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MATHEMATICAL MODEL FOR SOYBEAN MOSAIC DISEASE TRANSMISSION WITH *ENTOMOPATHOGEN* INTERVENTIONS AND PHOTOPERIODICITY

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Abstract: Mosaic is a severe disease of soybeans that has the potential to reduce the quality and quantity of soybean production. The Mosaic disease can infect soybean plants by the Aphis vector carrying Soybean Mosaic Virus (SMV). In this study, a mathematical model of the spread of Mosaic disease was built by considering two interventions, namely the application of *entomopathogen* and the regulation of photosynthesis intensity. This research focuses on knowing the effect of intervention in controlling Mosaic disease and increasing the population of susceptible generative plants. Using dynamical system theory, non-endemic and endemic equilibrium points and their stability are obtained. Then, the basic reproduction ratio $(\mathfrak{R}_0)$ is obtained for this model. Sensitivity analysis was carried out to determine the most influential parameters in the model. Optimal control theory was used to determine the optimal conditions of the model by considering the cost of *entomopathogen* application and photoperiodicity. The results of numerical simulations show that the application of *entomopathogen* and photosynthetic intensity can suppress the population of plant and vector infections and increase the population of susceptible plants in the generative phase at the same time.

Keywords: dynamical system; mosaic virus; optimal control; sensitivity analysis; soybean.

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1. INTRODUCTION

Soybean is an important food containing protein and oil and is commonly used in the program of diets for both humans and animals [1]. Soybean is often called a marvel plant because it has a high protein of about 39-44% and an oil content of 21% [2]. It is the best source of protein and oil that may be an alternative to meat. Soybean content is used in the manufacturing industry, such as oil, cakes, flour, herbal cheese, and some additional products in the food industry [3]. These facts show that soybean is a crop needed for daily consumption and industrial needs. However, the high demand for soybean is not followed by the ability of the soybean farms to produce the grains. The main factors include climate change, inappropriate growth time, planting space, weeds, and disease [4].

Soybean Mosaic Disease (SMD) is a significant issue in soybean agriculture. The dis-ease begins when the plants is eaten by Aphid [5], a vector transmission that brings the Soybean Mosaic Virus (SMV). SMV occurs in almost all the soybean agriculture areas of the world and potentially infects other economic crops [5]. SMD causes the yield of agriculture to reduce between 35-50% of what it should be [6]. As SMV is an aphid-transmitted and seed-transmitted virus, it has three ways to infect the plants: 1) mechanical transmission, 2) insect transmission, and 3) grafting transmission [5]. The primary method of SMV transmission is transmission by insects such as various Aphids [7].

The presence of Mosaic transmitting vectors, namely Aphid insects, which are possible to be present in agricultural ecosystems increases the possibility of spreading the virus. In the concept of Plant Pest Control (IPM), the control goal is not to completely eradicate the pest/disease population, but it is dominant to manage the population below the threshold [8]. The use of insecticide against vectors showed a slight decrease in Mosaic cases [5]. The concept of IPM considers this step, but in terms of the use of insecticides, it is necessary to pay more attention because it causes damage to plants. The author in [9] explains that *Lecanicillium Lecanii* (L. Lecanii) has the potential to be a natural bio-insecticide or *entomopathogen* for Aphid.

The mathematical model can be used to study the phenomena such as infectious disease transmission both on human [10,11] and plant population [12,13]. The model for this study was usually formed as a compartmental-based model, which divided the population into subpopulations with a unique description. Many researchers have developed a model to study the long-term behavior of various health problem. Disease such as COVID-19 [11], dengue [14], and

hepatitis [15] were studied with some interventions as an effort to control the disease. The problem of plant disease transmission is studied by several researchers using the same analogy of the transmission of human disease.

A mathematical model for plant disease transmission is included as a vector-borne disease model. The model indirectly represents the process of spreading disease from infected plants to susceptible plants by involving the Aphis as a vector transmission. This relationship is interpreted through one of the mathematical studies, namely differential equations system. By knowing the spreading patterns, the disease transmission can be studied through analysis and simulation with some scenarios to predict disease behavior and the impact of interventions involved in the model.

Many researchers conduct to develop a vector-borne model for describing the plant disease and studying its behavior. Jeger [16] built a mathematical model for plant disease considering the latent period of infection in the plant population. The local stability [17] and global stability [18] of the plant disease model is investigated to learn the disease behavior for the long term. Luo et al. [19] and Al-Basir et al. [20] developed a plant disease model that considered the *roguing* and replanting of plants as an effort to eradicate the disease. However, the plant disease epidemic may be prevented through curative fungicide application [21] and protect the plants with applied some methods, such as *roguing* and insecticide spraying [20]. In order to control the disease by IPM concept, the insecticide or bio-insecticide can reduce the vector population, which interprets as a parameter con-trol in a mathematical model [22]. Suryaningrat et al. [23] built a vector-borne model for Tungro disease with considered the existence of biological agents as predators of vector transmission.

Mathematical studies of optimal control theory also applied to some vector-borne models for plant disease. A parameter interprets numerous interventions in a mathematical model set to be a control parameter in an optimal control problem, see [24–26] for examples. Chowdhury et al. [24] considered a model for pest management in plant dis-ease models through pesticides. Dynamical analysis and optimal control theory are applied to study the behavior of diseases with pesticide effort. In [25], Anggriani et al. developed an optimal control model to study the plant disease model by determining the curative treatment as a parameter control in the optimal control problem. Bokil et al. [26] built a mathematical model to describe the plant disease transmission, especially African Cassava Mosaic Disease (ACMD), and study the disease's behavior through mathematical analysis and optimal control theory. The *roguing* and insecticide effort was set to be the control parameter

in the optimal control problem.

In this article, we construct a mathematical model considering both insecticides to reduce the vector population and photoperiodicity to optimize plant growth. Photoperiodicity is the ratio between day and night lengths that may impact the process of plant growth and development [27]. This research focuses on comparing the advantages of interventions, including 1) *entomopathogen* without photoperiodicity, 2) photoperiodicity without *entomopathogen*, and 3) combining *entomopathogen* and photoperiodicity to the model. We look for the equilibrium points of the model and study their stability. A sensitivity analysis through Latin Hypercube Sampling (LHS) and Partial Rank Correlation Coefficients (PRCC) was carried out to determine parameters that have the dominant influence on the model. Then, we set the insecticide and photoperiodicity as two control parameters in the optimal control problem. We formulate the optimal control model and solve it to determine the optimal condition of both controls and minimize the infected vector population and maximize the susceptible generative population. However, we first satisfy the necessary and sufficient condition of the Pontryagin Maximum Principle [28] before solving the optimal control problem. Numerical simulations were conducted to confirm the analytical result. Finally, we discussed the results comprehensively and presented some insights for future work.

2. MODEL FORMULATION

We have considered two populations in a mathematical model, which are the plant population and vector population. The plant and vector populations were divided into the three subpopulations and two subpopulations, respectively, namely susceptible vegetative plants (S_V), susceptible generative plants (S_G), infected population plants (I_H), susceptible vectors (S_A), and infected vectors (I_A). Photoperiodicity is a factor considered in the plant growth process from the vegetative to the generative phase. We use some assumptions to formulate the model, including 1) the soybean plants studied are the same cultivar, 2) the replanting rate is constant and continuous, 3) the vector recruitment rate is constant, 4) infected plants and infected vectors cannot be recovered, 5) plants are only reduced due to natural death, and 6) the applied *entomopathogen* only affects the vector population. There are some factors considered to be involved represented by parameters in the mathematical model (see Table 1). Finally, we can figure out a schematic diagram to describe the insect transmission process of the Mosaic virus in both plant and vector populations (see Figure 1).

