control the vector population. The question is whether there is an optimal combination.

We first introduce a mathematical model of ordinary differential equations describing the interaction between plants, vectors and predators. This model can be used with constant coefficients or with periodic coefficients such as the infection and birth and death rates. To determine the optimal amount of predators to introduce and insecticide to use, an objective function giving the total cost to the farmer of the disease. This function depends on the number of infected plants and on the cost of the predators and the insecticide. The cost of the insecticide can also include an environmental

cost. We find the controls that minimize the objective function subject to the population variables satisfying the differential equation model and initial conditions, together with constraints such that the controls are nonnegative.

There are a couple of different approaches that can be used to solve the optimal control problem: indirect and direct methods. Indirect methods are based on Pontryagin maximum principle. To use this method, we need to determine the Hamiltonian which involves the integrand of the objective function and the inner product of the adjoint variables with the right hand side of the system of differential equations. Then the Hamiltonian is used to determine the adjoint equations, the transversality condition at the end time, and the optimality condition. The numerical procedure used is to give an initial guess of the control functions, solve the population ordinary system forward in time numerically, the adjoint system back in time and use the optimality condition to obtain better estimates for the control functions, and iterate until convergence under a given criteria is obtained. Numerical simulations will be used to illustrate the results. We consider using only one control and keep the other with a constant value, and illustrate the difficulties using the indirect method to solve the problem.

The other approach is using a direct method. Direct methods are more robust than indirect methods, however they can be less precise. We use the BOCOP software which implements a direct method. First it converts the infinite dimensional problem to a finite dimensional problem then it uses the IPOPT algorithm to determine an optimal solution. We illustrate the results and compare to the indirect method. We then consider using the direct methods to solve the problem when periodicity is introduced as well as delays. From the practical side, the model can be used to help farmers determine the right balance of insecticide and predators while minimizing the total cost.

Keywords: periodic coefficients, plant virus propagation, optimal control

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1. Introduction

Plants are a food source for man and many species. They also are sources of medicines, fibers for clothes, and are essential for a healthy environment. But plants are subject to diseases many of which are caused by viruses. These viruses often kill the plant. As a result, billions of dollars are lost every year because of virus related crop loss. Most of the time, virus propagation is done by a vector, usually insects that bite infected plants, get themselves infected and then bite susceptible plants. Insect vectors typically have a seasonal behavior. They are very active in the warm months and not very active, almost dormant, in the cool months. To combat the vectors, chemical insecticides are commonly used as a control. Unfortunately, these chemicals not only are expensive but also have toxic effects on humans, animals and the environment in general. An alternative is to introduce a predator species, or just increase the number of a naturally present one, to prey on the insects and limit the spread of the virus. A combination of insecticide and predators can be used to control the vector population. The question is whether there is an optimal combination.

In our study we consider six populations: susceptible, infected and recovered plants, susceptible and infected vectors, and predators. We assume that the susceptible plants can become infected if an infected insect feeds and is able to transmit the virus to the plant; the infected plant will either die from the virus or recover; a healthy vector will can obtain the virus by feeding on an infected plant; the infected vectors have no ill effects from the virus so they do not fight the virus and therefore they do not recover; and the virus does not affect the predators. Also, the total number of plants is assumed to be constant since the farmers will replace a dead plant with a healthy one. The plant populations can be determined using the total constant plant population.

We first introduce a mathematical model of ordinary differential equations describing the interaction between plants, vectors and predators. This model can be used with constant coefficients or with periodic coefficients such as the infection and birth and death rates. To determine the optimal amount of predators to introduce and insecticide to use, an objective function giving the total cost

to the farmer of the disease. This function depends on the number of infected plants and on the cost of the predators and the insecticide. The cost of the insecticide can also include an environmental cost. We find the controls that minimize the objective function subject to the population variables satisfying the differential equation model and initial conditions, together with constraints such that the controls are nonnegative.

There are a couple of methods to determining the optimum cost. One is using indirect methods. This approach is based on Pontryagin maximum principle. Thus we need to determine the Hamiltonian which involves the integrand of the objective function and the inner product of the adjoint variables with the right hand side of the system of differential equations. Then the Hamiltonian is used to determine the adjoint equations, the transversality condition at the end time, and the optimality condition. The numerical procedure used is to give an initial guess of the control functions, solve the population ordinary system forward in time numerically, the adjoint system back in time and use the optimality condition to obtain better estimates for the control functions. And iterate until convergence under a given criteria is obtained. Numerical simulations will be used to illustrate the results.

Because the indirect method using Pontryagin's principle can present convergence issues, we would also like to consider a direct method to solve the problem. Direct methods have the advantage over indirect methods in that they are more straightforward to apply and more robust with respect to the initialization. The cost, however, is that some precision is lost [2]. The direct methods transforms the infinite dimensional optimal control problem into a finite dimensional problem. To do this, the direct method discretizes the dynamics equation as well as the state and control variables with respect to time using a direct transcription approach. BOCOP software that discretizes equations and the variables using a method of the user's choice. It then utilizes the IPOPT solver that implements a primal-dual interior point algorithm.

The paper is organized in the following way. In section 2 we outline the modeling assumptions and introduce a system of ordinary differential equations that model the interactions between

plants, vectors, and predators. In section 3, we introduce the optimal control problem, and use Pontryagin's principle to determine the minimum cost of insecticide and cost of infected plant. To compare with the indirect methods in section 3, we solve the optimal control problem using some direct methods in section 4. In section 5, seasonality is introduced to the problem and the resulting model is analyzed using the direct methods. In section 6, delays are considered and the problem is solved using the direct methods. In the final section, we wrap up the results in the conclusion.

