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To cite this article: N Anggriani et al 2018 IOP Conf. Ser.: Mater. Sci. Eng. 332 012036

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Optimal control issues in plant disease with host demographic factor and botanical fungicides

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Abstract. In this paper, we discuss a mathematical model of plant disease with the effect of fungicide. We assume that the fungicide is given as a preventive treatment to infectious plants. The model is constructed based on the development of the disease in which the monomolecular is monocyclic. We show the value of the Basic Reproduction Number (BRN) $\mathcal{R}_0$ of the plant disease transmission. The BRN is computed from the largest eigenvalue of the next generation matrix of the model. The result shows that in the region where $\mathcal{R}_0$ greater than one there is a single stable endemic equilibrium. However, in the region where $\mathcal{R}_0$ less than one this endemic equilibrium becomes unstable. The dynamics of the model is highly sensitive to changes in contact rate and infectious period. We also discuss the optimal control of the infected plant host by considering a preventive treatment aimed at reducing the infected host plant. The obtaining optimal control shows that it can reduce the number of infected hosts compared to that without control. Some numerical simulations are also given to illustrate our analytical results.

1. Introduction

Epidemiology of plant diseases studies the development of plant disease populations in space and time. This area implies that the epidemiology of plant diseases is a change of disease, not a disease state at a given time and space. The Plant disease control has been shown the process of change in relation to disease cycles and disease populations. Both are interrelated because the cycle of disease can change the population of the disease [1]. The disease cycle in plants generally consists of infection, sporulation and dissemination. Plant diseases caused by fungi in the infection process consist of germination, penetration, and colonization sub-processes; the sporulation process consists of sporophore production sub-processes, spore production, spore maturation; as well as the dissemination process comprising sub-process of spore release, spore transport, and spore deposition. In the first cycle of disease, the infection process begins with the spores that act as the primary inoculum.

The host plant not only passes the infection caused by the primary inoculum alone. Where the consequence of the time required to complete one cycle of disease is whether the inoculum can still or not re-infect by the primary inoculum. If there are a fragile host plant and an appropriate environmental condition, the first cycles of the disease will produce a secondary inoculum that will re-start the disease cycle. Secondary inoculum which has the chance to re-infect is said to be the secondary or secondary infection.
As a primary pathogen, the fungus can infect the host tissue before there is another pathogenic fungal attack and may cause symptoms. As a secondary pathogen it appears when the fungus infects the host plant after another pathogen attack, so the attack rate becomes so severe [2]. The epidemic which affected by host’s demography is very short compared with the pathogen. The dynamics like that are common for many plant diseases where comprise the natural population unit among which a pathogen spreads [8]. Based on the problem, it is required for farmers to control the disease by applying synthesis exterminator that is fungicide. Control of plant diseases with the use of synthetic fungicides continuously result in environmental pollution and poisoning in humans. In various studies, chemicals used have negative effects on humans and animals, such as various dangerous diseases such as cancer and disability [4]. Therefore, better control and preventive strategies are required to prevent plant disease transmission [5].

Mathematical models provide a powerful tool used to understand the dynamics of disease spread through a population and in decision making in regards to disease prediction and disease control [6]. A number of control and preventive strategies have been implemented such as the use of host plants resistant and chemical control [7]. Kermack and McKendrick in [8] developed a classical model for micro parasite host interactions in mammals which forms the basis of plant epidemiological models. The first models of temporal development of epidemic plant diseases were developed by Van der Plank in [9] and have formed the basis for plant disease modeling. Subsequently, various models have been developed. A mathematical model is commonly developed to examine the plant disease dynamics including the effects of control and preventive strategies on plant disease dynamics [6-23]. A number of researches studied the epidemiological models of the plant involving fungicides with curative treatment [13] and protection factor [12]. Furthermore, a discussion of the theory and plant epidemiology can be found in [14].

Mathematical models have been developed to investigate the dynamics of plant disease with and without roguing mechanism [16] and found that applying roguing mechanism can prevent plant disease transmission. Earlier theories on vector- borne diseases in [17, 18, 19, 20] highlight the importance of the direct interactions between the vector’s dynamics and disease prevalence in the host. A model with roguing and replanting has been developed [16] and solved using backward Euler method. Furthermore, mathematical models have been developed to study the effects of plant disease transmission dynamics with roguing, replanting and additional treatment such as curative [21] and preventive treatments of plants [22].

