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Modelling and Optimal Control of Insect Transmitted Plant Disease

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Abstract

Insect-vectored diseases pose one of the greatest threats to plants on a global scale. At present, few effective control strategies have been developed to prevent the transmission of insect-transmitted diseases. These strategies largely rely on the use of chemical insecticides which have negative impacts on the environments and human health. In this study, a mathematical model is formulated and analysed to study the optimal control of the insects transmitted plants diseases. The model is sub-divided into two sub-populations namely the plant population and the insect population. The plant population is divided into two classes, namely; susceptible plants and infected plants and vector (insect) population comprises susceptible vector and infected vector. The optimal control model is formulated and analysed to minimize the transmission of disease from an infected vector (insect) to susceptible plant. Optimal control method using Pontryagin's Maximum Principle was applied to determine the necessary conditions for the optimal control of the impact of plant inoculation. It is concluded that, if the plants are controlled then more plants will be produced compared with plants without control.

1. Introduction

Plant disease is an abnormal and harmful physiological condition brought by living (biotic) agents, such as fungi, bacteria, nematodes and virus, or by non-living (abiotic) factors, such as nutrient deficiencies and water stress [17]. The occurrence and prevalence of plant disease vary from season to season, depending on the interaction of pathogens, environmental conditions and crops [20].

Vectors are organisms that can introduce a pathogen such as a bacterium or virus into a plant to cause an infection. Insects, mites, and nematode vectors focus the movement of plant pathogens among immobile plants [10]. In the late 18th century, there were many scientists who began to research the essence of plant diseases [19]. Much research on vector transmission seeks to understand the transmission process so as to explain why only certain kinds of insects or mites can serve as vectors and to identify what factors are required for transmission [10]. Rice gall dwarf virus, Tomato yellow leaf curl geminivirus, Africa cassava mosaic geminivirus are some of examples of plant diseases which are transmitted by insects. Some studies have been carried out to analyse the control and transmission dynamics of insect transmitted plant disease. Cultural strategy includes replanting, and/or removing diseased plants [9, 11]. In crop production system people use agro-chemicals to eradicate plant disease [13]. Another disease control is by inducing resistance to plant which will enable the plant not to be infected even if the pathogen invades [1].

Mathematical modeling of the spread of infectious diseases continues to provide important insights into diseases behavior and control. Mathematical models of plant-virus (or pathogens) disease epidemics were developed to provide a detailed exposition on how to describe, analyse, and predict epidemics of plant disease for the ultimate purposes of developing and testing control strategies and tactics for crop protection [4]. Over the years, it has also become an important tool in understanding the dynamics of diseases and in decision making processes regarding intervention programs for controlling these diseases in many countries

Meng *et al.*, [14] did a research on the dynamics of plant disease model with continuous and impulsive cultural control strategies. The model comprised of susceptible and infected plants. They found that impulsive removing of diseased plants is more efficient and more economical than continuous removing. But they did not attempt to design optimal control strategies for disease control.

Abdullatif *et al.*, [1] conducted a research on Modelling induced resistance to plant diseases. The study analyzed the plant control by inducing resistance to plant which will enable the plant not to be infected even if the pathogens invade.

Cunniffe *et al.*, [6] performed a research on the efficacies of two cultural control measures; namely, spacing of host plant and rouging symptotic trees. They found that it was important to reduce plant density in order to slow down the spread of disease significantly if the distance between hosts is sufficiently large.

Cheryl *et al.*, [5] considered the economics of controlling insect-transmitted plant disease. Biological control methods for controlling plant diseases in wine grapes were examined using a spacial model of a disease transmitted by insects from host plants in an adjacent source area by knowing behaviour of the insect carrier, and the ability of insect to transmit the disease.

Nakazawa *et al.*, [15] analysed a model for plant disease dynamics co-mediated by herbivore and herbivore-borne phytopathogens to have better understanding of plant disease dynamics. The results of the model highlight the importance of the eco-epidemiological perspective that was integration of tripartite interactions among host plant, plant pathogen and herbivore which is crucial for the successful control of plant diseases.

The plant diseases which are transmitted by insect have been a problem in agricultural products. Some studies which have been carried out in plant disease control have failed to overcome the problem since the diseases are still causing the reduction of the yield and quality of harvested crops. However, none of these studies have considered the aspect of optimal control to reduce the spread of the insect transmitted plant disease. The goal of this research is to model and optimise the control of insect transmitted plant diseases by minimising the rate of transmission of disease from infected insects (vector) to susceptible plants (host). Therefore, this study intends to apply optimal control theory to minimize the spread disease by some control strategies and minimize the cost of applying controls, in order to best combat the spread of insect transmitted plant disease.