2. Model Assumptions and Model

With similar assumptions as in [4], [5], [6], [9], we consider 6 populations: susceptible plants $S(t)$, infected plants $I(t)$, recovered plants $R(t)$, susceptible insect vectors $X(t)$, infected insect vectors $Y(t)$, and predators $P(t)$. Each variable describes it's respective population at time t. Susceptible plants do not have the disease but could contract the disease if infected with the virus. The infected plants have the virus but cannot directly transmit the virus to susceptible plants. Infected plants can either die from the disease or recover. Additionally, since the infected plants can die from the viral infection their death rate is higher than that of plants that do not have the virus. We also assume that as soon as a plant dies either from the infection or from a natural death, it is immediately replaced with a new susceptible plant by a farm worker. Thus it is reasonable to assume that the plant population remains fixed and the total plant population will be denoted by K. This assumption has the modeling advantage that $K = S(t) + I(t) + R(t)$ can be used to eliminate the recovered population from the system of equations. The susceptible insects do not have the virus but can obtain the virus if they come in contact with a infected plant. Infected insects can transmit the virus to susceptible plants upon contact. We assume no vertical transmission of the virus with neither plants nor vectors. Moreover, we assume that the virus does not harm the vector and thus the vector does not defend against the virus and it retains the virus for the rest of its life. Since the insects do not show signs of being infected, the predators cannot differentiate between healthy and infected insects. Thus, we assume that the predators consume both infected and healthy insects at

the same rate. The interaction between vector and plant as well as that of predator and vector are assumed to have a limitation of the form of predator-prey Holling type 2. The following table lists the parameters in the model.

Table 1 gives a list of the parameters used in the model, their description and their value or range. The parameters were used from the ranges given in [9].

Parameter	Description	Value
K	Total plant host population	63
β	biting rate of plants due to vectors	0.01
β_1	biting rate of vectors due to plants	0.01
α	saturation constant of plants due to vectors	0.2
α_1	saturation constant of vectors due to plants	0.1
μ	natural death rate of plants	.01
m	natural death rate of vectors	.2974
γ	recovery rate of plants	0.01
Λ	replenishing rate of vectors	10
d	death rate of infected plants due to the disease	0.2
c_1	contact rate between predators and healthy insects	0.05
c_2	contact rate between predators and infected insects	0.05
δ	natural death rate of predators	0.05
ε	competition constant between predators	0.01
α_3	saturation of predators due to insects	0.01
α_4	conversion rate of predators due to insects	0.01
d_{in}	death rate due to insecticide	0-0.9
Λ_p	rate at which predators are added	0-10
G	Cost of Insecticide	0-1000
A	Cost of Infected Plant	0-100
F	Cost of Predators	0-1000

The following is a flow diagram that describes the interactions as described in the model as-

sumptions.

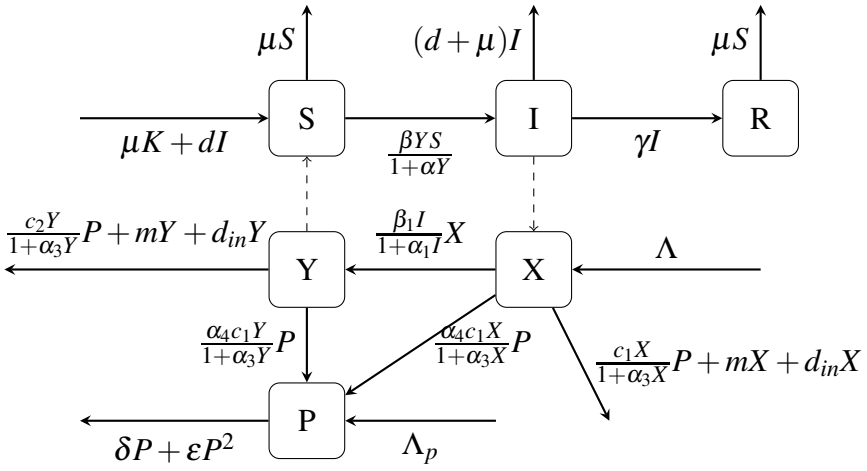

Figure 1: The above figure describes the interations between the plants, insects, and predators. A solid arrow toward a state represents an inflow of the given population. A solid arrow away from a population describes an outflow of the given population. A dotted line from one population to another illustrates a transmission of the virus.

The following is system of ordinary differential equations modeling the biological situation as outlined in the flow diagram.

$$\begin{aligned}
\frac{dS}{dt} &= \mu(K-S) + dI - \frac{\beta Y}{1+\alpha Y}S \\
\frac{dI}{dt} &= \frac{\beta Y}{1+\alpha Y}S - (d+\mu+\gamma)I \\
\frac{dX}{dt} &= \Lambda - \frac{\beta_1 I}{1+\alpha_1 I}X - \frac{c_1 X}{1+\alpha_3 X}P - mX - d_{in}(t)X \\
\frac{dY}{dt} &= \frac{\beta_1 I}{1+\alpha_1 I}X - \frac{c_2 Y}{1+\alpha_3 Y}P - mY - d_{in}(t)Y \\
\frac{dP}{dt} &= \Lambda_p + \frac{\alpha_4 c_1 X}{1+\alpha_3 X}P + \frac{\alpha_4 c_2 Y}{1+\alpha_3 Y}P - \delta P - \varepsilon P^2
\end{aligned}$$

Our goal is to minimize the cost of insecticide, predators and infected plants. To achieve such goals, we use the above equations as constraints to an objective function.