Table 1. Parameters definition

Parameter	Definition	Value
Λ	Replanting rate	4
τ	Plant natural growth rate	0.5
η	Maximum plant growth rate due to photoperiodicity	1
α_1	Virus transmission rate from vector to plant	0.0032
α_2	Virus transmission rate from plant to vector	0.0032
μ_H	Plant natural death rate	0.005
γ	Vector recruitment rate	6
μ_A	Vector natural death rate	0.18
δ	Vector death rate due to applied *entomopathogen*	0.25

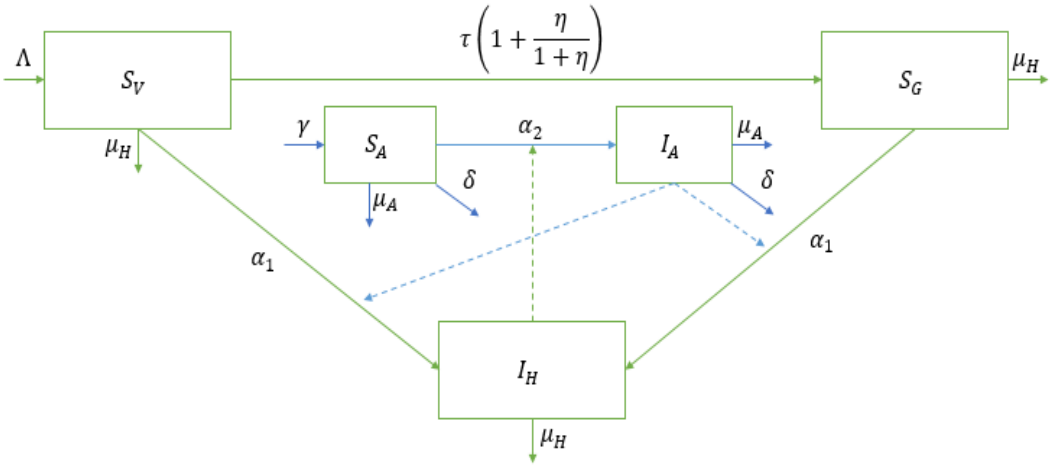


Figure 1. Schematic diagram of the spread of Mosaic disease in Soybean plants. The solid lines represent the transition of plants or vectors from one subpopulation to another, while the dashed line represents the subpopulations involved in the transition.

Based on Figure 1, the differential equations system for the spread of the disease is written as equation (1).

$$\begin{aligned}
\frac{dS_V}{dt} &= \Lambda - \tau\left(1+\frac{\eta}{1+\eta}\right)S_V - \alpha_1 S_V I_A - \mu_H S_V \\
\frac{dS_G}{dt} &= \tau\left(1+\frac{\eta}{1+\eta}\right)S_V - \alpha_1 S_G I_A - \mu_H S_G \\
\frac{dI_H}{dt} &= \alpha_1 I_A (S_V + S_G) - \mu_H I_H
\end{aligned} \tag{1}$$

$$\frac{dS_A}{dt} = \gamma - \alpha_2 S_A I_H - \mu_A S_A - \delta S_A$$
$$\frac{dI_A}{dt} = \alpha_2 S_A I_H - \mu_A I_A - \delta I_A$$

With the initial condition of each compartment as equation (2).

$$S_V(0) \geq 0, \;\; S_G(0) \geq 0, \;\; I_H(0) \geq 0, \;\; S_A(0) \geq 0, \;\; I_A(0) \geq 0 \tag{2}$$

3. MATHEMATICAL ANALYSIS

In this section, we carried out the equilibrium points, both non-endemic and endemic, and analyzed their stability. The basic reproduction ratio $(\Re_0)$ is obtained through the next-generation matrices method.

3.1. Non-Endemic Equilibrium Point

The non-endemic equilibrium point is a state which represents that there is no disease infection in the system with $\Re_0 < 1$. Based on the model in equation (1), the non-endemic equilibrium point is obtained as follows:

$$E^0 = \{S_V^0, S_G^0, I_H^0, S_A^0, I_A^0\}$$

$$E^0 = \left\{\frac{\Lambda(1+\eta)}{(2\tau+\mu_H)\eta+\tau+\mu_H}, \frac{\Lambda\tau(1+2\eta)}{\mu_H\big((2\tau+\mu_H)\eta+\tau+\mu_H\big)}, 0, \frac{\gamma}{\delta+\mu_A}, 0\right\} \tag{3}$$

3.2. Endemic Equilibrium Point

The endemic equilibrium point is a state which represents that there is disease infection in the system with $\Re_0 > 1$. Based on the model in equation (1), the endemic equilibrium point is obtained as follows:

$$E^* = \{S_V^*, S_G^*, I_H^*, S_A^*, I_A^*\}$$

where

$$S_V^* = \frac{\left(\eta+\frac{1}{2}\right)\big((\delta+\mu_A)\mu_H+\alpha_2\Lambda\big)^2(\delta+\mu_A)^2\tau}{\alpha_1\big((\delta+\mu_A)\mu_H+\alpha_1\gamma\big)(x+y)}$$

$$S_G^* = \frac{(1+\eta)\big((\delta+\mu_A)\mu_H+\alpha_2\Lambda\big)(\delta+\mu_A)\Lambda}{2(x+y)} \tag{4}$$

$$I_H^* = \frac{-(\delta+\mu_A)^2\mu_H^2+\alpha_1\alpha_2\gamma\Lambda}{\big((\delta+\mu_A)\mu_H+\alpha_1\gamma\big)\alpha_2\mu_H}$$

$$S_A^* = \frac{\big((\delta+\mu_A)\mu_H + \alpha_1\gamma\big)\mu_H}{\alpha_1\big((\delta+\mu_A)\mu_H + \alpha_2\Lambda\big)}$$

$$I_A^* = \frac{-(\delta+\mu_A)^2\mu_H^2}{(\delta+\mu_A)\big((\delta+\mu_A)\mu_H + \alpha_2\Lambda\big)\alpha_1}$$

with

$$x = \left(\frac{\alpha_2(1+\eta)\Lambda}{2} + \left(\eta+\frac{1}{2}\right)(\delta+\mu_A)\tau\right)(\delta+\mu_A)\mu_H$$

$$y = \alpha_2\left(\left(\eta+\frac{1}{2}\right)\tau\mu_A + \left(\eta+\frac{1}{2}\right)\delta\tau + \frac{\gamma\alpha_1(1+\eta)}{2}\right)\Lambda$$

3.3. Basic Reproduction Ratio

In epidemiology, the basic reproduction ratio is essential to know, which shows the potential emergence of the spread of disease. Biologically, this ratio indicates the number of subsequent infections from one infective host or vector to susceptible hosts or vectors. Mathematic looks at the ratio as a parameter in studying the disease transmission through a compartmental model. The next-generation matrices method in [30] was used to obtain this parameter. We set f as the new infection matrix and v as the matrix of changes in the infection compartment. Based on equation (1), the f and v matrices are obtained as follows:

$$f = \begin{pmatrix} 0 & \alpha_1(S_V+S_G)I_A \\ \alpha_2 S_A I_H & 0 \end{pmatrix} \text{ and } v = \begin{pmatrix} \mu_H I_H & 0 \\ 0 & (\delta+\mu_A)I_A \end{pmatrix}$$

We carried out F and V as the Jacobian from f and v matrices, then the spectral radius (dominant eigenvalue) of the FV^{-1} matrix is determined at the non-endemic equilibrium point (see equation 3). This process can be written in equation (5-8):

$$F = \begin{pmatrix} 0 & \alpha_1(S_V+S_G) \\ \alpha_2 S_A & 0 \end{pmatrix} \tag{5}$$

$$V = \begin{pmatrix} \mu_H & 0 \\ 0 & \delta+\mu_A \end{pmatrix} \rightarrow V^{-1} = \begin{pmatrix} \frac{1}{\mu_H} & 0 \\ 0 & \frac{1}{\delta+\mu_A} \end{pmatrix} \tag{6}$$