2. Model Formulation

This context of insect transmitted plant disease, grouped in plants and insects then two epidemiological stages are identified in each group. Plants: susceptible plants (P) and infected plants (F) and Insects: infected insects (I) and susceptible insects (S). The interaction of the groups/classes will cause the spread of the disease due to insects in which the population increases logistically with the rate (r) and the carrying capacity indicated by parameter (K). Insects are affected by disease from infected plants by the rate of transmission (β), and will leave the population due to disease with the rate (δ) also insect will die naturally with the rate (μ_2). The recruitment of new plants into the farm will be indicated by (Q). Plants are diseased by insects with the rate (a) and the diseased plants will die by the rate (m). Some of the diseased (infected) plants are recovered at the rate (b) and go back to susceptible plants and then susceptible plants will be harvested at the rate (μ_1). The new infections occur during the interaction between susceptible plants and infected insects and then infected plants and susceptible insects. We are going to minimize the transmission by increasing the disease resistance to the plants through plants inoculation.

In formulating the model, the following assumptions are taken into consideration:

The susceptible plant into the farm is due to recruitment (plantation of new plants). The susceptible Insects increase logistically. The infected Insects are due to infected plants and vice versa. The transmission is due to contact between infected plants (Host) and susceptible plants (Host). Vectors which are infected (and thus infective to hosts) do not recover (thus remain infective). The plants and insects populations are not constant. No interaction or contacts between plant and plant for transmission of the disease.

In view of the above considerations and assumptions, we have the following flow diagram illustrates the interactions of the different population.
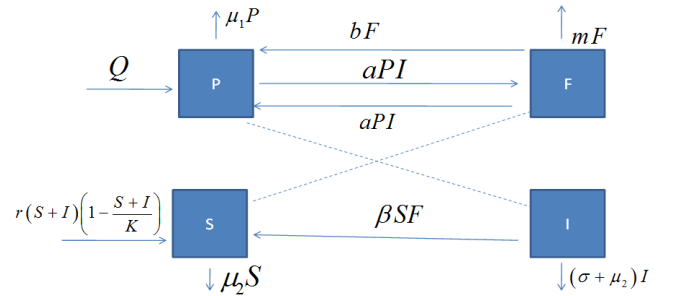

Figure 1. *Compartmental of insect transmitted plant disease.*

From the descriptions and compartmental diagram we have the following as the system of equations:

$$\frac{dP}{dt} = Q - aPI + bF - \mu_1 P$$

$$\frac{dF}{dt} = aPI - (b+m)F \qquad (1)$$

$$\frac{dS}{dt} = r(S+I)\left(1 - \frac{S+I}{K}\right) - \beta SF - \mu_2 S$$

$$\frac{dI}{dt} = \beta SF - (\delta + \mu_2) I$$

where $S + I < K$, aPI is the product of the rate of transmission of disease between the infected insects and

susceptible plants, $r(S+I)\left(1-\dfrac{S+I}{K}\right)$ is the logistic growth of the insect at a particular environment, $(S+I)$ is the total number of insect and the carrying capacity K should not be equal to number of insects present. The non-negative initial conditions of the system (1) are $P(0)\geq 0,\ F(0)\geq 0,\ S(0)\geq 0$ and $I(0)\geq 0$. Population of insects is not constant and plant population also is not constant, then there is interaction between insects and plants.

3. Model Analysis

The system of equations (1) analysed qualitatively to get insight into its dynamical features which will give a better understanding of the effects of control strategies on the insect transmitted plant disease. For the model (1) to be epidemiological meaningful, it is important to prove that all solutions with non- negative initial data will remain non-negative for all the time.

3.1. Positivity of the Solutions

Lemma 1: *Let the initial data be* $\left\{(P(0),F(0),S(0),I(0))\geq 0\right\}\in \Omega$. *Then, the solution set* $\left\{P(t),F(t),S(t),I(t)\right\}$ *of the model system (1) is positive for all* $t\geq 0$.

Proof: By considering the plant population then the equation of susceptible plant which is the first equation of the system (1), we have

$$\frac{dP}{dt}=Q-aPI+bF-\mu_1 P$$
$$\geq -[aI+\mu_1]P$$

Integration yields

$$P(t)\geq P(0)e^{-[aI+\mu_1]t}\geq 0,\ \ \forall t\geq 0 \qquad (2)$$

Similarly, for the remaining equations of the system (1), to get

$$\frac{dF}{dt}=aP-(b+m)F$$
$$\geq -(b+m)F$$
$$\Rightarrow F(t)\geq F(0)e^{-(b+m)t}\geq 0,\ \ \forall t\geq 0 \qquad (3)$$