3. Optimal Control with Pontryagin's Principle

We consider the case when insecticide is the only control. We want to minimize the cost functional

$$\int_0^T AI(t)^2 + Gd_{in}(t)^2 dt$$

with respect to $d_{in}(t)$ and subject to the constraints

$$\begin{aligned}
\frac{dS}{dt} &= \mu(K-S) + dI - \frac{\beta Y}{1+\alpha Y}S \\
\frac{dI}{dt} &= \frac{\beta Y}{1+\alpha Y}S - (d+\mu+\gamma)I \\
\frac{dX}{dt} &= \Lambda - \frac{\beta_1 I}{1+\alpha_1 I}X - \frac{c_1 X}{1+\alpha_3 X}P - mX - d_{in}(t)X \\
\frac{dY}{dt} &= \frac{\beta_1 I}{1+\alpha_1 I}X - \frac{c_2 Y}{1+\alpha_3 Y}P - mY - d_{in}(t)Y \\
\frac{dP}{dt} &= \frac{\alpha_4 c_1 X}{1+\alpha_3 X}P + \frac{\alpha_4 c_2 Y}{1+\alpha_3 Y}P - \delta P - \varepsilon P^2
\end{aligned}$$

Here, A represents the cost of an infected plant, G represents the cost of insecticide, and T represents the total time period. Notice that we are minimizing the functional using the differential equations in 1 as the constraints.

To solve our problem, we need some theory. Consider a problem with $\vec{x} = [x_1, \ldots x_n]$ state variables,$\vec{u} = [u_1, \ldots u_m]$ control variables, and a payoff function ϕ,

$$\min_{\vec{u}} \int_{t_0}^{t_1} f(t, \vec{x}(t), \vec{u}(t) dt + \phi \vec{x}(t_1)$$

subject to

$$\vec{x}'(t) = \vec{g}(t, \vec{x}(t), \vec{u}(t)), \quad \vec{x}(t_0) = \vec{x}_0$$

where f and $\vec{g}$ are continuously differentiable in all variables.

Define $\vec{u}^*$ to be a vector of optimal control functions and $\vec{x}^*$ be the vector of corresponding optimal state values and define the Hamiltonian by

$$H(t,\vec{x},\vec{u},\vec{\lambda}) = f(t,\vec{x},\vec{u}) + \vec{\lambda(t)} \cdot \vec{g}((t,\vec{x},\vec{u}))$$

where $\lambda_i(t)$ is the adjoint for each x_i for $i = 1,..,n$. Note $\vec{u}^*$ maximizes $H(t,\vec{x},\vec{u},\vec{\lambda})$.
Moreover, $\vec{u}^*$, $\vec{x}^*$, and $\vec{\lambda}$ satisfy

$$\begin{aligned} x_i'(t) &= \frac{\partial H}{\partial \lambda_i} = g_i(t,\vec{x},\vec{u}), \quad x_i(t_0) = x_{i0} \text{ for } i = 1,...,n \\ \lambda_j' &= \frac{\partial H}{\partial x_j}, \quad \lambda_j(t_1) = \phi_{x_j}(\vec{x}(t_1)) \text{ for } j = 1,...,n \\ 0 &= \frac{\partial H}{\partial u_k} \text{ at } u_k^* \text{ for } k = 1,...,m \end{aligned}$$

By satisfing these conditions, we will determine our optimal solution. [7]
The Hamiltonian for the system is

$$H = AI(t)^2 + Gd_{in}(t)^2 + \lambda_1(\mu(K-S) + dI - \tfrac{\beta Y}{1+\alpha Y}S) + \lambda_2(\tfrac{\beta Y}{1+\alpha Y}S - (d+\mu+\gamma)I) + \lambda_3(\Lambda - \tfrac{\beta_1 I}{1+\alpha_1 I}X - \tfrac{c_1 X}{1+\alpha_3 X}P - mX - d_{in}(t)X)$$

$$+\lambda_4(\tfrac{\beta_1 I}{1+\alpha_1 I}X - \tfrac{c_2 Y}{1+\alpha_3 Y}P - mY - d_{in}(t)Y) + \lambda_5(\Lambda_p + \tfrac{\alpha_4 c_1 X}{1+\alpha_3 X}P + \tfrac{\alpha_4 c_2 Y}{1+\alpha_3 Y}P - \delta P - \varepsilon P^2)$$