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\alpha_1(S_V+S_G)}{\delta+\mu_A} \\ \frac{\alpha_2 S_A}{\mu_H} & 0 \end{pmatrix} \tag{7}$$

$$\Re_0 = \rho(FV^{-1}) = \frac{\sqrt{\alpha_1\alpha_2(\delta+\mu_A)\mu_H S_A^0(S_V^0+S_G^0)}}{(\delta+\mu_A)\mu_H} \tag{8}$$

Finally, the parameter of the basic reproduction ratio is obtained radius spectral of FV^{-1} as follows:

$$\Re_0 = \frac{\sqrt{\alpha_1\alpha_2\gamma\Lambda}}{(\delta+\mu_A)\mu_H} \tag{9}$$

3.4. Stability Analysis

Analysis at the equilibrium points is carried out to determine the behavior of the system in the long term. The investigation began with formulating the Jacobian matrix for the model in equation (1). We write this matrix in equation (10).

$$J = \begin{pmatrix} -\tau\left(1+\frac{\eta}{1+\eta}\right)-\alpha_1 I_A-\mu_H & 0 & 0 & 0 & -\alpha_1 S_V \\ \tau\left(1+\frac{\eta}{1+\eta}\right) & -\alpha_1 I_A-\mu_H & 0 & 0 & -\alpha_1 S_G \\ \alpha_1 I_A & \alpha_1 I_A & -\mu_H & 0 & \alpha_1(S_V+S_G) \\ 0 & 0 & -\alpha_2 S_A & -\alpha_2 I_H-\delta-\mu_A & 0 \\ 0 & 0 & \alpha_2 S_A & \alpha_2 I_H & -\delta-\mu_A \end{pmatrix} \tag{10}$$

3.4.1. Non-endemic Point

Theorem 3.4.1. The non-endemic equilibrium point of the model in equation (1) is locally asymptotically stable if $\Re_0 < 1$.

Proof. Through the method in [31], the local stability of the non-endemic equilibrium point in (3) can be determined by substituting E^0 into the Jacobian matrix in (10). Then we get the characteristic polynomial, as follows:

$$CP_1 = \frac{1}{(\delta+\mu_A)\mu_H(1+\eta)}\Bigg((\delta+\mu_A+\lambda_1)(\mu_H+\lambda_2)\left(\left(\frac{\eta}{2}+\frac{1}{2}\right)\mu_H+\left(\frac{\eta}{2}+\frac{1}{2}\right)\lambda_3+\tau\left(\eta+\frac{1}{2}\right)\right)(a_1\lambda^2 + a_2\lambda + a_3)\Bigg) \tag{11}$$

From the equation (11), we get the eigenvalues of the evaluated Jacobian matrix at the non-endemic equilibrium point including $\{\lambda_1,\lambda_2,\lambda_3\} = \{-(\delta+\mu_A), -\mu_H, -\big((1+\eta)\mu_H + (1+2\eta)\tau\big)/(1+\eta)\}$ and determine the character of polynomial $a_1\lambda^2 + a_2\lambda + a_3$ through Routh-Hurwitz criterion. All coefficients $a_1, a_2,$ and a_3 are known and can be written as:

$a_1 = (\delta+\mu_A)\mu_H > 0$

$a_2 = (\delta^2 + 2\delta\mu_A + \mu_H\delta + \mu_A^2 + \mu_H\mu_A)\mu_H > 0$

$$a_3 = (\delta + \mu_A)^2\mu_H^2 - \alpha_1\alpha_2\gamma\Lambda = (\delta + \mu_A)^2\mu_H^2 - (\delta + \mu_A)^2\mu_H^2\Re_0^2 = (\delta + \mu_A)^2\mu_H^2(1 - \Re_0^2) > 0$$

$$a_3 = (1 - \Re_0^2) > 0 \rightarrow 1 > \Re_0^2 \rightarrow \Re_0 < 1.$$

Since λ_i with $i = 1,2,3$ are negative and $a_1, a_2, a_3 > 0$ indicates that λ_4 and λ_5 have negative values – also $\Re_0 < 1$. This completes the proof.

3.4.2. Endemic Point

Theorem 3.4.2. The endemic equilibrium point of the model in equation (1) is locally asymptotically stable if $\Re_0 > 1$.

Proof. Through the method in [31], the local stability of the endemic equilibrium point in (4) can be determined by substituting E^* into the Jacobian matrix in (10). Then we get the characteristic polynomial, as follows:

$$CP_2 = \frac{1}{xy(\delta + \mu_A)\mu_H}(\lambda + \mu_H)(\lambda + \delta + \mu_A)\Big((1 + \eta)\big((\delta + \mu_A)\mu_H + \alpha_2\Lambda\big)(\delta + \mu_A)\lambda + 2(x + y)\Big)(a_4\lambda^2 + a_5\lambda + a_6) \quad (12)$$

From the equation (12), we get the eigenvalues of the evaluated Jacobian matrix at the endemic equilibrium point including $\{\lambda_6, \lambda_7, \lambda_8\} = \Big\{-(\delta + \mu_A), -\mu_H, -2(x + y)/(1 + \eta)\Big(\big(\delta + \mu_A\big)\mu_H + \alpha_2\Lambda\Big)\big(\delta + \mu_A\big)\Big\}$ and determine the character of polynomial $a_4\lambda^2 + a_5\lambda + a_6$ through Routh-Hurwitz criterion. All coefficients $a_4, a_5,$ and a_6 are known and can be written as:

$$a_4 = \big((\delta + \mu_A)\mu_H + \alpha_1\gamma\big)(\delta + \mu_A)\big((\delta + \mu_A)\mu_H + \alpha_2\Lambda\big) > 0$$

$$a_5 = \Lambda\alpha_2(\delta + \mu_A)^2\mu_H^3 + (2\Lambda\alpha_2 + (\delta + \mu_A)^2)(\delta + \mu_A)\gamma\alpha_1\mu_H^2 + 2\alpha_2\left(\frac{\gamma\alpha_1}{2} + (\delta + \mu_A)^2\right)\gamma\alpha_1\Lambda\mu_H + \gamma\Lambda^2\alpha_1\alpha_2^2(\delta + \mu_A) > 0$$

$$a_6 = \big((\delta + \mu_A)\mu_H + \gamma\alpha_1\big)\big((\delta + \mu_A)\mu_H + \alpha_2\Lambda\big)(\Lambda\alpha_1\alpha_2 - (\delta + \mu_A)^2\mu_H^2) = \Lambda\gamma\alpha_1\alpha_2 - (\delta + \mu_A)^2\mu_H^2 > 0$$

$$a_6 = (\delta + \mu_A)^2\mu_H^2\Re_0^2 - (\delta + \mu_A)^2\mu_H^2 = (\delta + \mu_A)^2\mu_H^2(\Re_0^2 - 1) > 0 \rightarrow \Re_0^2 - 1 > 0 \rightarrow \Re_0 > 1$$

Since λ_i with $i = 6,7,8$ are negative and $a_4, a_5, a_6 > 0$ indicates that λ_9 and λ_{10} have negative values – also $\Re_0 > 1$. This completes the proof.

4. NUMERICAL SENSITIVITY ANALYSIS

The sensitivity analysis of the plant disease epidemic model is presented in this section. We combine the Latin Hypercube Sampling (LHS) with generate 5000 value samples and Partial Rank

Correlation Coefficients (PRCC) to determine the parameters which have a dominant influence on the model [32]. LHS is a method to perform stratified sampling without replacement. The parameters distribution is sampled and divided into probability intervals equations. Every parameter's intervals are sampled once and the entire range of every parameter is explored. Then, a matrix is generated with N rows and k columns representing the number of samples and varied parameters, respectively. The solution of the model is generated through the combining of parameters. We conducted a sensitivity analysis using the PRCC method on the basic reproduction ratio to determine the most influential factors (parameters) in spreading the disease by ranking the entire parameter in the system. The result of the sensitivity analysis is shown in Figure 2.

