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INVESTIGATING THE DIFFERENT VARIETY EFFECTS FOR PLANT DISEASE TRANSMISSION UNDER BIOINSECTICIDE AND ROGUING INTERVENTIONS: A MATHEMATICAL MODELING STUDY

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Abstract: A severe malady of soybean plants, called mosaic disease, can lower both the quality and quantity of soybean production. This disease can be transmitted by Aphis, a vector that carries the Soybean Mosaic Virus (SMV). This study constructs a deterministic model to represent its transmission mechanism and analyze the phenomenon. The model includes a bioinsecticide utilization mechanism to suppress the vector population and considers the roguing effort to reduce the risk of further infection of other plants. In addition, plant variety is investigated to explore the different effects of disease transmission based on the number of healthy plants at the harvesting period. Dynamical system theory is utilized to obtain the equilibrium solutions and their stability. The basic reproduction number ($\mathcal{R}_0$) for this model is obtained using the next-generation matrix method, and it is used to explore the existence of bifurcations. Next, a sensitivity analysis is performed to reveal the most influential factor in suppressing the disease. An optimal control study is conducted by applying three interventions, such as bioinsecticides, roguing, and preventive plant treatment, resulting in eight scenarios. Finally, numerical simulation is used to confirm the results of the analysis and provide dynamic predictions along the observation period. This study identifies a threshold number for

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implementing a control scenario based on the level of resistance, which is represented by the soybean variety.

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1. INTRODUCTION

Soybeans are an essential source of oil and protein, which are frequently incorporated into the nutritional regimes of humans and animals [1]. With protein and oil concentrations of around 39 to 44 percent and 21 percent, respectively, soybeans are often referred to as an extraordinary phytonutrient plant [2]. The levels of both render it an exemplary substitute for animal-based proteins. Soybean derivatives permeate a vast array of products, including vegan meals, flour, and plant-based cheese, among other foodstuffs, underlining their significance in both industrial utilization and everyday consumption [3].

Owing to its significance within the nutritional framework, soybean availability must scale up to keep pace with annual population growth and its escalating utilization as a fundamental input in the food industry. For instance, domestic demand for soybeans in Indonesia was found to increase by 2.73-3.29 million tons between 2020 and 2024 [4]. However, during the period of 2018 to 2020, an annual average decline in soybean production was revealed at a rate of 6.12%. Concurrently, soybean demand and imports exhibited a linear increase, with average annual increases of around 3.30% and 3.03%, respectively [5]. Khan et al. [6] revealed that a disease called Soybean Mosaic Disease (SMD) is one factor that must be considered when investigating the decline of soybean productivity. The presence of SMD in soybean cultivation accounts for a reduction in its yield ranging from 35-50% [7].

Soybean Mosaic Disease (SMD), caused by the Soybean Mosaic Virus (SMV), poses a significant agronomic challenge, impairing soybean yields. The virus can be transmitted through three pathways, namely mechanical transmission, insect transmission, and grafting transmission [8]. The pathogenesis of SMD is initiated by aphids, which act as vectors that facilitate SMV transmission among plants. Widyasari et al. [9] confirmed this by revealing that aphids are the

principal means of SMV transmission. It is widespread in all soybean-producing regions and possesses the capacity to infect a spectrum of other economic crops.

The transmission probability of the SMV increases due to the presence of Mosaic transmission vectors, including aphids, which are prevalent in the agricultural environment. Therefore, the use of bioinsecticides is considered to control their numbers in this study. Nevertheless, the aim of using bioinsecticides is not to eliminate the aphid population, but rather to maintain its existence while ensuring the aphids remain healthy. This aligns with the principle of Integrated Pest Management (IPM), which aims to maintain pest populations below economic threshold levels, rather than completely exterminating the pest or pathogen population [10]. IPM has been considered the application of insecticides targeting these kinds of vectors, which is associated with a marginal reduction in mosaic disease cases [8], [11]. However, it is important to pay closer attention to insecticide application, due to the potential phytotoxic effect on the plants. To prevent this risk, a natural bioinsecticide called *Lecanicillium Lecanii* (*L. Lecanii*) can be used. Diaz et al. [10] revealed that *L. Lecanii* is a fungus with pathogenic effects on insects and could serve as a potent biological control agent capable of managing agricultural pests. Amelia et al. [12] confirmed their findings when studying aphid dynamics in the presence of *L. Lecanii*. While the fungus plays a role in preventing SMV transmission to aphids, farmers can reduce the risk by conducting roguing efforts to accelerate the goal.