The Adjoint Equations

$$\lambda_1' = \tfrac{\partial H}{\partial S} = \lambda_1(-\mu - \tfrac{\beta Y}{1+\alpha Y}) + \lambda_2 \tfrac{\beta Y}{1+\alpha Y}$$

$$\lambda_2' = \tfrac{\partial H}{\partial I} = 2AI + \lambda_1 d - \lambda_2(d+\mu+\gamma) - \lambda_3 \tfrac{\beta_1 X}{(1+\alpha_1 I)^2} - \lambda_4 \tfrac{\beta_1 X}{(1+\alpha_1 I)^2}$$

$$\lambda_3' = \tfrac{\partial H}{\partial X} = \lambda_3(-\tfrac{\beta_1 I}{1+\alpha_1 I} - \tfrac{cP}{(1+\alpha_3 X)^2} - m - d_{in}) + \lambda_4 \tfrac{\beta_1 I}{1+\alpha_1 I} + \lambda_5 \tfrac{c\alpha_4 P}{(1+\alpha_3 X)^2}$$

$$\lambda_4' = \tfrac{\partial H}{\partial Y} = \tfrac{-\lambda_1 \beta S}{(1+\alpha Y)^2} + \tfrac{\lambda_2 \beta S}{(1+\alpha Y)^2} + \lambda_4(-m - d_{in} - \tfrac{cP}{(1+\alpha_3 Y)^2}) + \lambda_5(\tfrac{c\alpha_4 P}{(1+\alpha_3 Y)^2})$$

$$\lambda_5' = \frac{\partial H}{\partial P} = \frac{-\lambda_3 cX}{1+\alpha_3 X} - \frac{\lambda_4 cY}{1+\alpha_3 Y} + \lambda_5(-\delta - 2\varepsilon P + \frac{\alpha_4 cX}{1+\alpha_3 X} + \frac{\alpha_4 cY}{1+\alpha_3 Y})$$

We must satisfy the optimality condition

$$\frac{\partial H}{\partial d_{in}} = 2d_{in}G - \lambda_3 X - \lambda_4 Y = 0$$

The forward-backward sweep method for several variables used to solve the previous differential equations. The method uses Euler for systems. An initial guess of each control variable is made then, all states x_i are simultaneously solved forward in time. Afterwards all adjoints λ_i are simultaneously solved backward in time. Each control u_i^* is updated subject to its individual characterization. The process is repeated until convergence occurs. The following figure shows some numerical results using the above method.

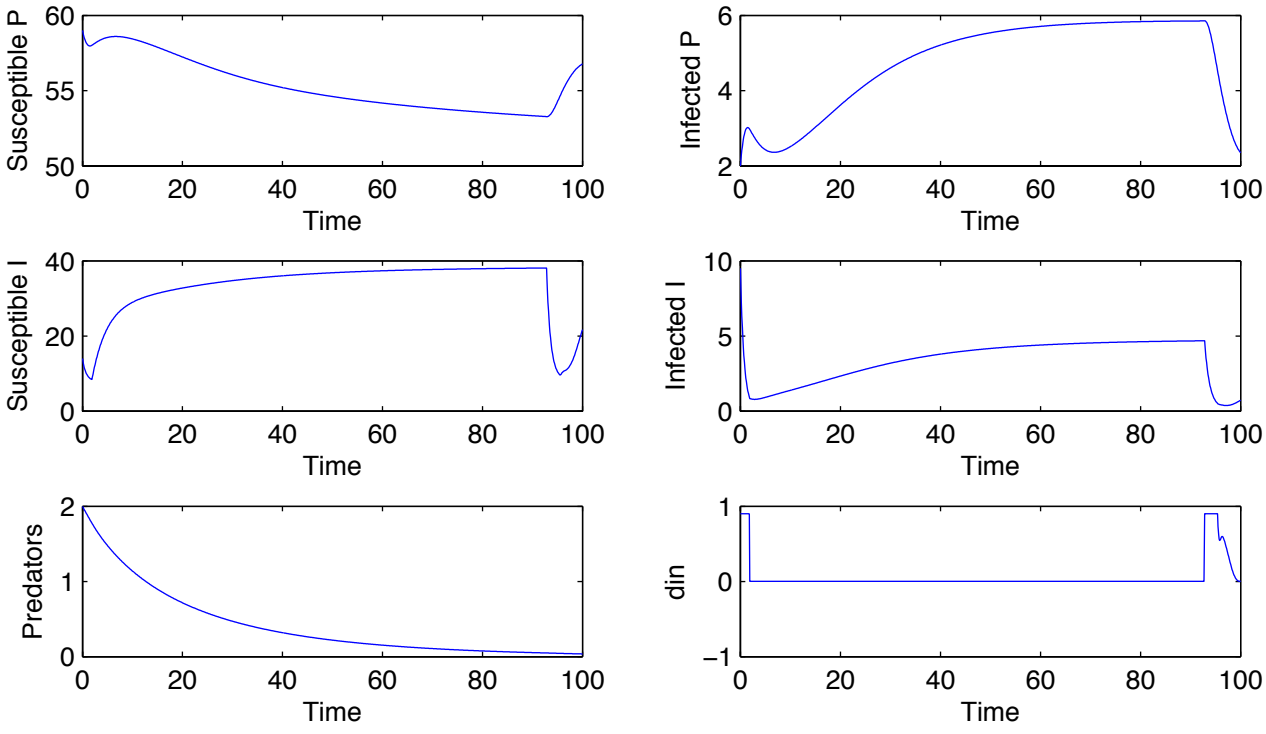

Figure 2: The above graphs show the dynamics of the system as well as the optimal function for the insecticide d_in in the interval [0,0.9]

Notice that when insecticide is present, both infected populations decreases. However, there are convergence issues at the endpoints of the insecticide. This illustrates one of the problems using an indirect methods. The challenge with using this method is that the numerical method to solve the system of constraints and adjoints is highly sensative to parameters and time interval, meaning that the method is not very robust.