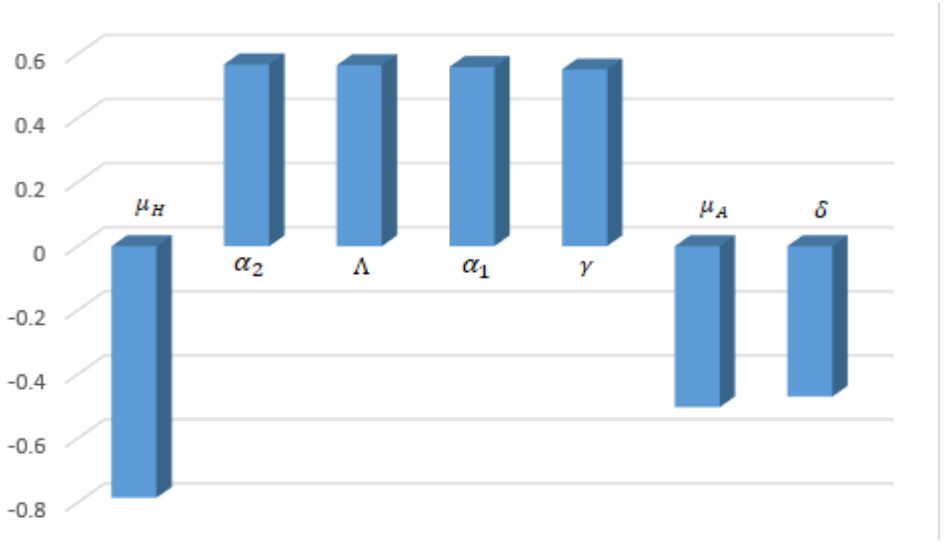


Figure 2. Sensitivity analysis of the basic reproduction ratio.

Figure 2 shows that the plant natural death rate (μ_H) is the most influential parameter with a negative relation to the basic reproduction ratio $(\Re_0)$ in the disease spread phenomenon. When the value of the plant's natural death rate increases, the value of the basic reproduction ratio decreases. It means that the number of infected plants and vectors increases and represents disease severity in the system. Therefore, the plant's natural death rate analysis is feasible to study further and serve an insight that it is essential to suppress the number of infected plants and vectors. The vector natural death rate (μ_A) and vector death rate due to applied *entomopathogen* (δ) also negatively affect this ratio. In contrast, the planting rate (Λ), the vector recruitment rate (γ), and the transmission rate, both plants to vector (α_1) and vector to plants (α_2), have a positive relation to $(\Re_0)$. The positive relation represents that if the parameter's value increases, the $(\Re_0)$ increases. Therefore, it means that decreasing the value of $\Lambda, \gamma, \alpha_1$ and α_2 can reduce the risk of losing crop yields.

5. OPTIMAL CONTROL PROBLEM

In order to control the spread of Mosaic disease with respect to the cost of interventions. Globally, our goal is to minimize the number of vector populations and maximize the number of susceptible generative subpopulations. But, keep in mind the cost of interventions, both *entomopathogen* (u_1) and photoperiodicity (u_2), remains low. We elaborated the optimal control problem to be three scenarios and explained it in the following subsection.

5.1. Scenario 1

The goal of optimal control in this case is to minimize the number of vector populations, both susceptible and infected, with respect to the cost of intervention through applied *entomopathogen*. The objective function for this case is written in the equation (11).

$$J_1(u_1) = \min \int_0^{tf} \left(P_1\left(I_A(t) + S_A(t)\right) + Q_1 u_1(t)^2\right) dt\,, \quad 0 \le t \le t_f, 0 \le u_1 \le 1 \tag{13}$$

Parameters P_1 and Q_1 represent the weight of the vector population and the cost of *entomopathogen* with the performance index satisfies $P_1, Q_1 \ge 0$. We solved the optimal control problem using the Pontryagin Maximum Principle with the variable $y(t) = \begin{bmatrix} S_V(t) \\ S_G(t) \\ I_H(t) \\ S_A(t) \\ I_A(t) \end{bmatrix}$ and the following constraints:

$$\begin{aligned}
\frac{dS_V(t)}{dt} &= \Lambda - \tau\left(1 + \frac{\eta}{1+\eta}\right) S_V(t) - \alpha_1 S_V(t) I_A(t) - \mu_H S_V(t) \\
\frac{dS_G(t)}{dt} &= \tau\left(1 + \frac{\eta}{1+\eta}\right) S_V(t) - \alpha_1 S_G(t) I_A(t) - \mu_H S_G(t) \\
\frac{dI_H(t)}{dt} &= \alpha_1 I_A(t)\left(S_V(t) + S_G(t)\right) - \mu_H I_H(t) \\
\frac{dS_A(t)}{dt} &= \gamma - \alpha_2 S_A(t) I_H(t) - \mu_A S_A(t) - u_1(t)\delta S_A(t) \\
\frac{dI_A(t)}{dt} &= \alpha_2 S_A(t) I_H(t) - \mu_A I_A(t) - u_1(t)\delta I_A(t) \\
S_V(t), S_G(t), I_H(t), S_A(t), I_A(t) &\ge 0
\end{aligned} \tag{14}$$

Note that the control u_1 represents the level of applied *entomopathogen* to the system, especially impacting the vector populations. The value of control shows the maximum effort that will be

given in administering the *entomopathogen* over time. The Hamiltonian function $H_1 = f(y, u_1, t) + \lambda' g(y, u_1, t)$ which is equal to:

$$\begin{aligned} H_1 = {} & P_1\big(S_A(t) + I_A(t)\big) + Q_1 u_1(t)^2 \\ & + \lambda_{S_V}\left(\Lambda - \tau\left(1 + \frac{\eta}{1+\eta}\right)S_V(t) - \alpha_1 S_V(t) I_A(t) - \mu_H S_V(t)\right) \\ & + \lambda_{S_G}\left(\tau\left(1 + \frac{\eta}{1+\eta}\right)S_V(t) - \alpha_1 S_G(t) I_A(t) - \mu_H S_G(t)\right) \\ & + \lambda_{I_H}\left(\alpha_1 I_A(t)\big(S_V(t) + S_G(t)\big) - \mu_H I_H(t)\right) \\ & + \lambda_{S_A}\big(\gamma - \alpha_2 S_A(t) I_H(t) - \mu_A S_A(t) - u_1(t)\delta S_A(t)\big) \\ & + \lambda_{I_A}\big(\alpha_2 S_A(t) I_H(t) - \mu_A I_A(t) - u_1(t)\delta I_A(t)\big) \end{aligned} \tag{15}$$

Where $\lambda_{S_V}, \lambda_{S_G}, \lambda_{I_H}, \lambda_{S_A}$, and λ_{I_A} are the Lagrange multipliers on the optimal control theory. The necessary conditions for scenario 1 should satisfy the following Pontryagin Maximum Principle:

- State equations for this problem can be rewritten as
  $S_V(0) \geq 0, S_G(0) \geq 0, I_H(0) \geq 0, S_A(0) \geq 0, I_A(0) \geq 0$
- Co-state variables

$$\begin{aligned} \frac{d\lambda_{S_V}}{dt} &= -\lambda_{S_V}\left(\tau\left(1 + \frac{\eta}{1+\eta}\right) - \alpha_1 I_A(t) - \mu_H\right) - \lambda_{S_G}\left(\tau\left(1 + \frac{\eta}{1+\eta}\right)\right) - \lambda_{I_H}\big(\alpha_1 I_A(t)\big) \\ \frac{d\lambda_{S_G}}{dt} &= -\lambda_{S_G}(-\alpha_1 I_A(t) - \mu_H) - \lambda_{I_H}\big(\alpha_1 I_A(t)\big) \\ \frac{d\lambda_{I_H}}{dt} &= -\lambda_{I_H}(-\mu_H) - \lambda_{S_A}\big(-\alpha_2 S_A(t)\big) - \lambda_{I_A}\big(\alpha_2 S_A(t)\big) \\ \frac{d\lambda_{S_A}}{dt} &= -P_1 - \lambda_{S_A}(-\alpha_2 I_H(t) - \mu_A - u_1(t)\delta) - \lambda_{I_A}\big(\alpha_2 I_H(t)\big) \\ \frac{d\lambda_{I_A}}{dt} &= -P_1 - \lambda_{S_V}\big(-\alpha_1 S_V(t)\big) - \lambda_{S_G}\big(-\alpha_1 S_G(t)\big) - \lambda_{I_H}\left(\alpha_1\big(S_V(t) + S_G(t)\big)\right) \\ &\quad - \lambda_{I_A}(-\mu_A - u_1(t)\delta) \end{aligned}$$

The stationary condition of the system for scenario 1 is $\frac{\partial H_1}{\partial u_1} = 0$, then $u_1 = \frac{\lambda_{S_A}\delta S_A(t) + \lambda_{I_A}\delta I_A(t)}{2Q_1}$.

Since $0 \leq u_1(t) \leq 1$ then we get

$$u_1^* = \min\left\{\max\left\{0, \frac{\lambda_{S_A}\delta S_A(t) + \lambda_{I_A}\delta I_A(t)}{2Q_1}\right\}, 1\right\} \quad (16)$$

Note that $\frac{\partial^2 H_1}{\partial u_1^2} = 2Q_1 > 0$ satisfies the minimum criterion of optimal control theory with u_1^* being the optimal control of the system.

5.2. Scenario 2

The goal of optimal control in this case is to maximize the number of susceptible generative subpopulations with respect to the cost of intervention through controlling the photoperiodicity. The objective function for this case is written in the equation (17).

$$J_2(u_2) = \max \int_0^{tf} (P_2 S_G(t) - Q_2 u_2(t)^2)\, dt\,, \quad 0 \le t \le t_f, 0 \le u_2 \le 1 \quad (17)$$

Parameters P_2 and Q_2 represent the weight of the susceptible generative subpopulation and the cost of photoperiodicity with the performance index satisfies $P_2, Q_2 \ge 0$. We solved the optimal control problem using the Pontryagin Maximum Principle with the variable $y(t) = \begin{bmatrix} S_V(t) \\ S_G(t) \\ I_H(t) \\ S_A(t) \\ I_A(t) \end{bmatrix}$ and the following constraints:

$$\begin{aligned}
\frac{dS_V(t)}{dt} &= \Lambda - \tau\left(1 + u_2(t)\,\frac{\eta}{1+\eta}\right) S_V(t) - \alpha_1 S_V(t) I_A(t) - \mu_H S_V(t) \\
\frac{dS_G(t)}{dt} &= \tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right) S_V(t) - \alpha_1 S_G(t) I_A(t) - \mu_H S_G(t) \\
\frac{dI_H(t)}{dt} &= \alpha_1 I_A(t)\big(S_V(t) + S_G(t)\big) - \mu_H I_H(t) \\
\frac{dS_A(t)}{dt} &= \gamma - \alpha_2 S_A(t) I_H(t) - \mu_A S_A(t) - \delta S_A(t) \\
\frac{dI_A(t)}{dt} &= \alpha_2 S_A(t) I_H(t) - \mu_A I_A(t) - \delta I_A(t) \\
S_V(t), S_G(t), I_H(t), S_A(t), I_A(t) &\ge 0
\end{aligned} \quad (18)$$

Note that the control u_2 represents the level of controlling photoperiodicity in the system. The value of control shows the maximum effort that will be given in administering the photoperiodicity over time. The Hamiltonian function $H_2 = f(y, u_2, t) + \lambda' g(y, u_2, t)$ which is equal to:

$$
\begin{aligned}
H_2 = {} & P_2 S_G(t) - Q_2 u_2(t)^2 \\
& + \lambda_{S_V}\left(\Lambda - \tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right)S_V(t) - \alpha_1 S_V(t) I_A(t) - \mu_H S_V(t)\right) \\
& + \lambda_{S_G}\left(\tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right)S_V(t) - \alpha_1 S_G(t) I_A(t) - \mu_H S_G(t)\right) \\
& + \lambda_{I_H}\left(\alpha_1 I_A(t)\left(S_V(t) + S_G(t)\right) - \mu_H I_H(t)\right) \\
& + \lambda_{S_A}\left(\gamma - \alpha_2 S_A(t) I_H(t) - \mu_A S_A(t) - \delta S_A(t)\right) \\
& + \lambda_{I_A}\left(\alpha_2 S_A(t) I_H(t) - \mu_A I_A(t) - \delta I_A(t)\right)
\end{aligned} \tag{19}
$$

Where $\lambda_{S_V}, \lambda_{S_G}, \lambda_{I_H}, \lambda_{S_A}$, and λ_{I_A} are the Lagrange multipliers on the optimal control theory. The necessary conditions for scenario 2 should satisfy the following Pontryagin Maximum Principle:

- State equations for this problem can be rewritten as
  $S_V(0) \geq 0, S_G(0) \geq 0, I_H(0) \geq 0, S_A(0) \geq 0, I_A(0) \geq 0$
- Co-state variables

$$
\begin{aligned}
\frac{d\lambda_{S_V}}{dt} &= -\lambda_{S_V}\left(\tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right) - \alpha_1 I_A(t) - \mu_H\right) - \lambda_{S_G}\left(\tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right)\right) \\
&\quad - \lambda_{I_H}\left(\alpha_1 I_A(t)\right) \\
\frac{d\lambda_{S_G}}{dt} &= -P_2 - \lambda_{S_G}(-\alpha_1 I_A(t) - \mu_H) - \lambda_{I_H}\left(\alpha_1 I_A(t)\right) \\
\frac{d\lambda_{I_H}}{dt} &= -\lambda_{I_H}(-\mu_H) - \lambda_{S_A}\left(-\alpha_2 S_A(t)\right) - \lambda_{I_A}\left(\alpha_2 S_A(t)\right) \\
\frac{d\lambda_{S_A}}{dt} &= -\lambda_{S_A}(-\alpha_2 I_H(t) - \mu_A - \delta) - \lambda_{I_A}\left(\alpha_2 I_H(t)\right) \\
\frac{d\lambda_{I_A}}{dt} &= -\lambda_{S_V}\left(-\alpha_1 S_V(t)\right) - \lambda_{S_G}\left(-\alpha_1 S_G(t)\right) - \lambda_{I_H}\left(\alpha_1\left(S_V(t) + S_G(t)\right)\right) - \lambda_{I_A}(-\mu_A - \delta)
\end{aligned}
$$

The stationary condition of the system for scenario 2 is $\frac{\partial H_2}{\partial u_2} = 0$, then $u_2 = \frac{-\lambda_{S_V}\tau\left(\frac{\eta}{1+\eta}\right)S_V + \lambda_{S_G}\tau\left(\frac{\eta}{1+\eta}\right)S_V}{2Q_2}$. Since $0 \leq u_2(t) \leq 1$ then we get

$$
u_2^* = \min\left\{\max\left\{0, \frac{-\lambda_{S_V}\tau\left(\frac{\eta}{1+\eta}\right)S_V + \lambda_{S_G}\tau\left(\frac{\eta}{1+\eta}\right)S_V}{2Q_2}\right\}, 1\right\} \tag{20}
$$

Note that $\frac{\partial^2 H_2}{\partial u_2^2} = -2Q_2 < 0$ satisfies the maximum criterion of optimal control theory with u_2^* being the optimal control of the system.