Roguing, a traditional identification and removal technique of infected plants, remains a cornerstone of Integrated Pest Management (IPM). However, the efficacy of both IPM and PDM are heavily dependent on the roguing effort. Adrakey et al. [13] revealed that roguing effort encompasses the frequency, accuracy, and labor intensity in inspecting the field. Traditionally, this effort requires significant experienced human resources to visually identify early symptoms, which is time-consuming and more economically burdensome. Furthermore, roguing is vulnerable to human error due to fatigue, leading to incomplete disease eradication and subsequent outbreaks.

Given the challenges posed by SMD and its substantial impact on agricultural productivity, it is imperative to explore the dynamics of SMV transmission and disease spread rigorously.

Moreover, the various transmission mechanisms of SMV require a comprehensive examination to formulate effective control strategies. Mathematical modeling emerges as a pivotal tool in this context. This approach has been used to examine phenomena such as the spread of infectious diseases within the human population [14], [15] and plants [16].

Many researchers have formulated mathematical models to study and analyze the long-term behavior of various transmissible issues and their effects. Compartmental-based models, a specific type of mathematical model, were used in these studies by separating the population into multiple subpopulations with distinct descriptions. This approach can also be used to study transmissible diseases in plants. Tresna et al. [16] presented this in their literature review, which identified and revealed gaps in the study of mathematical modeling in plant epidemiology.

A variety of vector-borne disease models have been developed to characterize the epidemiology of plant disease. To account for the latent period of infection in the plant population, Jeger [17] proposed a mathematical model for plant disease. Examining local [18] and global stability [19] within plant disease models, as explored in previous studies, facilitates a deeper understanding of its behavior over extended periods. Luo et al. [20] and Al-Basir et al. [21] considered management strategies such as roguing and replanting to mitigate a broader spread. Additionally, the application of pesticides has been shown to halt the spread of the disease [22]. In the IPM context, the suppression of vector populations through insecticides or bioinsecticides is presented as a parameter within mathematical models for controlling disease spread and reducing its effect. Suryaningrat et al. [23] extended the study by including the presence of biological organisms as vector-transmission predators.

Several vector-borne mathematical models for plant disease were also investigated using optimal control theory. In an optimal control problem, a parameter represents various interventions in a mathematical model and is treated as a control parameter; see [18], [24], [25] for examples. Chowdury et al. [24] investigated pest control through pesticide use within plant disease frameworks using dynamical analysis alongside optimal control theory. Anggriani et al. [18] proposed an optimal control model to explore the long-term behavior of plant disease transmission

under the presence of a curative treatment as a control parameter. Furthermore, Bokil et al. [25] formulated a mathematical model of African Cassava Mosaic Disease (ACMD) transmission and investigated its behavior using mathematical analysis and optimal control theory.

Given the vector-borne nature of SMD, there is a crucial view that a compartmental model can meet the needs of exploring its behavior. This approach yields a robust analysis for understanding disease management. The aphids are viewed as vectors in the model to depict the process of disease transmission from infected to susceptible plants. By analyzing disease-spread patterns in the model, it can also simulate disease transmission under various scenarios to predict transmission and the effects of model-based interventions.

In this study, we formulate a mathematical model that incorporates the use of bioinsecticides to suppress vector numbers and roguing to eliminate infected plants from the ecosystem. In addition, the model also accounts for varying levels of plant resistance to reveal the different conditions of SMD behavior. We examine the equilibrium solution of the formulated model and investigate its stability. A sensitivity analysis was carried out using Latin Hypercube Sampling (LHS) and Partial Rank Correlation Coefficients (PRCC) to identify the most influential parameters in terms of disease spread. Next, we set both bioinsecticide and roguing as control parameters, and another preventive control as a multiplier effect of the plant resistance level. We formulate and resolve the optimal control model to establish the most effective control conditions. Prior to this, we satisfy the prerequisites of the Pontryagin Maximum Principle to ensure the solution's viability [26]. The analytical findings are substantiated through numerical simulations. Finally, we conclude the study with a thorough analysis of the findings and propose some recommendations for further studies.