A mathematical model discussed in [10] investigate the combination of non-dimensionalization to simplify an SIRX model with reducing the numbers of free parameters to assist biological interpretation. By using $R_0$ the author in [10] also analyze disease invasion and endemic stability to characterize disease persistence [11]. In this paper we investigate the effectiveness of botanical fungicides by looking at the dynamics between epidemiology and host demographics with the density of protected host based on [11]. A protected host population is defined as a population that have been given botanical fungicides. Host demographics are shown from the monomolecular host growth function.

2. Dynamical Model

To develop the mathematical model, we consider the following assumptions:

1. There is a dependence between the host growth rate with the maximum host population
2. The infected host population is divided to two compartments: the primary infection caused by inoculum primary and the secondary infection caused by inoculum secondary
3. The susceptible host population do not affect the infected primer host population
4. The susceptible plant is given botanical fungicide
5. Primary infection population is a monocyclic process with primary inoculum as a source of infection and is characterized by symptoms that occur first in a season.
6. The population consists of healthy hosts (vulnerable) who have been given preventative botanical fungicides.
Based on the assumption above, the interaction between all sub-population can be depicted as in Figure 1.

**Figure 1. Transmission Diagram**

| Parameter/Variable | Definition                                                                 | Dimension | Value $(R_0 < 1)$ | Value $(R_0 > 1)$ |
|--------------------|-----------------------------------------------------------------------------|-----------|------------------|------------------|
| $g(S)$             | production of susceptible hosts                                             | Analog    | 0.05             | 0.5              |
| $f(S)$             | The dynamic of vulnerable hosts without infection.                          |           |                  |                  |
| $f(S) = g(S) - hS$  | $f(S) = \frac{g(S)}{b}$                                                   |           |                  |                  |
| $S$                | Density of susceptible host                                                 |           |                  |                  |
| $I$                | Density of Infected host                                                    |           |                  |                  |
| $R$                | Density of Removed Host                                                     |           |                  |                  |
| $X$                | Density of primary infection Host                                           |           |                  |                  |
| $P$                | Density of Protection Host                                                  |           |                  |                  |
| $\beta_p$          | Rate of Primary infection                                                   |           | 0.075            | 0.75             |
| $\beta_s$          | Rate of secondary infection                                                 |           |                  |                  |
| $m$                | rate of disease-induced mortality for infected hosts                        |           |                  |                  |
| $h$                | rate of removal (all classes of host)                                       |           | 0.25             | 0.25             |
| $b$                | Birth rate                                                                  |           | 0.5              | 0.5              |
| $k$                | carrying capacity                                                           |           | 0.3              | 0.3              |
| $a$                | rate of production of inoculum by infected hosts                            |           | 0.25             | 0.25             |
| $c$                | rate of decay of inoculum                                                   |           | 0.8              | 0.8              |
| $\pi$              | the effectiveness of botanical fungicides                                   |           | 0.1              | 0.1              |
| $\varepsilon$      | The rate of damage to the botanical fungicide given to the plant host       |           | 0.01             | 0.01             |

Therefore, their dynamics can be described by the following autonomous system:

$$
\frac{dS}{dt} = g(S) + \varepsilon P - (\beta_p X + \beta_s I)S - hS - hS
$$

$$
\frac{dI}{dt} = (\beta_p X + \beta_s I)S - mI - hI
$$
\[
\begin{align*}
\frac{d\hat{R}}{dt} &= \hat{m}I - \hat{h}\hat{R} \\
\frac{d\hat{X}}{dt} &= \hat{a}I - \epsilon\hat{X} \\
\frac{d\hat{P}}{dt} &= \hat{n}\hat{S} - \hat{e}\hat{P} - \hat{h}\hat{P}
\end{align*}
\]

where \( \hat{S} + \hat{I} + \hat{R} + \hat{X} + \hat{P} = \hat{\kappa} \).

The description of all variables and parameters are given in Table 1 with the values of all parameters are non-negative.

2.1. Non-dimensionalization

To proceed further, we consider the following dimensionless variables:

\[
S = \frac{\hat{s}}{\hat{\kappa}}, \quad I = \frac{\hat{I}}{\hat{\kappa}}, \quad X = \frac{\hat{X}}{\hat{\kappa}}, \quad R = \frac{\hat{R}}{\hat{\mu}R}, \quad P = \frac{\hat{P}}{\hat{\kappa}} \quad \text{and} \quad t = \hat{b}t
\]

with the normalized parameter:

\[
\beta_p = \frac{\beta_p\hat{m}}{b^2}, \quad \beta_s = \frac{\beta_s\hat{m}}{b}, \quad \mu = \frac{\mu}{b}, \quad c = \frac{c}{b}, \quad h = \frac{h}{b}, \quad \epsilon = \frac{\epsilon}{b}, \quad \pi = \frac{\pi}{b} \quad \text{where} \quad \hat{m} = \hat{m} + \hat{h}
\]

to obtain a reduced and normalized model of (1) in the form:

\[
\begin{align*}
\frac{ds}{dt} &= f(S) - (\beta_p X + \beta_s I)S - \pi S + \epsilon P \\
\frac{dI}{dt} &= (\beta_p X + \beta_s I)S - \mu I \\
\frac{dR}{dt} &= I - hR \\
\frac{dX}{dt} &= I - cX \\
\frac{dP}{dt} &= \pi S - \epsilon P - hP
\end{align*}
\]

Note that here we have:

\[
\hat{S} + \hat{I} + \hat{R} + \hat{X} + \hat{P} = \frac{\hat{s}}{\hat{\kappa}} + \frac{\hat{I}}{\hat{\kappa}} + \frac{\hat{X}}{\hat{\kappa}} + \frac{\hat{R}}{\hat{\kappa}} + \frac{\hat{P}}{\hat{\kappa}} = \frac{\hat{\kappa}}{\hat{\kappa}} = 1.
\]

2.2. Monomolecular Model

The monomolecular model is the SIRXP model with the monomolecular function given by:

\[
f(S) = b(\hat{\kappa} - q(S))
\]

which after normalization becomes

\[
f(S) = 1 - S
\]

Substitute (4) into (2) to obtain:

\[
\begin{align*}
\frac{dS}{dt} &= 1 - S - (\beta_p X + \beta_s I)S - \pi S + \epsilon P \\
\frac{dI}{dt} &= (\beta_p X + \beta_s I)S - \mu I \\
\frac{dR}{dt} &= I - hR \\
\frac{dX}{dt} &= I - cX
\end{align*}
\]
3. Mathematical Analysis

3.1. Equilibrium Point
There are two different equilibrium points for model (1). First is the disease free equilibrium point
where all infected compartment are equal to zero, given by

\[ E_{M0} = (S_0, I_0, R_0, X_0, P_0) = \left( \frac{\varepsilon + h}{\varepsilon + h + \pi h}, 0, 0, 0, \frac{\pi}{\varepsilon + h + \pi h} \right) \]  \hspace{1cm} (6)

Second is the non-trivial endemic equilibrium point which is given by

\[ E_{M1} = (S_1, I_1, R_1, X_1, P_1) = \left( \frac{\mu c}{\beta_p + \beta_p' c'}, \frac{\beta_p' + \beta_p c}{\beta_p' + \beta_p c + \mu c - \mu c - \mu c h}, \frac{\beta_p h + \beta_p' h + \beta_p c + \mu c - \mu c h}{\beta_p h + \beta_p' h + \beta_p c + \mu c - \mu c h}, \frac{\mu c}{\beta_p h + \beta_p' h + \beta_p c + \mu c - \mu c h}, \frac{\mu c}{\beta_p h + \beta_p' h + \beta_p c + \mu c - \mu c h} \right) \]  \hspace{1cm} (7)

3.2. Basic Reproduction Ratio
The Basic Reproduction Ratio represents the expected numbers of secondary cases produced by a
typical infected individual during its entire period of infectiousness in completely susceptible
population (see the detail in [23,24]). The Basic Reproduction Ratio, denoted by \( R_0 \), is taken from
spectral radius of the next generation matrix \([24,25,26]\) of system (5),

\[ G = \left[ \frac{\beta_p h \left( \frac{\varepsilon + h}{\varepsilon + h + \pi h} \right)} {\beta_p' \left( \frac{\varepsilon + h}{\varepsilon + h + \pi h} \right) + \beta_p \left( \frac{\varepsilon + h}{\varepsilon + h + \pi h} \right) + \frac{\mu c}{\beta_p + \beta_p' c'}} \right] \]

Taking the spectral radius of matrix \( G \) will bring us to the form of the Basic Reproduction Ratio

\[ R_0 = \left( \frac{\beta_p c + \beta_p'} {\frac{\varepsilon + h}{\varepsilon + h + \pi h}} \right) \left( \frac{\varepsilon + h}{\varepsilon + h + \pi h} \right) \]

3.3. Local Stability Analysis

Theorem 1
The disease-free equilibrium point of the system (1)-(5) is locally asymptotically stable if \( R_0 < 1.0 \).