5.3. Scenario 3

The goal of optimal control, in this case, is to minimize the number of vector populations and maximize the number of susceptible generative subpopulations. We are concerned about the cost of interventions, both *entomopathogen* and photoperiodicity. The objective function for this case is written in the equation (21).

$$J_3(u_1, u_2) = \min \int_0^{tf} \Big(P_1\big(S_A(t) + I_A(t)\big) + Q_1 u_1(t)^2 + Q_2 u_2(t)^2 - P_2 S_G(t)\Big)\, dt, \quad 0 \le t \le t_f, 0 \le u_1, u_2 \le 1 \tag{21}$$

Parameters P_1 and Q_1 represent the weight of the vector population and the cost of *entomopathogen*, while P_2 and Q_2 represent the weight of the susceptible generative subpopulation and the cost of photo-periodicity. The performance index for each parameter is $P_1, Q_1, P_2, Q_2 \ge 0$. We solved the optimal control problem using the Pontryagin Maximum Principle with the variable $y(t) = \begin{bmatrix} S_V(t) \\ S_G(t) \\ I_H(t) \\ S_A(t) \\ I_A(t) \end{bmatrix}$ and the following constraints:

$$\begin{aligned}
\frac{dS_V(t)}{dt} &= \Lambda - \tau\left(1 + u_2(t)\,\frac{\eta}{1+\eta}\right) S_V(t) - \alpha_1 S_V(t) I_A(t) - \mu_H S_V(t) \\
\frac{dS_G(t)}{dt} &= \tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right) S_V(t) - \alpha_1 S_G(t) I_A(t) - \mu_H S_G(t) \\
\frac{dI_H(t)}{dt} &= \alpha_1 I_A(t)\big(S_V(t) + S_G(t)\big) - \mu_H I_H(t) \\
\frac{dS_A(t)}{dt} &= \gamma - \alpha_2 S_A(t) I_H(t) - \mu_A S_A(t) - u_1(t)\delta S_A(t) \\
\frac{dI_A(t)}{dt} &= \alpha_2 S_A(t) I_H(t) - \mu_A I_A(t) - u_1(t)\delta I_A(t) \\
S_V(t), S_G(t), I_H(t), S_A(t), I_A(t) &\ge 0
\end{aligned} \tag{22}$$

Note that the control u_1 and u_2 represent the level of controlling *entomopathogen* and photoperiodicity in the system. The value of control shows the maximum effort that will be given in administering the *entomopathogen* and photoperiodicity over time. The Hamiltonian function $H_3 = f(y, u_1, u_2, t) + \lambda' g(y, u_1, u_2, t)$ which is equal to:

$$
\begin{aligned}
H_3 = {} & P_1\big(S_A(t) + I_A(t)\big) + Q_1 u_1(t)^2 + Q_2 u_2(t)^2 - P_2 S_G(t) \\
& + \lambda_{S_V}\left(\Lambda - \tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right)S_V(t) - \alpha_1 S_V(t) I_A(t) - \mu_H S_V(t)\right) \\
& + \lambda_{S_G}\left(\tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right)S_V(t) - \alpha_1 S_G(t) I_A(t) - \mu_H S_G(t)\right) \\
& + \lambda_{I_H}\left(\alpha_1 I_A(t)\big(S_V(t) + S_G(t)\big) - \mu_H I_H(t)\right) \\
& + \lambda_{S_A}\big(\gamma - \alpha_2 S_A(t) I_H(t) - \mu_A S_A(t) - u_1(t)\delta S_A(t)\big) \\
& + \lambda_{I_A}\big(\alpha_2 S_A(t) I_H(t) - \mu_A I_A(t) - u_1(t)\delta I_A(t)\big)
\end{aligned} \tag{23}
$$

Where $\lambda_{S_V}, \lambda_{S_G}, \lambda_{I_H}, \lambda_{S_A}$, and λ_{I_A} are the Lagrange multipliers on the optimal control theory. The necessary conditions for scenario 3 should satisfy the following Pontryagin Maximum Principle:

- State equations for this problem can be rewritten as
  $S_V(0) \geq 0, S_G(0) \geq 0, I_H(0) \geq 0, S_A(0) \geq 0, I_A(0) \geq 0$
- Co-state variables

$$
\begin{aligned}
\frac{d\lambda_{S_V}}{dt} &= -\lambda_{S_V}\left(\tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right) - \alpha_1 I_A(t) - \mu_H\right) - \lambda_{S_G}\left(\tau\left(1 + u_2(t)\frac{\eta}{1+\eta}\right)\right) \\
&\quad - \lambda_{I_H}\big(\alpha_1 I_A(t)\big) \\
\frac{d\lambda_{S_G}}{dt} &= -P_2 - \lambda_{S_G}(-\alpha_1 I_A(t) - \mu_H) - \lambda_{I_H}\big(\alpha_1 I_A(t)\big) \\
\frac{d\lambda_{I_H}}{dt} &= -\lambda_{I_H}(-\mu_H) - \lambda_{S_A}\big(-\alpha_2 S_A(t)\big) - \lambda_{I_A}\big(\alpha_2 S_A(t)\big) \\
\frac{d\lambda_{S_A}}{dt} &= -\lambda_{S_A}(-\alpha_2 I_H(t) - \mu_A - u_1(t)\delta) - \lambda_{I_A}\big(\alpha_2 I_H(t)\big) \\
\frac{d\lambda_{I_A}}{dt} &= -\lambda_{S_V}\big(-\alpha_1 S_V(t)\big) - \lambda_{S_G}\big(-\alpha_1 S_G(t)\big) - \lambda_{I_H}\left(\alpha_1\big(S_V(t) + S_G(t)\big)\right) \\
&\quad - \lambda_{I_A}(-\mu_A - u_1(t)\delta)
\end{aligned}
$$

The stationary condition of the system for scenario 3 is $\frac{\partial H_3}{\partial u_1} = 0$ and $\frac{\partial H_3}{\partial u_2} = 0$. Since $0 \leq u_1(t), u_2(t) \leq 1$ then we get

$$
u_1^* = \min\left\{\max\left\{0, \frac{\lambda_{S_A}\delta S_A + \lambda_{I_A}\delta I_A}{2Q_1}\right\}, 1\right\} \tag{24}
$$

$$u_2^* = \min\left\{\max\left\{0, \frac{\lambda_{S_V}\tau\left(\frac{\eta}{1+\eta}\right)S_V - \lambda_{S_G}\tau\left(\frac{\eta}{1+\eta}\right)S_V}{2Q_2}\right\}, 1\right\}$$

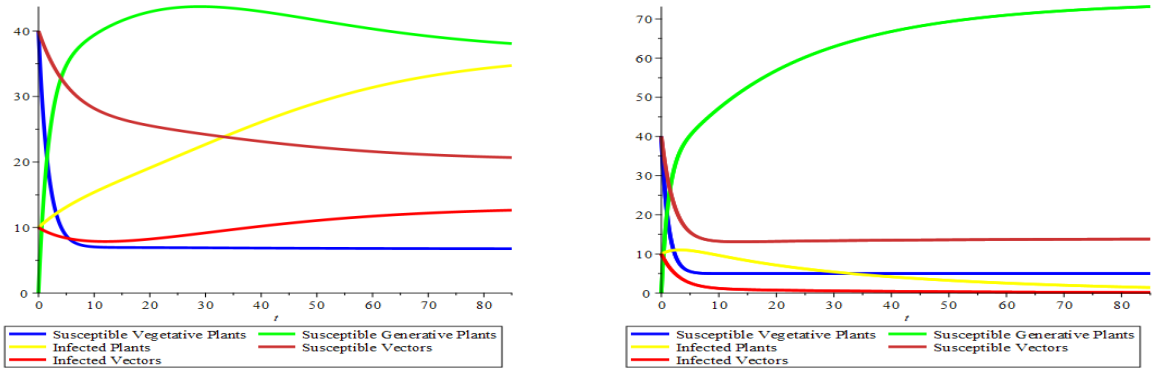
Note that $\frac{\partial^2 H_3}{\partial u_1^2} = 2Q_1 > 0$ $\frac{\partial^2 H_3}{\partial u_2^2} = 2Q_2 > 0$ satisfies the minimum criterion of optimal control theory with u_1^* and u_2^* being the optimal control of the system.