For the case of insect, when we consider the susceptible insect in equation three in the model (1)

$$\frac{dS}{dt}=r(S+I)\left(1-\frac{S+I}{K}\right)-\beta FS-\mu_2 S\geq -(\beta F+\mu_2)S$$

$$\Rightarrow S(t)\geq S(0)e^{-(\beta F+\mu)t}\geq 0,\ \forall t\geq 0 \qquad (4)$$

Similarly, for the remaining equations of the system (1)

$$\frac{dI}{dt}=\beta SF-(\delta+\mu_2)I$$
$$\geq -(\delta+\mu_2)I$$
$$\Rightarrow I(t)\geq I(0)e^{-(\delta+\mu_2)t}\geq 0 \qquad (5)$$

From above proof it is clear that, the solution set $\left\{P(t),F(t),S(t),I(t)\right\}$ of the model (1) is positive for all $t\geq 0$.

3.2. Disease Free Equilibrium Point

The disease free equilibrium point is obtained by setting the system of equations (1) equal to zero, to get

$$\frac{dP}{dt}=Q-aPI+bF-\mu_1 P=0 \qquad (6)$$

$$\frac{dF}{dt}=aPI-(b+m)F=0 \qquad (7)$$

$$\frac{dS}{dt}=r(S+I)\left(1-\frac{S+I}{K}\right)-\beta SF-\mu_2 S=0 \qquad (8)$$

$$\frac{dI}{dt}=\beta SF-(\delta+\mu_2)I=0 \qquad (9)$$

From equation (6)

$$\frac{dP}{dt}=Q-aPI+bF-\mu_1 P=0$$

This implies that $Q-\mu_1 P=0$ and thus get

$$P=\frac{Q}{\mu_1} \qquad (10)$$

Then from equation (8), it follows that

$$\frac{dS}{dt}=r(S+I)\left(1-\frac{S+I}{K}\right)-\beta SF-\mu_2 S=0$$

$$r(S)\left(1-\frac{S}{K}\right)-\mu_2 S=0$$

$$r(S)\left(1-\frac{S}{K}\right)=\mu_2 S$$

$$r\left(1-\frac{S}{K}\right)=\mu_2$$

We then obtain

$$S=\frac{K}{r}(r-\mu_2) \qquad (11)$$

Therefore, the Disease Free Equilibrium (DFE) of the model system (1) is given by

$$(P,F,S,I) = \left(\frac{Q}{\mu_1}, 0, \frac{K}{r}(r-\mu_2), 0 \right) \tag{12}$$

3.3. Local Stability of the Disease Free Equilibrium (DFE)

Determination of the local stability of DFE depends on the basic reproduction number, R_0, defined as the average number of secondary infected individuals produced by single infectious individual during the course of his/her infectiousness typically in susceptible population [7]. By considering the system (1), the reproduction number can be easily computed using the next generation matrix approach as used by [8]. Reproduction number (R_0) is determined by taking the infective classes and considering the disease free equilibrium.

$$(P, F, S, I) = \left(\frac{Q}{\mu_1}, 0, \frac{K}{r}(r-\mu_2), 0 \right)$$

The infections population is to be taken from system (1), which are:

$$\frac{dF}{dt} = aPI - (b+m)F$$

$$\frac{dI}{dt} = \beta SF - (\delta + \mu_2)I$$

The operator G_i, reflects the rate at which new infections arise and the operator V_i, reflects the rate at which compartments corresponding to infection are exited

$$G_i = \begin{pmatrix} aPI \\ \beta SF \end{pmatrix} \text{ where } G_i = \begin{pmatrix} G_1 \\ G_2 \end{pmatrix} \tag{13}$$

and

$$V_i = \begin{pmatrix} (b+m)F \\ (\delta + \mu_2)I \end{pmatrix} \text{ where } V_i = \begin{pmatrix} V_1 \\ V_2 \end{pmatrix} \tag{14}$$

The Jacobian for the two infectious groups is as follows:

$$G = \begin{pmatrix} \frac{\partial G_1}{\partial F} & \frac{\partial G_1}{\partial I} \\ \frac{\partial G_2}{\partial F} & \frac{\partial G_2}{\partial F} \end{pmatrix} \text{ and } V = \begin{pmatrix} \frac{\partial V_1}{\partial F} & \frac{\partial V_1}{\partial I} \\ \frac{\partial V_2}{\partial F} & \frac{\partial V_2}{\partial I} \end{pmatrix}$$

then

$$G = \begin{pmatrix} 0 & aP \\ \beta S & 0 \end{pmatrix} \tag{15}$$

and

$$V = \begin{pmatrix} (b+m) & 0 \\ 0 & (\delta + \mu_2) \end{pmatrix} \tag{16}$$

Thus,

$$V^{-1} = \frac{1}{(b+m)(\delta+\mu_2)} \begin{pmatrix} (\delta+\mu_2) & 0 \\ 0 & (b+m) \end{pmatrix} \text{ or } V^{-1} = \begin{pmatrix} \frac{1}{(b+m)} & 0 \\ 0 & \frac{1}{(\delta+\mu_2)} \end{pmatrix} \tag{17}$$