2. MATHEMATICAL MODEL

In modeling the SMD transmission, both plant and vector populations are considered. First, plants are divided into three subpopulations, namely susceptible (S_h), exposed (E_h), and infected (I_h) plants. Next, vectors are divided into two subpopulations, namely susceptible (S_v) and infected (I_v) vectors. To formulate the model, we use three assumptions, including 1) infected

plants and infected vectors cannot be recovered; 2) susceptible and exposed plants are dead naturally; 3) the applied bioinsecticides only affect the vector population; and 4) the variety planted crop in the field is same. Also, some certain aspects that are thought to be involved are represented by parameters (see Table 1). Finally, we can figure out a schematic diagram to describe the transmission process of the Mosaic virus in both plant and vector populations (see Figure 1).

Table 1. Descriptions and values of parameters.

Parameter	Description	Value	Unit
Λ_h	Replanting rate	4	$plant \times day^{-1}$
μ_h	Plant natural death rate	0.005	day^{-1}
β_h	Transmission rate on plant	0.9	day^{-1}
α	Transition rate into infectious plant	0.2	day^{-1}
μ_d	Plant death rate due to the disease	0.01	day^{-1}
Λ_v	Vector natural birth rate	6	$plant \times day^{-1}$
μ_v	Vector natural death rate	0.18	day^{-1}
K	Carrying capacity	1000	$plant$
β_v	Transmission rate on vector	0.5	day^{-1}
δ	Vector death rate due to bioinsecticides	0.25	day^{-1}
u_1	Preventing effort	0.5	Nondimensional
u_2	Roguing effort	0.5	Nondimensional
u_3	Spraying effort	0.5	Nondimensional

Given that all parameters are non-negative.

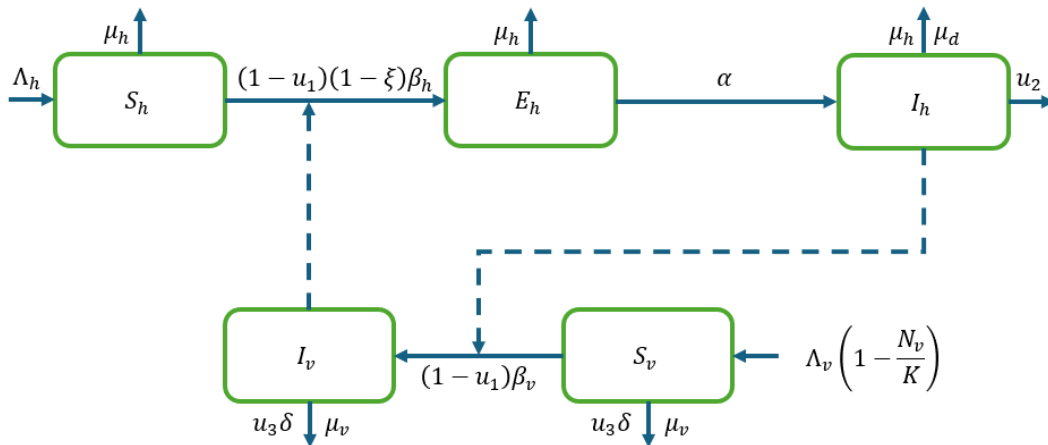


Figure 1. Schematic diagram of the spread of Mosaic disease in Soybean plants. The solid lines represent the transition of plants or vectors from one subpopulation to another, while the dashed line represents the subpopulations involved in the transmission term.

