Optimal control for the use of botanical fungicides in the spread of plant diseases

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Abstract. One of the main obstacles in crop cultivation is the occurrence of fungal infections. Fungal spores can spread due to direct contact between susceptible hosts and infected hosts, which can be controlled using botanical fungicides. We constructed a mathematical model of the growth of logistics on the spread of plant diseases. From this model, we show the value of the Basic Reproduction Number ($\mathcal{R}_0$) of plant diseases transmission. The value of $\mathcal{R}_0$ is obtained by finding the largest eigenvalue of the next generation matrix. The results obtained show that when $\mathcal{R}_0$ is greater than one ($\mathcal{R}_0 > 1$), the endemic equilibrium point will be stable. However, if $\mathcal{R}_0$ is less than one ($\mathcal{R}_0 < 1$), the endemic equilibrium point is unstable. In addition, the results of this discussion indicate that using botanical fungicides can reduce the infected host population. We provide numerical simulations to describe the results of the analysis obtained.

1. Introduction

Plant cultivation in the tropics can make easier for farmers to determine when the planting season is good, with the hope of satisfying production. Unfortunately, current seasonal changes are not regular, resulting in very low productivity. Low crop productivity is also thought to be caused by various factors, including poor seed quality, decreased soil fertility, the application of unfavourable planting, and problems with plant pests and diseases [1].

Disease problems in plants can be caused by pathogens, such as fungi, oomycetes, bacteria, viruses, viroid, and pathogenic nematodes [2]. Plants that are infected with fungi, can last long time, and include diseases that are transmitted through the soil. In addition, fungi can survive in the roots of decaying dead plants, and spores can spread due to direct contact between vulnerable hosts and infected hosts [3]. Many prevention and control methods have been used by several researchers, such as chemical resistance and control of plants [4]. Usually farmers control plant diseases caused by fungi using fungicides [5]. However, the use of excessive fungicides in addition to requiring large costs can also inhibit plant growth. To see the effectiveness of the use of fungicides, of course other knowledge is needed to get a more accurate analysis, one of which uses mathematical modelling.

A classic model for micro parasitic interactions host in mammals has been developed by Kermack and McKendrick [6]. Meanwhile, Van der Plank carried out the development of epidemic plant diseases [7]. In addition, the use of mathematical models for plant epidemics has been developed by many researchers to determine the dynamics of spread of plant diseases including the effect of giving control and prevention strategies on dynamics of the spread of plant diseases.

Theory and analysis of plant pathology can be found in Zang et al. [8], whereas some material to understand plant disease epidemics can be studied in Laurence V Madden et al. [9]. Further the theory
of vector-transmitted plant diseases has been discussed [10-16]. While, Ruiqing [17] combines prey-predator mathematical models with vector-host theory. Then, vector epidemic host models with non-linear and linear events have been created and analyzed [18]. Wang et al. [19] discusses the global dynamics of vector-borne disease models. Furthermore, epidemiological models of plant diseases involving protectant [20] and curative fungicides [21]. Anggriani et al [16] discuss the effects of insecticides on the Tungro rice vector. Chan M S and Jeger M have developed the dynamics of mathematical model in plant diseases with and without rougging mechanism [22], it shows that the application of the rougging mechanism can prevent transmission of plant diseases. Then the simplification of the SIRX model has been done by reducing a number of free parameters to help for interpret biologically [23]. Meanwhile, Anggriani et al. [24] discussed optimal control of mathematical model of plant diseases by applying fungicides, to reduce infected plants.

In this paper we develop an optimal control model that has been carried out by Anggriani et al. [24], with the growth function that we use is the logistics function. The use of logistic growth functions is considered to be the most illustrative model of the growth of organism dynamics in limited habitats compared to monomolecular, because this function shows that growth rates in populations introduced to the first environment are proportional to population size at time t and material resources left in habitat still available for exploitation [25].

2. Dynamical Model
The model is constructed from the same assumptions as [24], but in this model the growth of plants follows the logistic function, where \( f(S) = S(1 - S) \). From the schematic diagram in Figure 1 [24], it is obtained:

\[
\begin{align*}
\frac{dS}{dt} &= S(1 - S) - (\beta_1 X + \beta_2 I)S - nS + \epsilon P = 0 \\
\frac{dR}{dt} &= (\beta_1 X + \beta_2 I)S - \mu l = 0 \\
\frac{dI}{dt} &= l - hR = 0 \\
\frac{dX}{dt} &= l - cX = 0 \\
\frac{dP}{dt} &= nS - \epsilon P - hp = 0
\end{align*}
\]  

(2.1)