Then

$$K = GV^{-1}$$

$$K = \begin{pmatrix} 0 & aP \\ \beta S & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{(b+m)} & 0 \\ 0 & \frac{1}{(\delta+\mu_2)} \end{pmatrix} \Rightarrow K = \begin{pmatrix} 0 & \frac{aP}{(\delta+\mu_2)} \\ \frac{\beta S}{(b+m)} & 0 \end{pmatrix} \tag{18}$$

$$|K - \lambda I| = \begin{vmatrix} -\lambda & \frac{aP}{(\delta+\mu_2)} \\ \frac{\beta S}{(b+m)} & -\lambda \end{vmatrix} \Rightarrow \lambda^2 - \frac{aP\beta S}{(b+m)(\delta+\mu_2)} = 0 \tag{19}$$

When we introduce the value of DFE

$$(P, F, S, I) = \left(\frac{Q}{\mu_1}, 0, \frac{K}{r}(r-\mu_2), 0 \right)$$

to the above quadratic equation we get

$$\lambda^2 - \frac{(r-\mu_2)a\beta QK}{r\mu_1(b+m)(\delta+\mu_2)} = 0 \tag{20}$$

Thus the eigenvalues are

$$\lambda_1 = \sqrt{\frac{(r-\mu_2)a\beta QK}{r\mu_1(b+m)(\delta+\mu_2)}} \tag{21}$$

and

$$\lambda_2 = -\sqrt{\frac{(r-\mu_2)a\beta QK}{r\mu_1(b+m)(\delta+\mu_2)}} \tag{22}$$

The reproduction number is obtained by taking the largest eigenvalue of the model system (1), so it is given by:

$$R_0 = \sqrt{\frac{(r-\mu_2)a\beta QK}{r\mu_1(b+m)(\delta+\mu_2)}} \tag{23}$$

If $R_0 < 1$, each individual (insect) transmit an average less than one new infected individual (plant) and hence diseases dies out. If $R_0 > 1$, each individual produces more than one new infected individual and hence the disease is able to invade the susceptible population. Following [8] we have the following result:

Theorem 1: *The disease free equilibrium of the system (1) is locally asymptotically stable if $R_0 < 1$ and unstable If $R_0 > 1$.*

3.4. Endemic Equilibrium Point Analysis

The existence and stability of endemic equilibrium is determined through the investigation of the possibility of existence of the backward or forward bifurcation due to existence of endemic equilibrium using the Centre Manifold Theory [3]. As a disease invades the population, it reduces the number of susceptible individuals in the population, which tends to reduce its reproductive rates. Figure 2 shows backward bifurcation for the chosen numerical data:

$$r = 0.5, \beta = 0.0005, \delta = 4.09, \mu_1 = 0.14,$$
$$K = 105, a = 3.994, m = 0.2, u = 0.5,$$
$$b = 9.999, \mu_2 = 0.002, Q = 100$$

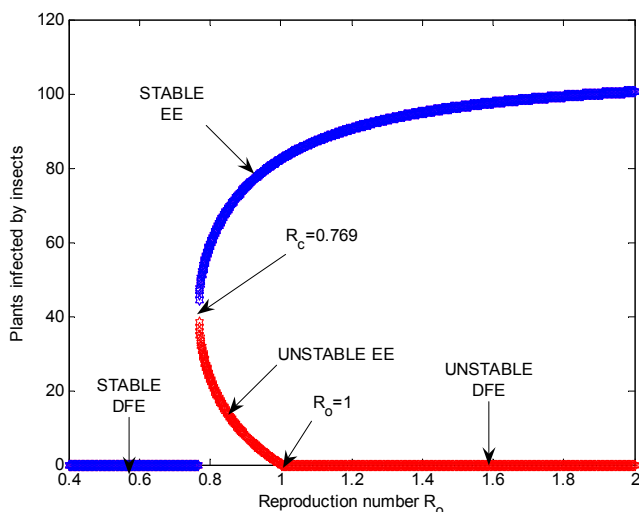***Figure 2.*** *The figure of plants infected by insects versus reproduction number R_0.*

The backward bifurcation implies that the reduction of R_0 below unity alone is not enough to eradicate insect transmitted plant disease, except when the initial cases of infection in both population is considered, a backward bifurcation is possible when the stable DFE co-exists with a stable endemic equilibrium for $R_0 < 1$ [2]. The biological implication of backward bifurcation is that the classical requirement of having the reproduction number less than unity, although necessary, is no longer sufficient for disease control. This implies that effective disease control is dependent on the initial sizes of the sub-population of the model [2]. However, this equilibrium may not be globally asymptotically stable for $R_0 < 1$.