Based on Figure 1, the compartmental-based model for the spread of the SMD can be written as Equations (1).

$$\begin{aligned}
\frac{dS_h(t)}{dt} &= \Lambda_h - (1 - u_1)(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} S_h(t) - \mu_h S_h(t), \\
\frac{dE_h(t)}{dt} &= (1 - u_1)(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} S_h(t) - \alpha E_h(t) - \mu_h E_h(t), \\
\frac{dI_h(t)}{dt} &= \alpha E_h(t) - \mu_d I_h(t) - u_2 I_h(t) - \mu_h I_h(t), \\
\frac{dS_v(t)}{dt} &= \Lambda_v N_v(t) \left(1 - \frac{N_v(t)}{K}\right) - (1 - u_1)\beta_v \frac{I_h(t)}{N_h(t)} S_v(t) - \mu_v S_v(t) - \delta u_3 S_v(t), \\
\frac{dI_v(t)}{dt} &= (1 - u_1)\beta_v \frac{I_h(t)}{N_h(t)} S_v(t) - \mu_v I_v(t) - \delta u_3 I_v(t),
\end{aligned} \tag{1}$$

where $N_h(t) = S_h(t) + E_h(t) + I_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$. Note that the initial conditions of each compartment as equation (2).

$$S_h(0) \geq 0, E_h(0) \geq 0, I_h \geq 0, S_v(0) \geq 0, I_v(0) \geq 0. \tag{2}$$

3. MAIN RESULTS

3.1. Properties of Solution

Theorem 1. *Given that the initial values of system (1) are $S_h(0), E_h(0), I_h(0), S_v(0), I_v(0) \geq 0$, the solution of $S_h(t), E_h(t), I_h(t), S_v(t)$, and $I_v(t)$ are always non-negative for all time $t \geq 0$.*

Proof. Refer to non-negativity proving by Erick and Mayengo [27]. If $S_h(0), E_h(0), I_h(0), S_v(0), I_v(0) \geq 0$, then from the first equation in system (1) we have

$$\begin{aligned}
\frac{dS_h(t)}{dt} &= \Lambda_h - (1 - u_1)(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} S_h(t) - \mu_h S_h(t), \\
\frac{dS_h(t)}{dt} &\geq -(1 - u_1)(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} S_h(t) - \mu_h S_h(t).
\end{aligned}$$

Separating the variables of $S_h(t)$ and integrating both sides yield

$$\int \frac{dS_h(t)}{S_h(t)} \geq \int \left(-(1 - u_1)(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} - \mu_h \right) dt,$$

$$\ln S_h(t) \geq \left(-(1 - u_1)(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} - \mu_h \right) t + C,$$

$$S_h(t) \geq B e^{-\left((1 - u_1)(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} + \mu_h \right) t}.$$

If we substitute $t = 0$ as our initial condition, we obtain $S_h(0) \geq B$. Hence, the above expression can be rewritten as

$$S_h(t) \geq S_h(0)e^{-\left((1-u_1)(1-\xi)\beta_h\frac{I_v(t)}{N_v(t)}+\mu_h\right)t}.$$

Theoretically, $S_h(t)$ will always non-negative once $t \geq 0$. By conducting the same procedures to all equations in system (1) for all $t \geq 0$, we obtain that $E_h(t), I_h(t), S_v(t), I_v(t) \geq 0$. Therefore, the solution of system (1) is non-negative for all time $t \geq 0$. ■

Theorem 2. *The total plant (N_h) and vector (N_v) population in system (1) is eventually bounded as $t \rightarrow \infty$.*

Proof. First, we adding the first to third equations in system (1), we obtain

$$\begin{aligned} \frac{dN_h(t)}{dt} &= \Lambda_h - \mu_h N_h - (\mu_d + u_2)I_h, \\ \frac{dN_h(t)}{dt} &\leq \Lambda_h - \mu_h N_h. \end{aligned} \tag{3}$$

Solving the second expression of equation (3) with respect to $N_h(t)$ and with a non-negative initial condition $N_h(0) \geq 0$ gives

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h}\right)e^{-\mu_h t}. \tag{4}$$

Hence, if we take $t \rightarrow \infty$, then we have that $N_h(t)$ is eventually bounded by Λ_h/μ_h . For vector population, by adding the fourth and fifth equations in system (1), we have

$$\frac{dN_v(t)}{dt} = N_v \left((\Lambda_v - \mu_v - \delta u_3) - \frac{\Lambda_v}{K} N_v \right). \tag{5}$$