4. Direct Methods for solving the Optimization Problem

Due to the sensitivity of the indirect method, Pontryagin's principle, we now consider a direct method to solve the problem. The idea is to discretize the control problem, then apply Nonlinear Programming (NLP) techniques to the resulting finite-dimensional optimization problem. They have been proved successful for many complex applications and take advantage of the power of state-of-the-art NLP solvers see [1], [2], [3]. They can be applied to ODE's, Discrete systems, DAE's, PDE's, delay equations, etc. The three main variants are: direct simultaneous approach, direct sequential approach, direct multiple-shooting approach.

Direct collocation method principle: convert optimal control problem into a finite-dimensional NLP through discretization of both control and state variables. State collocation:

$$x(t) = X_k(t), t_{k-1} \leq t_k, k = 1, \ldots, n_s$$

using Lagrange Polynomials of degree N. Very large-scale NLP problems in the variables which are the coefficients of the Lagrange polynomials. The numbers of time stages and collocation points as well as the position of the collocation points must be chosen prior. Path constraints are easily accommodated by enforcing inequality constraint at the collocation points.

Direct sequential method principle:Convert into a finite-dimensional NLP through discretization of the control variables only, while the differential equations are embedded in the NLP problem. That is, minimize the objective function with respect to the coefficients of the collocation

of u subject to the dynamic, path and boundary condition constraints ODE/DAE integrator + NLP solver.

There are several software packages for direct methods three of which are CasADi, ACADO toolkit and BOCOP. CasADi utilizes Python and MATLAB/Octave. It is a general-purpose tool for gradient-based numerical optimization with a strong focus on optimal control. Implements all three direct methods. Not trivial to extend to several controls and delay equations as delay differential integrator must be coded by the user[3]. ACADO toolkit utilizes C++ and Matlab. It is a software environment and algorithm collection for automatic control and dynamic optimization. Easy to use with multiple controls but no delay equation integrator [1]. Can also be used for parameter estimation and sensitivity analysis. BOCOP uses C++ and includes a GUI. Uses direct sequential method. Solves problems with multiple controls and delay equations, but one has to write several routines [2].

Direct methods have the advantage over indirect methods in that they are more straightforward to apply and more robust with respect to the initialization. The cost, however, is that some precision is lost. [2]. BOCOP first discretizes the dynamics equation and state and control variables using a method of the users choice. It then utilizes the NLP solver IPOPT which implements a primal-dual interior point algorithm. For information about the algorithm see [10]. To descretize our problem, we chose an implicit Gaussian integrator of order 4 with 1000 subintervals between 0 and 365. The discritization was then passed to IPOPT optimizer, and the following figures show the numerical results.

Notice that in figure 4 for the solution for the rate of insecticide introduced decreases with time, Contrasting this with the solution for the insecticide using indirect methods the rate of insecticide decreases sharply, remains constant, then increases sharply. Also, the infected plants and insects are close to zero as seen in figure 3 for the optimal solution.

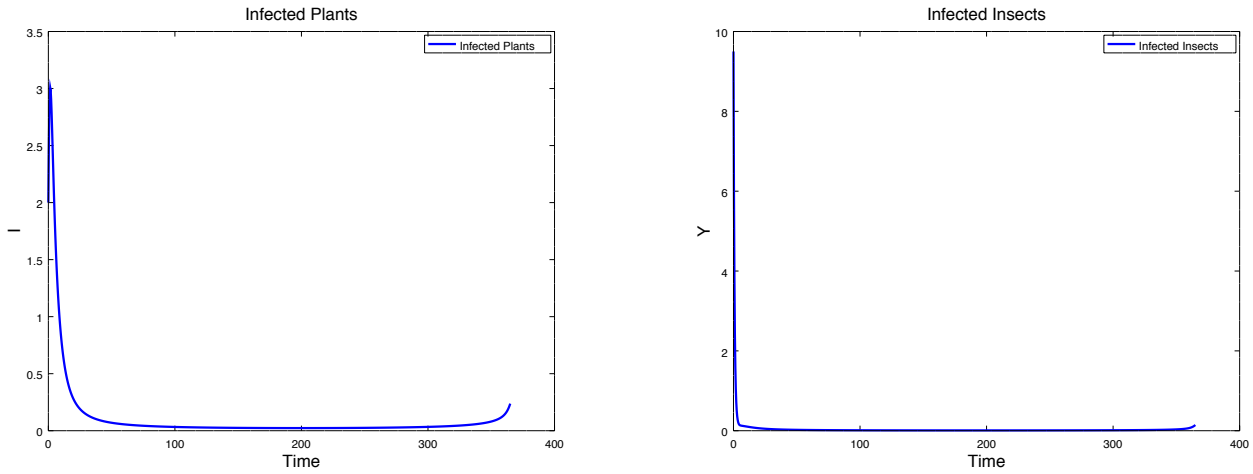

Figure 3: The above figures show the infected plants, I, and infected insects Y for the optimal function

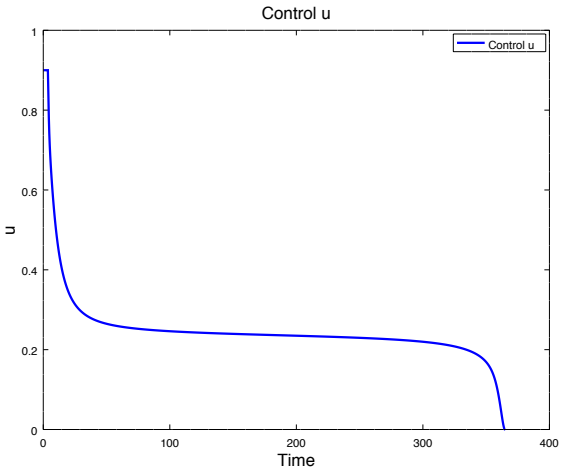

Figure 4: The above graphs show the dynamics of the system as well as the optimal function for the insecticide $d_i n$ in the interval [0,0.9]