4. Analysis of Optimal Control

In this section, we apply optimal control method using Pontryagin's Maximum Principle to determine the necessary conditions for the optimal control of the impact of plant inoculation. The system (1) is extended by incorporate time-dependent control $u(t)$ which is control measure due to plant inoculation for prevention of plant diseases. This control measure will control the rate of transmission from infected insects to susceptible plants. The model will be as follow;

$$\frac{dP}{dt} = Q - (1-u)aPI + bF - \mu_1 P$$

$$\frac{dF}{dt} = (1-u)aPI - (b+m)F \tag{24}$$

$$\frac{dS}{dt} = r(S+I)\left(1 - \frac{S+I}{K}\right) - \beta SF - \mu_2 S$$

$$\frac{dI}{dt} = \beta SF - (\delta + \mu_2)I$$

where $0 \le u(t) \le 1$ is the control on plant inoculation to reduce plant disease. The objective functional J is defined over a feasible set of control $u(t)$ applied over the finite time interval $\left[0, t_f\right]$ which is

$$J(u) = \min \int_{t_0}^{t_f}\left(\phi_1 P + \phi_2 F + \frac{1}{2}Bu^2(t)\right)dt \tag{25}$$

where ϕ_1, ϕ_2 are the costs associated with susceptible plants and infected plants respectively, while B is a relative cost weights for control measure. With the objective function $J(u)$, our goal is to minimize the number of infected plants, while minimizing the cost of control $u(t)$. Thus we thus seek an optimal control $u^*(t)$ such that

$$J\left(u^{*}\right)=\min\{J(u)\} \qquad (26)$$

The necessary conditions that an optimal control problem must satisfy come from Pontryagin's maximum principle [18]. This principle converts (25)-(28) into a problem of minimizing pointwise a Hamiltonian H, with respect to u defined by;

$$
\begin{aligned}
H = &\phi_1 P + \phi_2 F + \frac{1}{2}Bu^2(t) + \lambda_p\left[Q-(1-u)aPI+bF-\mu_1 P\right] + \lambda_F\left[(1-u)aPI-(b+m)F\right] \\
&+ \lambda_I\left[\beta SF-(\delta+\mu_2)I\right] + \lambda_S\left[r(S+I)\left(1-\frac{S+I}{K}\right)-\beta SF-\mu_2 S\right]
\end{aligned}
\qquad (27)
$$

Where λ_p, λ_F, λ_I and λ_s are adjoint variables or co-state variables.

Consider $\dfrac{\partial H}{\partial y}=-\dfrac{\partial \lambda}{\partial t}$

$$\frac{\partial H}{\partial P}=-\frac{d\lambda}{dt}=-\left(\phi_1+\lambda_p\left[-(1-u)aI-\mu_1\right]+\lambda_F(1-u)aI\right)$$

$$\frac{\partial \lambda_p}{\partial t}=\lambda_p\left[(1-u)aI-\mu_1\right]-\phi_1-\lambda_F(1-u)aI$$

$$\frac{\partial H}{\partial F}=-\frac{d\lambda_F}{dt}=-\left(\phi_2+\lambda_p b-\lambda_F(b+m)-\lambda_s\beta S+\lambda_I\beta S\right)$$

$$\frac{d\lambda_F}{dt}=\lambda_F(b+m)+\lambda_s\beta S-\phi_2-\lambda_I\beta S-\lambda_p b$$

$$\frac{\partial H}{\partial S}=-\frac{d\lambda_S}{dt}=-\left(\lambda_S\left(r-\frac{2r(S+I)}{K}-\beta F-\mu_2\right)+\lambda_I\beta F\right) \qquad (28)$$

$$\frac{d\lambda_S}{dt}=\lambda_S\left(-r+\frac{2r(S+I)}{K}+\beta F+\mu_2\right)-\lambda_I\beta F$$

$$\frac{\partial H}{\partial I}=-\frac{d\lambda_I}{dt}=-\left(-\lambda_p(1-u)Pa+\lambda_F(1-u)Pa+\lambda_s\left(r-\frac{2r(S+I)}{K}\right)-\lambda_I(\delta+\mu_2)\right)$$

$$\frac{d\lambda_I}{dt}=\lambda_I(\delta+\mu_2)+\lambda_p(1-u)Pa-\lambda_F(1-u)Pa-\lambda_s\left(r-\frac{2r(S+I)}{K}\right)$$