3. Mathematical Analysis

3.1. Equilibrium point
System (2.1) has two equilibrium points, the first equilibrium point is a Disease-Free Equilibrium Point (DFEP) where compartments \( I, R, \) and \( X \) are zero, given by:

\[
E_{L0} = (S_0, I_0, R_0, X_0, P_0) = \left( \frac{e + h - nh}{e + h}, 0, 0, 0, \frac{\pi(e + h - nh)}{(e + h)^2} \right).
\]

(3.1)

The second equilibrium point is the Endemic Equilibrium Point (EEP), given by:

\[
E_{L1} = (S_0, I_0, R_0, X_0, P_0) = \left( \frac{\mu c}{\beta_p + \beta_c (e + h)}, \frac{c(e + h - nh)(\beta_1 + \beta_2) - c\mu(e + h)}{(\beta_1 + \beta_2)(e + h)}, \frac{c(e + h)(\beta_1 + \beta_2) - c\mu(e + h)}{(\beta_1 + \beta_2)(e + h)}, \frac{c(e + h)(\beta_1 + \beta_2) - c\mu(e + h)}{(\beta_1 + \beta_2)(e + h)}, \frac{\mu c n}{(e + h)(\beta_1 + \beta_2)} \right)
\]

(3.2)
3.2. Basic Reproduction Number (BRN)

We use the next generation method [26-28] in determining the BRN ($\mathcal{R}_0$) for the system (1), where $F$ is the rate of new infection and $V$ is the rate of transfer in and out of the compartment of the infected plant, this is given by:

$$ F = \left[ \beta_s \left( \frac{e^{h-nh}}{e+h} \right) \beta_p \left( \frac{e^{h-nh}}{e+h} \right) \right] \text{and } V = \begin{bmatrix} \mu & 0 \\ 1 & \epsilon \end{bmatrix}. $$

$\mathcal{R}_0$ is obtained from spectral radius $\zeta$ (dominant eigen value) of the $FV^{-1}$ matrix denoted by $\zeta$, so that it is obtained:

$$ \mathcal{R}_0 = \zeta(FV^{-1}) = (\beta_sc + \beta_p) \left( \frac{e^{h-nh}}{c\mu(e+h)} \right) \quad (3.3) $$

3.3. Local Stability Analysis

**Theorem 1** The DFEP of the system (2.1) is locally asymptotically stable if $\mathcal{R}_0 < 1$.

**Proof:**

Based on [27] and from (3.1), The Jacobian matrix for DFEP is:

$$ JE_{L0} = \begin{bmatrix} 1 - \frac{2(e + (1 - \pi)h)}{h + e} - \pi & -\frac{\beta_s(e + (1 - \pi)h)}{h + e} & 0 & -\frac{\beta_p(e + (1 - \pi)h)}{h + e} & \epsilon \\ 0 & \frac{\beta_s(e + (1 - \pi)h)}{h + e} & -\mu & 0 & 0 \\ 0 & 1 & -h & 0 & 0 \\ 0 & 1 & 0 & -c & 0 \\ \pi & 0 & 0 & 0 & -(e + h) \end{bmatrix} (3.4) $$

The characteristic equation of $JE_{L0}$ is $P(\lambda) = \frac{1}{(h+e)}(\lambda + h)P_1(\lambda)P_2(\lambda)$. The characteristics equation $P_1(\lambda)$ and $P_2(\lambda)$ have negative roots if the coefficient of $\lambda^2$, $\lambda^3$, $\lambda^0$ is positive. One coefficient of $\lambda^2$ namely $a_0$ at $P_1(\lambda)$ is clearly positive, while $a_1 > 0$ if $\mathcal{R}_0 < 1$ and $a_2 > 0$ if $\mathcal{R}_0 < 1$. Whereas for $P_2(\lambda)$, $a_0 > 0$, $a_1 > 0$ with the terms $\epsilon > h$ and $a_2 > 0$ with the terms $e + h > \pi h$. This is completed this proof.

**Theorem 2** The EEP of the system (2.1) is locally asymptotically stable if $\mathcal{R}_0 > 1$.

**Proof:**

The points remain endemic if written in $\mathcal{R}_0$ are:

$$ S = \frac{\mu c}{\beta_s + \beta_s} ; I = \frac{c^2\mu(\mathcal{R}_0 - 1)}{(\beta_p + \beta_c)^2} ; R = \frac{c^2\mu(\mathcal{R}_0 - 1)}{(\beta_p + \beta_c)^2} ; X = \frac{c\mu(\mathcal{R}_0 - 1)}{(\beta_p + \beta_c)^2} ; \text{and } P = \frac{\mu c}{(e + h)(\beta_p + \beta_c)}. $$

This point can be simplified as follows

$$ p = \frac{mc}{\beta_p + \beta_c} , q = \frac{c^2\mu}{h(\beta_p + \beta_c)^2} , s = \frac{c^2\mu}{h(\beta_p + \beta_c)^2} , r = \frac{c\mu}{(\beta_p + \beta_c)^2} , l = \frac{\mu c}{(e + h)(\beta_p + \beta_c)}. $$