5. Direct Methods solving the Periodic Optimization Problem

We now extend the problem to include seasonality. Due to the periodic nature of insects, active in the warm months and dormant in the cooler ones, we introduce periodic coefficients to the constraint equations. Specifically, we include periodic contact rates between the insects and the plants. Thus, the optimization problem is as follows:

$$\min_{d_{in}(t),\Lambda_p} \int_0^T AI(t)^2 + Gd_{in}(t)^2 + F\Lambda_p^2 dt$$

subject to

$$\begin{aligned}
\frac{dS}{dt} &= \mu(K-S) + dI - \frac{\beta(t)Y}{1+\alpha Y}S \\
\frac{dI}{dt} &= \frac{\beta(t)Y}{1+\alpha Y}S - (d+\mu+\gamma)I \\
\frac{dX}{dt} &= \Lambda - \frac{\beta_1(t)I}{1+\alpha_1 I}X - \frac{c_1 X}{1+\alpha_3 X}P - mX - d_{in}(t)X \\
\frac{dY}{dt} &= \frac{\beta_1(t)I}{1+\alpha_1 I}X - \frac{c_2 Y}{1+\alpha_3 Y}P - mY - d_{in}(t)Y \\
\frac{dP}{dt} &= \Lambda_p + \frac{\alpha_4 c_1 X}{1+\alpha_3 X}P + \frac{\alpha_4 c_2 Y}{1+\alpha_3 Y}P - \delta P - \varepsilon P^2
\end{aligned}$$

where

$$\beta(t) = \beta(1+h\cos(\frac{2\pi t}{365})) \qquad \beta_1(t) = \beta_1(1+h\cos(\frac{2\pi t}{365})). \tag{1}$$

To descretize our problem, we chose an implicit Gaussian integrator of order 4 with 1000 subintervals between 0 and 365. The discritization was then passed to IPOPT optimizer, and the following figures show the numerical results.

From figure 6, notice that both controls are necessary for an optimal solution that minimizes the infected plants and insects shown in figure 5. Moreover, the infected plants and insects are both

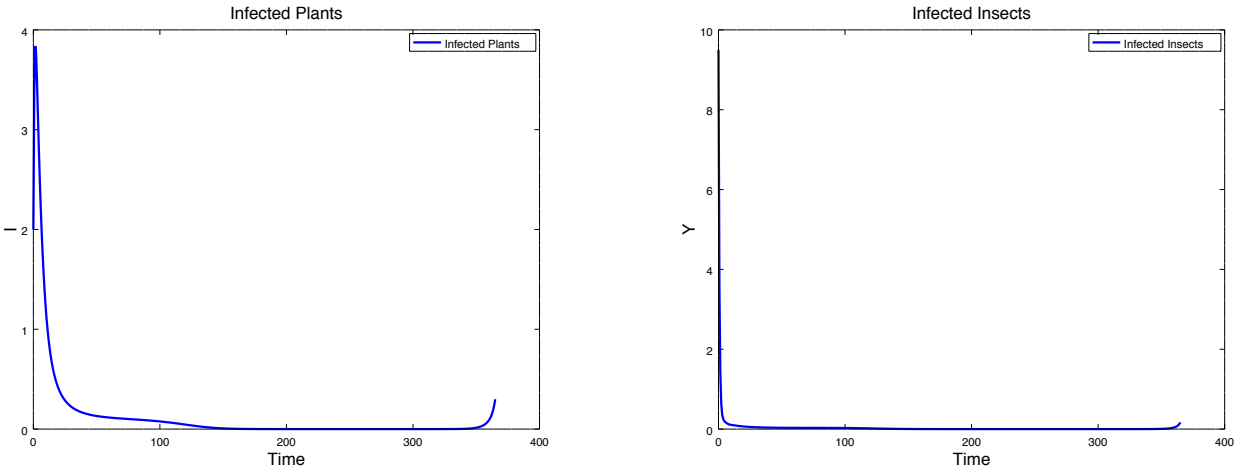

Figure 5: The above figures show the infected plants, I, and infected insects Y for the optimal function

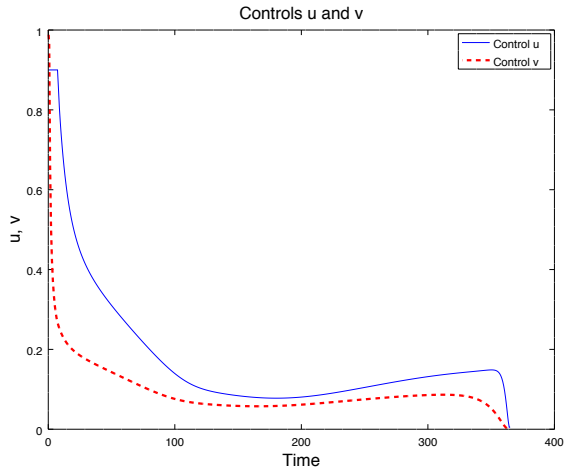

Figure 6: The above graphs show the insecticide and amount of predators to introduce that minimize the cost functional

close to zero for the optimal solution.