Transversality conditions

$$\lambda_p\left(t_f\right)=\lambda_F\left(t_f\right)=\lambda_S\left(t_f\right)=\lambda_I\left(t_f\right)=0 \quad \text{for } t_0\le t\le t_f \qquad (29)$$

Optimal control variable defined as:

$$u^{*}=\max\left(0,\min(1,u)\right)$$

$$\frac{\partial H}{\partial u}=Bu+\lambda_p aPI-\lambda_F aPI=0$$

$$Bu+\lambda_p aPI-\lambda_F aPI=0$$

$$u=\frac{1}{B}\left(\lambda_F-\lambda_p\right)aP$$

$$u^{*}=\max\left(0,\min\left(1,\frac{\left(\lambda_F-\lambda_p\right)aP}{B}\right)\right) \qquad (30)$$

By standard control arguments involving the bounds on the controls, we conclude:

$$u^{*}(t)=\begin{cases}u_{\min} & if \quad \dfrac{\partial H}{\partial u}<0 \\[2mm] u_s & if \quad \dfrac{\partial H}{\partial u}=0 \\[2mm] u_{\max} & if \quad \dfrac{\partial H}{\partial u}>0\end{cases} \qquad (31)$$

Due to the a priori boundedness of the state and adjoint functions and the resulting lipschitz structure of the ODEs, we obtain the uniqueness of the optimal control for small $\left[t_f \right]$ [12]. The uniqueness of the optimal control follows from uniqueness of the optimality system, which consists of (28) and (29) with characterization (30). There is a restriction on the length of time interval in order to guarantee the uniqueness of the optimality system. This smallness restriction of the length on the time is due to opposite time orientations of (28) and (29); the state problem has initial values and the adjoint problem has a final values. This restriction is very common in control problem [16].

5. Numerical Simulation and Discussion

In this section, we study numerically the effects of transmission parameters of the model (1) and the effects of the time dependent control on the transmission of the disease. The numerical simulations of the model are carried out using the following set of estimated parameter values:

$$a = 0.004, \; r = 0.5, \; \delta = 0.05, \; \mu_1 = 0.4,$$
$$\mu_2 = 0.002, \; \beta = 0.014, \; b = 0.05,$$
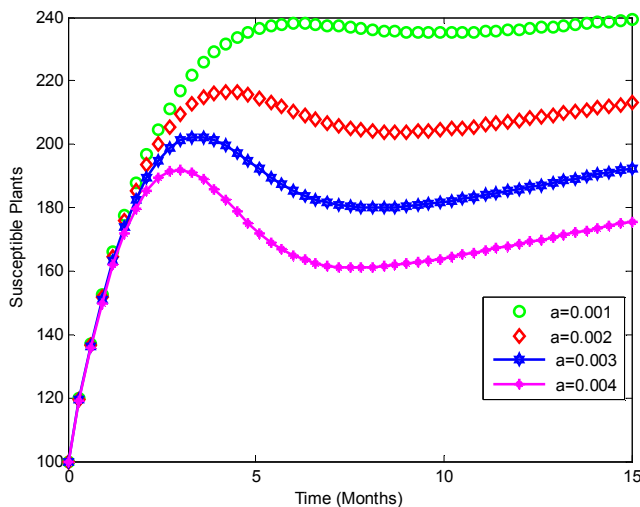$$m = 0.002, \; Q = 110, \; K = 105$$

Figure 3. *Variation of susceptible plant population for different values of α.*

Figure 3 shows that the number of susceptible plants decreases as the rate of transmission of the disease increases since the susceptible plants are not controlled.

Figure 4 shows that the number of infected plant increase as the value of rate of transmission from infected insect to susceptible plants is increases with time.