By letting $r_v = \Lambda_v - \mu_v - \delta u_3$, the fifth equation can be rewritten as

$$\frac{dN_v(t)}{dt} = N_v(t) \left(r_v - \frac{\Lambda_v}{K} N_v(t) \right). \tag{6}$$

To ensure that the solution of vector population is also bounded, we have to consider two possibilities as follows:

- For $r_v > 0$, which net growth rate is positive. The equation (6) can be reformulated as

$$\frac{dN_v(t)}{dt} = r_v N_v(t) \left(1 - \frac{N_v(t)}{K_{eff}} \right), \text{ where } K_{eff} = K \left(1 - \frac{\mu_v + \delta u_3}{\Lambda_v} \right) < K.$$

Solving the above equation using the same procedure as in obtaining equation (4) yields

$$N_v(t) = \frac{K_{eff}}{1 + \left(\frac{K_{eff}}{N_v(0)} - 1 \right) e^{-r_v t}}.$$

Hence, if we take $t \rightarrow \infty$, then we have that $N_v(t)$ is eventually bounded by $K_{eff} < K$ once $r_v > 0$.

- For $r_v < 0$, which net growth rate is negative. Since we know that $-\Lambda_v N_v^2 / K$ is negative, then the equation (6) can be reformulated as

$$\frac{dN_v(t)}{dt} \leq r_v N_v \leq 0.$$

By using the same procedure as in obtaining equation (4) for the three parties, we can write the solution as

$$N_v(t) \leq N_v(0) e^{r_v t} \leq N_v(0).$$

Consequently, $N_v(t) \rightarrow 0$ as $t \rightarrow \infty$.

Based on the results of both analyses for positive and negative net growth, we can conclude that the vector population remains bounded as

$$0 \leq N_v(t) \leq \max\{N_v(0), K\}. \blacksquare$$

According to the proofs of Theorems 1 and 2, the result of system (1) always fulfils the biological means as it is non-negative and bounded.

3.2. Equilibrium Solution

The equilibrium solution of the model is obtained by equating all time derivatives to zero. In other words,

$$\frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dI_h}{dt} = \frac{dS_v}{dt} = \frac{dI_v}{dt} = 0.$$

Based on the equation above, two equilibrium solutions are obtained: the disease-free equilibrium (DFE) and the endemic equilibrium (EE). The DFE is given by

$$Eq_{DFE} = (S_h^*, E_h^*, I_h^*, S_v^*, I_v^*),$$

$$Eq_{DFE} = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{K(\Lambda_v - u_3 \delta - \mu_v)}{\Lambda_v}, 0 \right).$$

The disease-free equilibrium is employed in the computation of the basic reproduction number

($\mathfrak{R}_0$). In this study, $\mathfrak{R}_0$ is calculated using the Next-Generation Matrix (NGM) approach by evaluating the new-infection and transition matrices at the DFE. The derivation begins by identifying the infected compartments and expressing the corresponding model equations as two vector-valued functions. The vector c represents the rate of newly generated infections, whereas the vector d accounts for all other transitions involving the infected compartments. Accordingly, these vectors are defined as

$$c = \begin{pmatrix} (1 - u_1(t))(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} S_h(t) \\ 0 \\ (1 - u_1(t))\beta_v \frac{I_h(t)}{N_h(t)} S_v(t) \end{pmatrix}, d = \begin{pmatrix} \alpha E_h(t) + \mu_h E_h(t) \\ -\alpha E_h(t) + \mu_d I_h(t) + u_2(t) I_h(t) + \mu_h I_h(t) \\ \mu_v I_v(t) + \delta u_3(t) I_v(t) \end{pmatrix}.$$