6. Direct Methods solving the Delay Optimization Problem

Since it takes time for the virus to spread throughout the plant and insect, we now consider an optimal control problem with delays. Let τ_1 be the time it takes a plant to become infected after contagion and τ_2, to be the time it takes a vector to become infected after contagion. Then the problem with the two discrete delays is

$$\min_{d_{in}(t),\Lambda_p} \int_0^T AI(t)^2 + Gd_{in}(t)^2 + F\Lambda_p^2 dt$$

subject to

$$\begin{aligned}
\frac{dS}{dt} &= \mu(K-S) + dI - \frac{\beta(t)Y(t-\tau_1)}{1+\alpha Y(t-\tau_1)}S(t-\tau_1) \\
\frac{dI}{dt} &= \frac{\beta(t)Y(t-\tau_1)}{1+\alpha Y(t-\tau_1)}S - (d+\mu+\gamma)I \\
\frac{dX}{dt} &= \Lambda - \frac{\beta_1(t)I(t-\tau_2)}{1+\alpha_1 I(t-\tau_2)}X(t-\tau_2) - \frac{c_1 X}{1+\alpha_3 X}P - mX \\
\frac{dY}{dt} &= \frac{\beta_1(t)I(t-\tau_2)}{1+\alpha_1 I(t-\tau_2)}X(t-\tau_2) - \frac{c_2 Y}{1+\alpha_3 Y}P - mY \\
\frac{dP}{dt} &= \frac{\alpha_4 c_1 X}{1+\alpha_3 X}P + \frac{\alpha_4 c_2 Y}{1+\alpha_3 Y}P - \delta P - \varepsilon P^2
\end{aligned} \tag{2}$$

To solve this problem, we again use the BOCOP software. In addition to solving optimal control problems with ODE constraints, BOCOP can handle problems whose dynamics are of the form:

$$y'(t) = f(t,u(t),y(t),u(t-\tau_1),...,u(t-\tau_M),y(t-\tau_{M+1},...,u(t-\tau_{M+N}))$$

where y_i are the state variables and u_i are the controls. The values of $y_i(t-\tau)$ or $u_i(t-\tau)$ can be computed by interpolation on the time steps or stages respectively. To descretize our problem, we

chose an implicit Gaussian integrator of order 4 with 1000 subintervals between 0 and 365. The discritization was then passed to IPOPT optimizer, and the following figures show the numerical results. The figures 7 and 8 show the resuls is when G= 1, A=1, F=1. Figures 9 and 10 show the results G= 4, A=1, F=1.

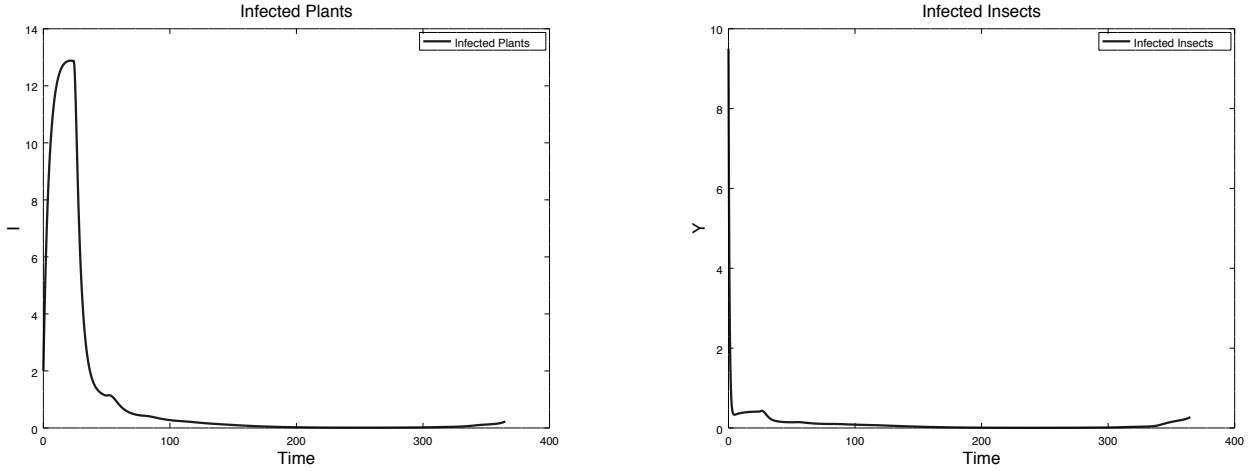

Figure 7: The above figures show the infected plants, I, and infected insects Y for the optimal function when G= 1, A=1, F=1

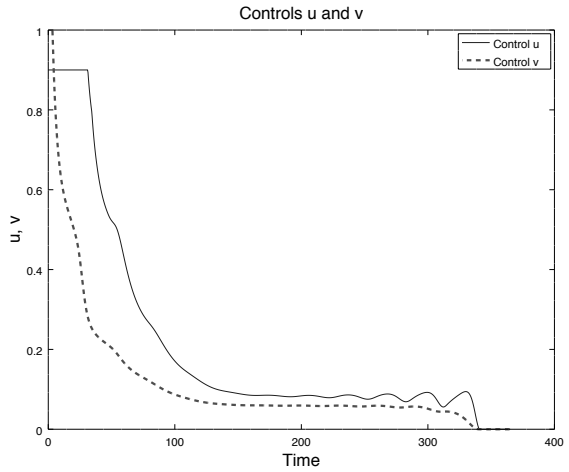

Figure 8: The above graphs show the insecticide and amount of predators to introduce that minimize the cost functional when G= 1, A=1, F=1

These figures demonstrate that a both controls are necessary for an optimal solution. Also, notice that as the cost of the insecticides change, the controls for the optimal solution also change.