It is observed that as the rate of transmission from infected insect to susceptible plants increases, the population of susceptible insect decreases with time. The number of insects decrease due to disease and moved to the infected insect class.
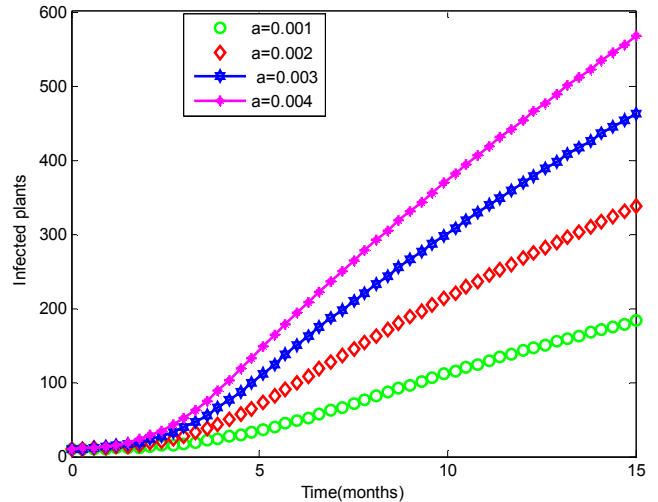
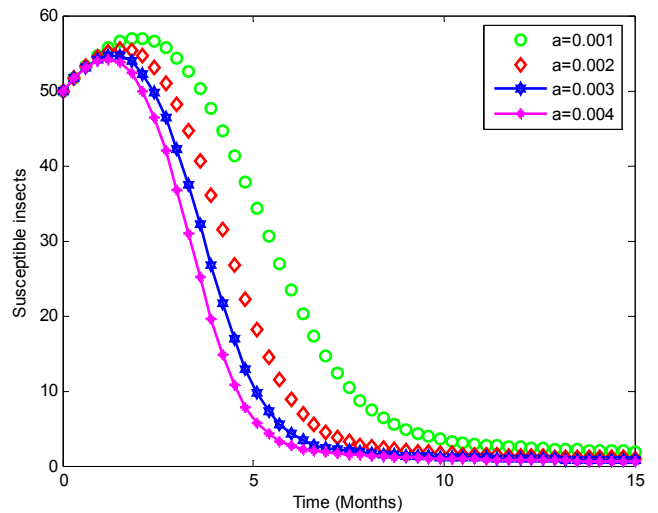***Figure 4.*** *Variation in infected plant population for different values of α.*

Figure 5. *Variation in Susceptible insects' population for different values of α.*
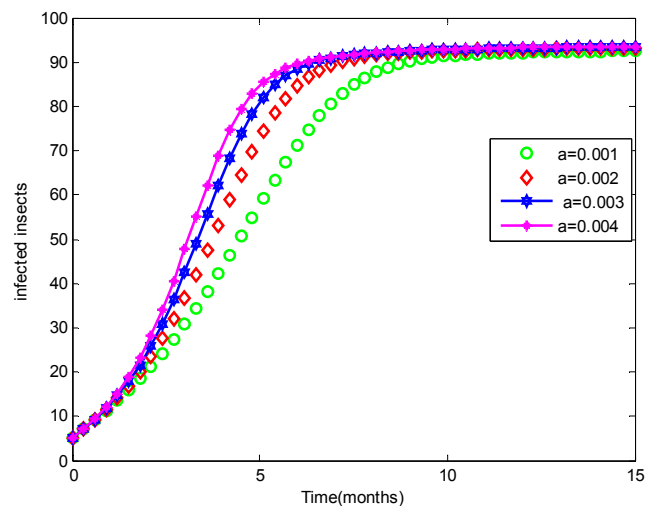
Figure 6. *Variation in infected insects' population for different values of α.*

Figure 6 shows that infected insects are increasing due to the increase of the rate of transmission.

6. Simulation for the Optimal Control Problem

This research intends to determine the impact of optimal control on the transmission of plant disease, so we want to control the rate of transmission from infected insects to the susceptible plants. The control is performed by inoculate the plants to increasing the resistance to disease. This control is applied in 15 months which implies that the final time (t=15), where the initial value of the plants and insects population are given as $(P,F,S.I)=(100,10,50,5)$ the same as without the control graphs.
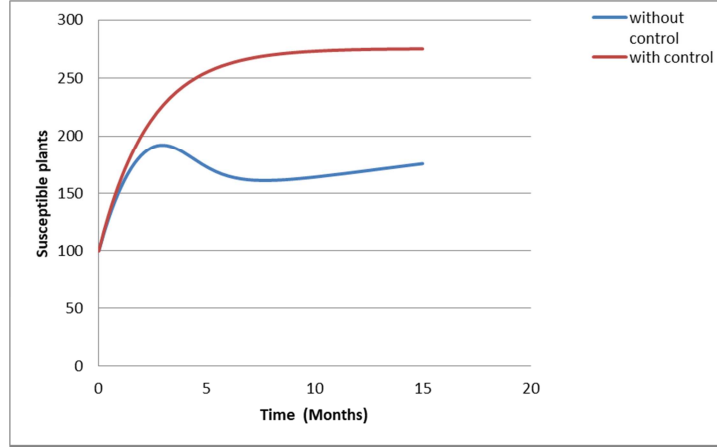

Figure 7. Variation in susceptible plants with control and without control with respect to time.