Proof:
By following [23] and from (6), the Jacobian matrix for non-endemic equilibrium point is

\[ J_T = \begin{bmatrix}
-1 - \pi & -\beta_p (\varepsilon + h) & 0 & -\mu & \varepsilon \\
0 & 0 & -\mu & 0 & 0 \\
\beta_p S - (\varepsilon + h) - \mu & 0 & 0 & 0 & 0 \\
\pi & 0 & 0 & -c & -\varepsilon - h \\
0 & 0 & 0 & 0 & 0 \\
\end{bmatrix} \]

The polynomial characteristic of \( J_T \) is \( P(\lambda) = (\lambda + h)P_1(\lambda)P_2(\lambda) \) with:
The Jacobian matrix of the endemic equilibrium (7) gives the characteristic polynomial:

\[ P_2(\lambda) = \lambda^2 + (\epsilon + h + \pi h)\lambda + \left( (\epsilon + h + \pi h)\beta_0 + (\mu + \nu + \pi\nu)\lambda - \beta_0 \epsilon \lambda - \beta_0 \nu \lambda - \beta_0 \nu h + \beta_0 \delta h \right) \]

The eigenvalues of \( P_2(\lambda) \) are \( -\alpha \) and the roots of the polynomial \( p_1(\lambda) \) and \( p_2(\lambda) \). The coefficient \( q_1 > 0 \) if \( R_0 < 1 \), hence all the roots of polynomial \( P_T \) have negative real part when \( R_0 < 1 \). This means that the non-endemic equilibrium point \( E_M = (S_0, I_0, R_0, X, P_0) \) is locally asymptotically stable when \( R_0 < 1 \) [23, 27]. This proves the theorem.

**Theorem 2**

The endemic equilibrium point of the system (1)-(5) is locally asymptotically stable if \( R_0 > 1 \).

**Proof:**

The Jacobian matrix of the endemic equilibrium (7) gives the characteristic polynomial:

\[ Pe(\lambda) = (\lambda + h)P_3(\lambda) \]

The eigenvalues of \( P_3(\lambda) \) are \( -\alpha \) and the roots of the polynomial \( P_3(\lambda) \). From Routh-Hurwitz criterion we have \( a_0, a_1, a_2, a_3, a_4 > 0 \), \( a_2a_3 - a_1a_4 > 0 \), and \( a_2a_3 - a_1^2a_4 - a_0a_3^2 > 0 \), hence all the roots of polynomial \( P_T \) have negative real part when \( R_0 > 1 \) [20]. This means that the non-endemic equilibrium point \( E_{M_1} = (S_1, I_1, R_1, X_1, P_1) \) is locally asymptotically stable when \( R_0 > 1 \) [23, 27]. This proves the theorem.

4. **Optimal Control Problem**

To obtain a decrease in the number of infected populations, the dynamic model of the spread of plant diseases is given a preventive treatment of botanical fungicides. Therefore, optimal control is required on the treatment given to the cost in order to keep the infected plant population at a minimum. In completion of this optimal control, the Pontryagin Maximum Principle is used with the following objective functions [28]:

\[ J = \int_{t_0}^{t_f} [A_0 J(t) + A_1 u^2(t)] dt \]

The variable \( u(t) \) is the control variable of the preventive treatment given (i.e. botanical fungicide) and \( A_0 \) is the weight of infected population balance and \( A_1 \) is the weight of the preventive cost equilibrium cost. The state variable for the model \( x(t) = \begin{bmatrix} S(t) \\ I(t) \\ R(t) \\ X(t) \\ P(t) \end{bmatrix} \) and the constraint:

\[ \frac{dS}{dt} = 1 - S - (\beta_p X + \beta_z I)S - \nu S + \epsilon P \]
\[ \frac{dI}{dt} = (\beta_p X + \beta_z I)S - \mu I \]
\[ \frac{dR}{dt} = I - hR \]
\[ \frac{dX}{dt} = I - cX \]
\[
\frac{dP}{dt} = \pi nS - \varepsilon P - hP
\]

The system should satisfy the condition:
\[
0 \leq u(t) \leq 1, 0 \leq t \leq t_f, S(0) = S_0 > 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0, X(0) = X_0 \geq 0, P(0) = P_0 \geq 0
\]

We define Hamiltonian function as:
\[
H = A_0 l(t) + A_1 u^2(t) + \lambda_1 (1 - S - (\beta_p X + \beta_s I)S - u\pi S + \varepsilon P) + \lambda_2 \left((\beta_p X + \beta_s I)S - \mu l\right)
\]
where \(\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t), \) and \(\lambda_5(t)\) are the co-state variable or the Lagrange multiplier of the optimization problem. The necessary conditions that an optimal control is achieved, it must satisfy the following Pontryagin’s Maximum Principle:

- **State equation**
  \[
  \dot{S} = \frac{\partial H}{\partial \lambda_1} = 1 - S - (\beta_p X + \beta_s I)S - u\pi S + \varepsilon P
  \]
  \[
  \dot{I} = \frac{\partial H}{\partial \lambda_2} = (\beta_p X + \beta_s I)S - \mu l
  \]
  \[
  \dot{R} = \frac{\partial H}{\partial \lambda_3} = l - hR
  \]
  \[
  \dot{X} = \frac{\partial H}{\partial \lambda_4} = l - cX
  \]
  \[
  \dot{P} = \frac{\partial H}{\partial \lambda_5} = u\pi S - \varepsilon P - hP
  \]

- **Co-state equation**
  \[
  \dot{\lambda}_1 = -\frac{\partial H}{\partial S} = \lambda_1 (1 + \beta_p X + \beta_s I + u\pi) - \lambda_2 (\beta_p X + \beta_s I) - u\pi \lambda_5
  \]
  \[
  \dot{\lambda}_2 = -\frac{\partial H}{\partial I} = -A_0 + \beta_i S \lambda_1 + \mu \lambda_2 - \lambda_3 - \lambda_4
  \]
  \[
  \dot{\lambda}_3 = -\frac{\partial H}{\partial R} = h \lambda_3
  \]
  \[
  \dot{\lambda}_4 = -\frac{\partial H}{\partial X} = \beta_p S (\lambda_1 - \lambda_2) + c \lambda_4
  \]
  \[
  \dot{\lambda}_5 = -\frac{\partial H}{\partial P} = h \lambda_5 + \varepsilon \lambda_5 - \varepsilon \lambda_1
  \]

- **Stationer condition**
  \[
  \frac{\partial H}{\partial u} = 0 \Rightarrow u = \frac{(\lambda_1 - \lambda_5)\pi S}{2A_1}
  \]

Since \(0 \leq u \leq 1\), then we get:
\[
\min \left\{ \max \left(0, \frac{(\lambda_1 - \lambda_5)\pi S}{2A_1}\right), 1 \right\}
\]
as the optimal control of the system.

5. **Numerical Result**

In order to illustrate the dynamics of each compartment, we give numerical example with control and without control. We use the values of the parameters shown in Table 1 and the initial condition for each compartment is respectively, \(S(0) = 1, I(0) = 0.8, R(0) = 0, X_0(0) = 0.85, P_0(0) = 0\).

From figure 2 and figure 3, the primary and the secondary infected susceptible rates increase until day 25 for \(R_0 > 1\), so the host infected population or the host removed plant will be increase at the same time. When \(R_0 < 1\) the host of primary infected plant and the secondary infected plants decreases or does not occur outbreak. The host protected plant by the fungicide will be decreases when \(R_0 > 1\) and increases when \(R_0 < 1\).
Figure 2. The dynamical of population plant when $R_0 > 1$

Figure 3. The dynamical of population plant when $R_0 < 1$.

Figure 4. Dynamic of Susceptible Plant with control and without control.

Figure 5. Dynamic of Infected Plant with control and without control.

Figure 6. Dynamic of Removed Plant with control and without control.

Figure 7. Dynamic of Primary Infected Plant with control and without control.
Figure 8. Dynamic of Protected Plant with control and without control.

Figure 4 show that with control, the dynamics of susceptible host plant will increase at 2.5 months then decrease until harvesting or retraction. In figure 7 show that the dynamics of primary infected host plant will decrease until at a half of month and then will be increasing this is caused the infected secondary plant increasing. The dynamics of removed host plant and the protective host plant with the fungicide will be increasing after control treatment. Figure 4 until figure 8 show that the host plant will decrease after control treatment.

6. Conclusion
In this paper, we develop a mathematical model of plant disease with the effect of fungicide. It has been shown numerically, that the optimal control strategy exists. The numerical example shows that the dynamics of primary infected host plant will decrease until at a half of month and then it will be increasing. This eventually causes the infected secondary plant increasing. The dynamics of removed host plant and the protective host plant with the fungicide will be increasing after control treatment.

Acknowledgments
The primary work is carried out with funding from PUPT RISTEKDIKTI 2017, with contract number 718/UN6.3.1/PL/2017.

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