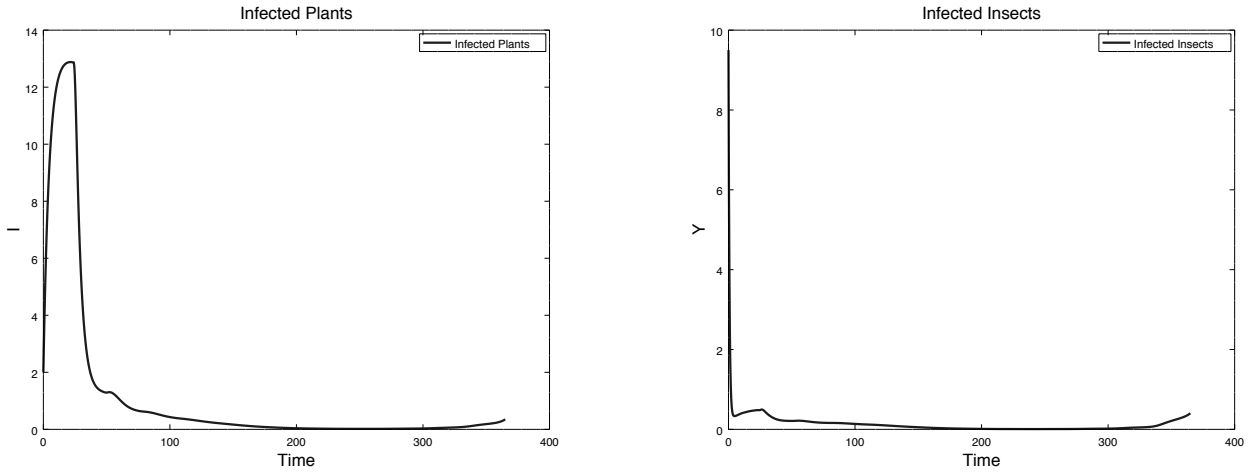

Figure 9: The above figures show the infected plants, I, and infected insects Y for the optimal function when G= 4, A=1, F=1

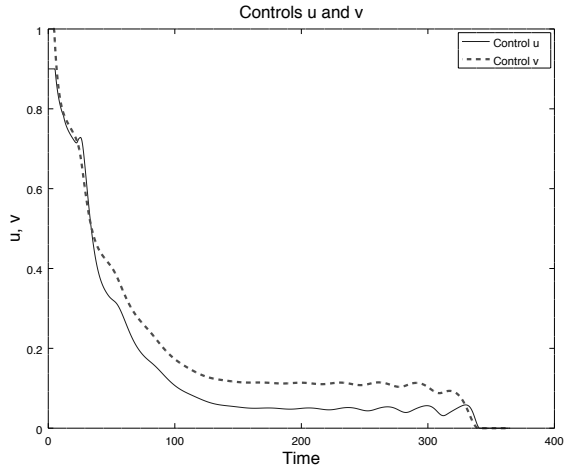

Figure 10: The above graphs show the insecticide and amount of predators to introduce that minimize the cost functional when G= 4, A=1, F=1

In figure 8, when G = 1, the rate of insecticide introduced, u, is higher than the rate of predators introduced, v. However, in figure 10, we see that when the cost of insecticide increases to G = 4, rate of insecticide introduced is less than rate of predators. Hence relative costs are important.

7. Conclusions

For our particular models, the direct methods from BOCOP are more robust than the indirect methods using Pontryagin maximum principle. Moreover, introducing periodicity and delays makes the model more realistic at the cost of making the model more complex. However, with the BOCOP software, we are able to view the state values at the optimal solution for specific parameter values. So this will encourage farmers to measure the parameters necessary to determine an optimal cost for a particular situation. Also, relative costs are important. As the cost for pesticides and predators change, While the simulations only provide specific examples, they can give insight to farmers who want to minimize the cost of predators and pesticide.

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Chapter 6
Conclusions

In this dissertation, plant virus interactions were modeled by both ordinary and delay differential equations, a predator was introduced to combat the spread of the vectors, periodicity in the coefficients was introduced to study the effects of seasonality, and optimal control problems to minimize the cost of predators and insecticide were studied. When modeling the using the delay differential equations, we create a more realistic model than with ordinary differential equations. We notice that the ODE and DDE solutions are different. In the study of the models, we the stability of the disease free equilibrium is dependent upon the parameters of the model. Moreover, the stability analysis had to be done numerically for the DDE compared to the ODE. Introducing a predator showed to be an effective method for eliminating the vectors that spread the virus. The challenge with studying stability of equilibria is that there are many parameters that influence the model. Thus numerical simulations must be done in order to understand the dynamics. By introducing seasonality, the model is made more realistic. To study the dynamics of the model, the basic reproductive numer was used. There were two approaches: time average and linear operator method. The linear operator method shows to be a better approach as evidenced by the numerical results. For the optimal control problem, we used two approaches to solve: direct and indirect methods. The indirect methods had difficulties converging, but the direct methods worked well.