It is observed in Figure 7 that the number of susceptible plants increases due to control strategy susceptible plants while the number of susceptible plants decreases when there is no control. The increase of number of susceptible plants population is also due to the increase of the number of recovered plants from the infected plant class.
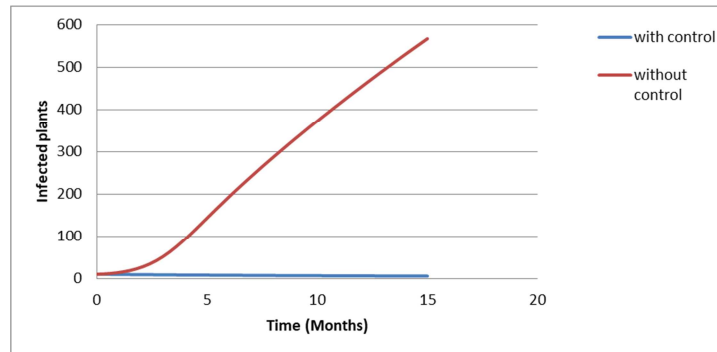

Figure 8. Variation in infected plants with control and without control with respect to time.

Figure 8 shows a significant difference in the number of infected plant with optimal strategy compared to case without controls. Specifically, it is observed that the control strategy lead to a decrease in the number of infected plants as against increases in the uncontrolled case

It is observed in Figure 9 that due to the control strategy, the number susceptible insects increase while the population of susceptible insects decreases when there is no control.
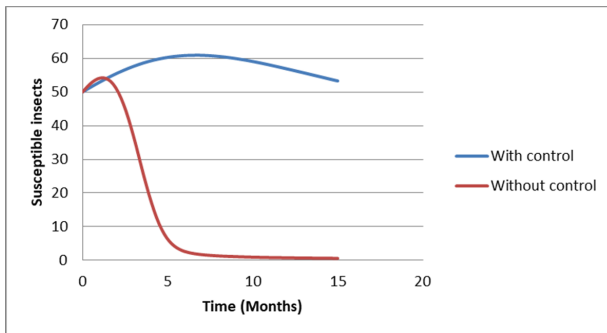

Figure 9. Variation in susceptible insects with respect to time when plants are controlled and uncontrolled.
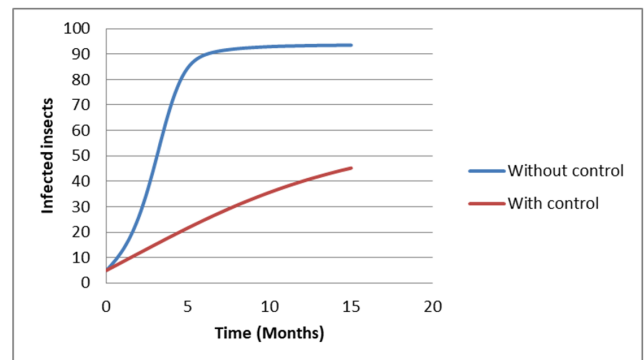

Figure 10. Variation in infected insects with respect to time when plants are controlled and uncontrolled.

In Figure 10, the results show a significant difference in the number of infected insects with optimal strategy compared to case without controls. It is observed that the control strategy lead to a decrease in the number of infected insects as against increases in the uncontrolled case
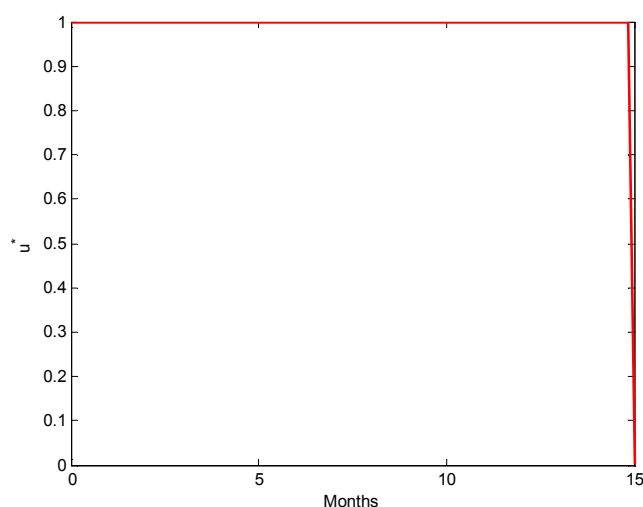

Figure 11. *Variation of control profile with control and without control with respect to time.*

Figure 11, using optimal control requires to maintain the control at 100% for 14.9 months before dropping to its lower bound.

7. Conclusion

In this paper, the optimal control analysis for plant disease model was performed using Pontryagin's maximum principle. Conditions for optimal control of the disease were derived and analysed with an effective use of plant inoculation to reduce plant diseases. The results suggest that the effective use of plant inoculation has a significant impact in reducing the plant disease. It is also observed that due to control strategy, the number of plants increases the number of infective plants decreases. It is concluded that plant inoculation is the best way to minimize transmission of plant disease is by inoculation which is used to increase resistance in preservation of plants.

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