6. NUMERICAL SIMULATION

In this section, we evaluate the model to confirm the system behavior and all scenarios of optimal control problems through a numerical approach. The value parameters used are described in Table 1. Note that scenario 1 and 2 in the optimal control problem has a different use of parameter, including 1) scenario 1 $(\eta = 0)$ and 2) scenario 2 $(\delta = 0)$. It represents there are no use interventions in the scenario, respectively. We obtained some graphical simulation that represents the population dynamics in the phenomenon of soybean Mosaic disease spreads.

6.1. Population Dynamics without Optimal Control Theory

In this subsection, the population dynamics without intervention and with interventions are presented to compare the spread of Mosaic disease in soybean plants. We also confirm the stability of each equilibrium point through the simulation for the long term.



(a) Without intervention represented by $(\Re_0 = 1.7 > 1)$

(b) With intervention represented by $(\Re_0 = 0.7 < 1)$.

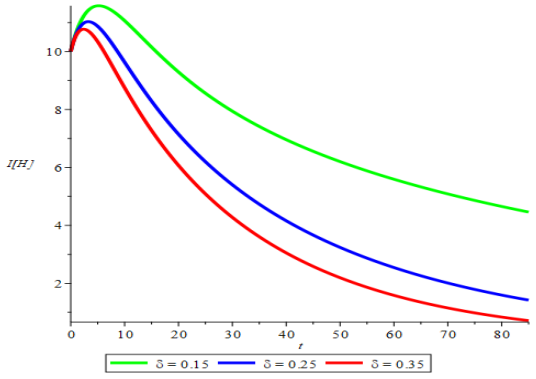
Figure 3. Numerical solution of systems (1)

Figure 3 shows population dynamics without considering the optimal control theory. Based on Figure 3(a), it can be interpreted that the number of vectors, both susceptible and infected, increases without any interventions. Then, the number of infected populations increases over time.

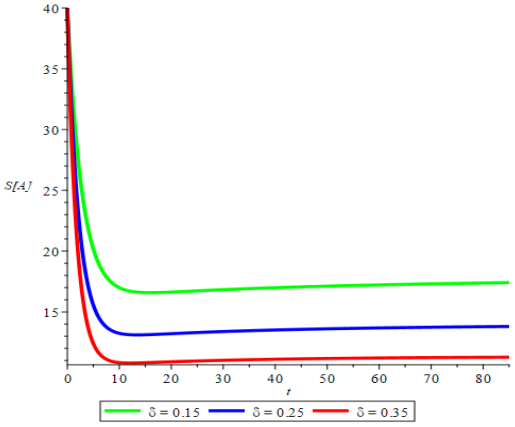
While Figure 3(b) shows that the number of infected populations will go to zero. It is caused by the number of vectors controlled; moreover the subpopulation of the infected plant will go to zero. The explanation can be concluded that the risk of Mosaic disease spreading can be reduced.

6.2. Effect of Entomopathogen

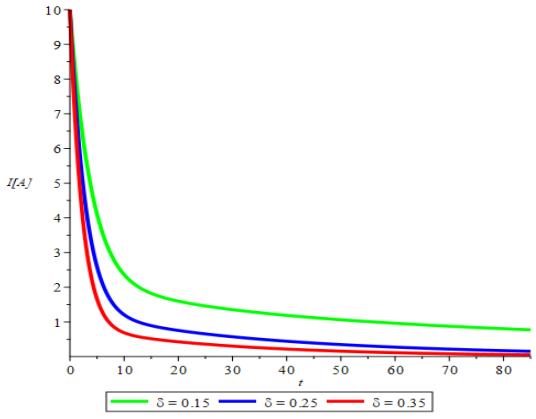
In this subsection, the population dynamics of infected plant, susceptible vector, and infected vector are compared by several value of parameter δ which represents the vector death rate due to *entomopathogen*. Based on previous numerical simulation, it can be shown that *entomopathogen* can suppress both susceptible and infected vector population. Then, it shows that the infected plant decreases over time.



(a) Infected plant population



(b) Susceptible vector population



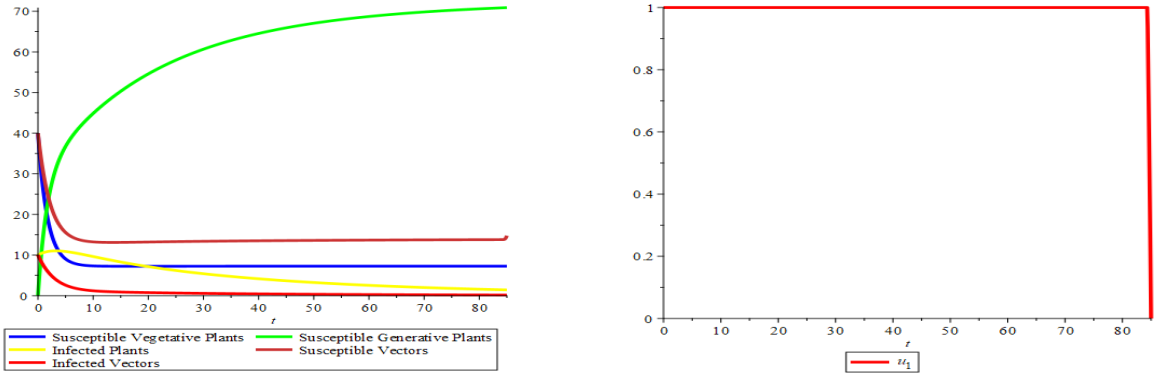
(c) Infected vector population

Figure 4. Numerical solution of systems (1) with various values of δ

Figure 4 show that the higher value of δ on the third case has a significant effect to the system. In this case, the population of infected plant, susceptible vector, and infected vector can be more suppressed if the level of applied *entomopathogen* is higher.

6.3. Population Dynamics with Optimal Control Scenario 1

In scenario 1, a simulation is carried out by considering the control optimal of *entomopathogen*, but without photoperiodicity intervention. Therefore, parameter value $u_1(t) = u_1^*(t)$ is applied to the simulation. The result is obtained in Figure 5.



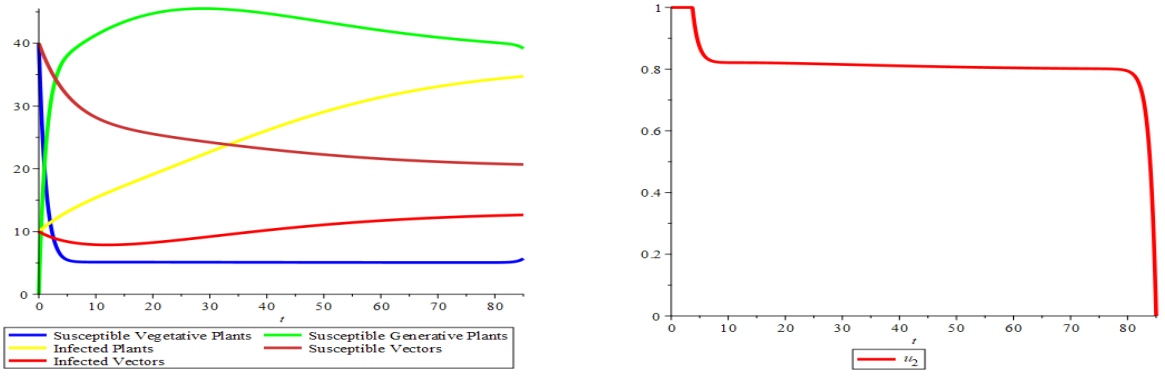
(a) Population dynamics (b) Control function of *entomopathogen*

Figure 5. Numerical solution of optimal control in scenario 1

Figure 5(b) shows that the level of applied *entomopathogen* was almost always on the top level $(u_1^*(t) = 1)$ over time. Therefore, the vector population can be controlled and simultaneously impacted to the system, represented by the number of infected plants going to zero (see Figure 5(a)). Then, scenario 1 can be said to successfully control the disease by reducing the number of vector populations.