The characteristic equation obtained from the Jacobian matrix for endemic equilibrium points is:

$$ P(\lambda) = a_0\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 \quad (3.5) $$

The eigen values of $P(\lambda)$ are negative, if $a_0, a_1, a_2, a_3, a_4 > 0$ and $b_1, c_1, d_1, e_1 > 0$, so $E_{L1}$ is locally asymptotically stable when $\mathcal{R}_0 < 1$, with the provision of $2n_{11}n_7 - n_{12}n_{15} > 0, n_{13}n_5 - n_{11}n_8 > 0, a_2b_1 - a_1b_2 > 0, c_1b_2 - b_1c_2 > 0, 4n_{10}n_7 - n_{12}n_3 - 2n_{12}n_4 > 0, 2n_{13}n_4 - n_9n_8 - 2n_{10}n_8 - n_{11}n_6 - 2n_{11}n_7 - n_{12}n_{15} > 0, n_{13}n_5 - n_{11}n_8 > 0$.

4. Optimal control problem

Optimal control in this model has the same goal as optimal control in the monomolecular model, namely minimizing the number of infected populations by looking at the minimum cost [24]. The objective function is given based on Pontryagin's Maximum Principle as follows [29]:

$$ J = \int_{t_0}^{t_f} [B_0I(t) + B_1u^2(t)] \, dt \quad (4.1) $$

The variable $u(t)$ is the control variable for application botanical fungicide and $B_0$ is the weight of infected population and $B_1$ is the weighting equal to the cost of given botanical fungicide. Using the method analyzed by [24], it is obtained:

$$\frac{\partial H}{\partial u} = 0 \Rightarrow u = \frac{(\lambda_1 - \lambda_5)\pi S}{2B_1}.$$  \hspace{1cm} (4.2)

Since $0 \leq u \leq 1$, then optimal control is obtained: $u = \min \left\{ \max \left( 0, \frac{(\lambda_1 - \lambda_5)\pi S}{2B_1} \right), 1 \right\}$. 

5. Numerical Simulation
We provide numerical examples with control and without control to illustrate the dynamics of the spread of plant diseases in each compartment. We use parameter values and initial conditions for each of compartment as in Table 1 [24], with the initial conditions for each compartment $S(0) = 1, I(0) = 0.8, R(0) = 0, X(0) = 0.85$, and $P(0) = 0$.

![Figure 1. Dynamics of plant populations when $R_0 < 1$.](image)

![Figure 2. Dynamics of plant populations when $R_0 > 1$.](image)

![Figure 3. Dynamics of susceptible plant population with botanical fungicides and without botanical fungicides.](image)

![Figure 4. Dynamics of infected plant population with botanical fungicides and without botanical fungicides.](image)

![Figure 5. Dynamics of removed plant population with botanical fungicides and without botanical fungicides.](image)

![Figure 6. Dynamics of infected plat population with botanical fungicides and without botanical fungicides.](image)
Figure 7. Dynamical of protected plant population with botanical fungicides and without botanical fungicides.

Figure 8. Control of botanical fungicides.

From Figure 1 and Figure 2, it can be seen that the primary and secondary infection rates will decrease on day 5 for $R_0 < 1$, and will oscillate from day 5 to month 3 for $R_0 > 1$. Thus, the level of plants removed will increase when $R_0 > 1$ and the level of protected plants increases when $R_0 < 1$. Whereas for Figure 3 until Figure 7 it can be seen that the infected population decreases when given botanical fungicide control when compared to those without control. While for plants that are susceptible to decline because they move to plant populations that are removed and protected. In Figure 8, it can be seen that the given botanical fungicide from each period has decreased and from the fourth period it appears that plants do not need to be given back fungicides.

6. Conclusion
In this paper, we have discussed the optimal control model for the use of botanical fungicides in the spread of plant diseases, where the growth of these plants is a logistic growth function. From the numerical simulation results show that there is an effect of the application of botanical fungicides to the spread of diseases in plants. It can be seen that the dynamics of infected hosts decreases after being given botanical fungicides when compared to those without botanical fungicides, then the dynamics of the removed plant population and the protection plant population with fungicides will increase after being given botanical fungicides as a control treatment.

These results indicate that there is a difference between the growth function that follows the monomolecular function and logistics, including: population dynamics when $R_0 > 1$ for logistic growth experiences temporary oscillation for monomolecular growth does not experience oscillation. In contrast to growth that follows a monomolecular function, growth following a logistical function for plant populations is susceptible to decline because populations move to plant populations that are removed and protected, so the possibility of plants experiencing infection is lower than monomolecular growth.

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