6.4. Population Dynamics with Optimal Control Scenario 2

In scenario 2, a simulation is carried out by considering the control optimal of photoperiodicity but without *entomopathogen* intervention. Therefore, parameter value $u_2(t) = u_2^*(t)$ is applied to the simulation. The result is obtained in Figure 6.



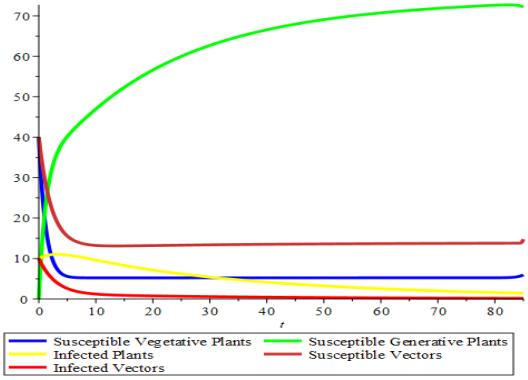
(a) Population dynamics (b) Control function of photoperiodicity

Figure 6. Numerical solution of optimal control in scenario 2

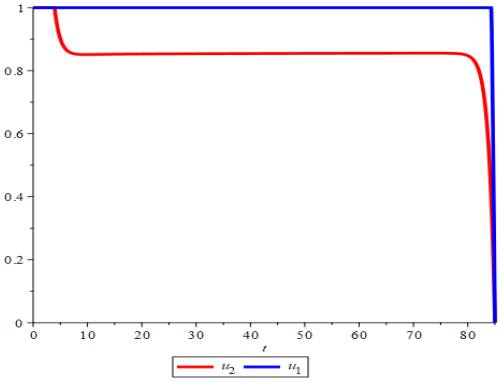
Based on Figure 6, it is presented that the disease is still spreading in the system. The control in this scenario is indirect to impact the vector populations, but optimize the growth and development of plants. So the number of vectors is uncontrolled and causes the infected plant to increase. We conclude that scenario 2 is not successful in reducing the risk of disease spreading.

6.5. Population Dynamics with Optimal Control Scenario 3

A simulation is carried out in scenario 3 by considering the control optimal of *entomopathogen* and photoperiodicity intervention. Therefore, parameter value $u_1(t) = u_1^*(t)$ and $u_2(t) = u_2^*(t)$ is applied to the simulation. The result is obtained in Figure 7.



(a) Population dynamics



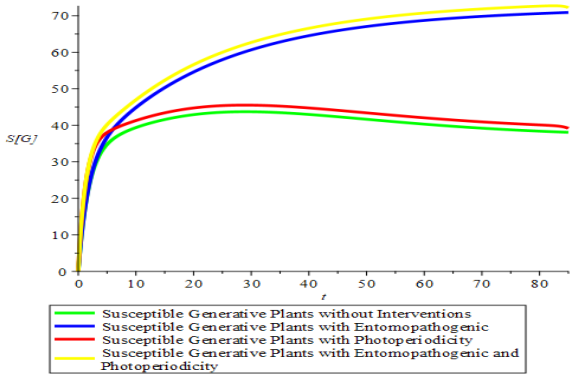
(b) Control function of *entomopathogen* and photoperiodicity

Figure 7. Numerical solution of optimal control in scenario 3

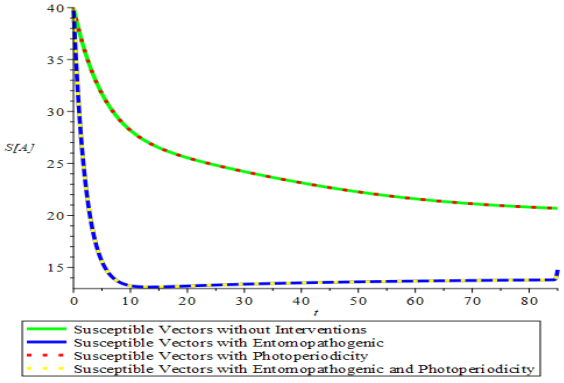
The control function graph shows that the control values for each $u_1(t)$ and $u_2(t)$ are not different from those obtained in scenarios 1 and 2. Globally, we see that the number of infected plants and vectors can be reduced. It represents that the disease is not spreading in the system and scenario 3 can be said to control the disease successfully. But, we have to compare the population dynamics in each scenario, including no control, 1, 2, and 3, to see the difference specifically.

6.6. Comparison of Population Dynamics

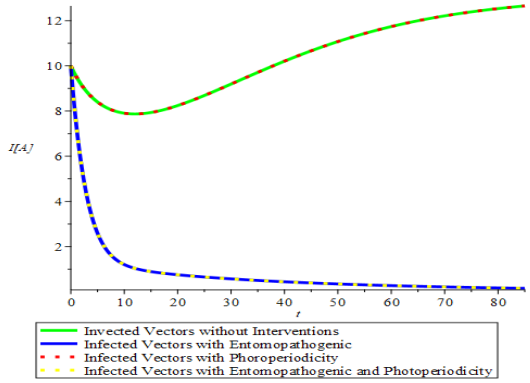
This subsection shows the population dynamics in the susceptible generative plants, susceptible vectors, and infected vectors as the main subpopulations of optimal control targets through four scenarios. The first scenario is to show population dynamics without intervention in the system. While the second, third, and fourth scenarios are scenarios 1, 2, and 3 in the optimal control problem. The results obtained are shown in Figure 8.



(a) Susceptible generative plant population



(b) Susceptible vector population



(c) Infected vector population

Figure 8. Comparison of population dynamics in several compartments

Figure 8 shows that the intervention taking into the optimal control theory impacted the system. It represents the success of the applied intervention in controlling the spread of Mosaic disease by reducing the number of vectors population and optimizing the growth and development of plant processes. Based on Figure 8(a), it can be seen that the scenario of exercising control of *entomopathogen* and photoperiodicity in the system can produce a number of susceptible generative plants higher than the other three scenarios. This is caused by reduced vector populations, both susceptible and infected, in the system that plays a virus spreader role.

7. CONCLUSIONS

In this paper, we formulated a mathematical model for studying the spread of Mosaic disease considering applying the *entomopathogen* and control photoperiodicity as interventions. The model is a compartmental-based model where the plant and vector population divided to be five compartments, including 1) susceptible vegetative plants (S_V), 2) susceptible generative plants

(S_G), 3) infected plants (I_H), 4) susceptible vectors (S_A), and 5) infected vectors (I_A). The model is analyzed by mathematics theories, and the result shows that there are one non-endemic equilibrium point and one endemic equilibrium point. Then the basic reproduction ratio is obtained by using the next-generation matrix method. The equilibrium points are stable for each condition, respectively, it showed analytically in section 3. Sensitivity analysis is presented to show the most influential parameter in the system. The optimal condition is obtained by applying the Pontryagin Maximum Principles to the optimal control problem. Numerical simulations is presented to confirm the analytical result and figure out the population dynamics by graphic to describing the Mosaic disease phenomenon. The result shows that the intervention impacted the system of Mosaic disease spreading. Combined interventions such as *entomopathogen* and photoperiodicity is the best scenario to suppress the vector populations and infected plants that impacted to reduce the risk of crop loss. The result obtained may be used as a reference to preventing and controlling the spread of Mosaic disease.

ACKNOWLEDGMENTS

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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