The Jacobian matrices of c and d , denoted by F and V , are evaluated at the DFE, yielding

$$F = \begin{pmatrix} 0 & 0 & (1 - u_1(t))(1 - \xi)\beta_h \left(\frac{\Lambda_v \Lambda_h}{K\mu_h(\Lambda_v - u_3\delta - \mu_v)} \right) \\ 0 & 0 & 0 \\ 0 & (1 - u_1(t))\beta_v \left(\frac{K\mu_h(\Lambda_v - u_3\delta - \mu_v)}{\Lambda_v \Lambda_h} \right) & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \alpha + \mu_h & 0 & 0 \\ -\alpha & \mu_d + u_2(t) + \mu_h & 0 \\ 0 & 0 & \mu_v + \delta u_3(t) \end{pmatrix}.$$

Therefore, the basic reproduction number is given by

$$\mathfrak{R}_0 = \sqrt{\frac{\alpha\beta_h\beta_v(1 - u_1)^2(1 - \xi)}{(\alpha + \mu_h)(u_2 + \mu_d + \mu_h)(u_3\delta + \mu_v)'}}$$

where $\xi \in [0,1]$.

The second equilibrium is the endemic equilibrium, denoted by Eq_{EE} , corresponds to the steady state in which the disease remains present in both the human and vector populations, i.e.,

$$Eq_{EE} = (S_h^{**}, E_h^{**}, I_h^{**}, S_v^{**}, I_v^{**}),$$

with

$$S_h^{**} = \frac{\Lambda_h(\alpha((-1 + u_1)\beta_v - u_3\delta - \mu_v) - (u_2 + \mu_d + \mu_h)(u_3\delta + \mu_v))}{\alpha((u_2 + \mu_d)(u_3\delta + \mu_v) + (-1 + u_1)\beta_v(\mu_h + (-1 + u_1)\beta_h(-1 + \xi)))},$$

$$E_h^{**} = \frac{\Lambda_h B}{\alpha(\alpha + \mu_h)((u_2 + \mu_d)(u_3\delta + \mu_v) + (-1 + u_1)\beta_v(\mu_h + (-1 + u_1)\beta_h(-1 + \xi)))},$$

$$\begin{aligned}
I_h^{**} &= \frac{\Lambda_h B}{(\alpha + \mu_h)(u_2 + \mu_d + \mu_h)((u_2 + \mu_d)(u_3\delta + \mu_v) + (-1 + u_1)\beta_v(\mu_h + (-1 + u_1)\beta_h(-1 + \xi)))}, \\
S_v^{**} &= \frac{K(u_3\delta + \mu_v)(u_3\delta - \Lambda_v + \mu_v)(C + \alpha(\mu_d + \mu_h + (-1 + u_1)\beta_h(-1 + \xi)))}{(-1 + u_1)\beta_h\Lambda_v(\alpha((-1 + u_1)\beta_v - u_3\delta - \mu_v) - (u_2 + \mu_d + \mu_h)(u_3\delta + \mu_v))(-1 + \xi)}, \\
I_v^{**} &= -\frac{K(u_3\delta - \Lambda_v + \mu_v)B}{(-1 + u_1)\beta_h\Lambda_v(\alpha((-1 + u_1)\beta_v - u_3\delta - \mu_v) - (u_2 + \mu_d + \mu_h)(u_3\delta + \mu_v))(-1 + \xi)}.
\end{aligned}$$

where

$$B = \mu_h(u_2 + \mu_d + \mu_h)(u_3\delta + \mu_v) + \alpha((u_2 + \mu_d + \mu_h)(u_3\delta + \mu_v) + (u_1 - 1)^2\beta_h\beta_v(\xi - 1)),$$

$$C = \alpha(\mu_d + \mu_h + (-1 + u_1)\beta_h(-1 + \xi)).$$

3.3. Local Stability of Disease-free Equilibrium

Theorem 3. *The disease-free equilibrium is locally asymptotically stable if $\mathfrak{R}_0 < 1$ and $\Lambda_v > \mu_v + u_3\delta$. Conversely, the disease-free equilibrium is unstable if $\mathfrak{R}_0 > 1$.*

Proof. To examine the local stability of non-endemic equilibrium, consider the Jacobian matrix of the model evaluated at the disease-free equilibrium. The resulting matrix is given by

$$J_{DFE} = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & D \\ 0 & -\alpha - \mu_h & 0 & 0 & -D \\ 0 & \alpha & -u_2 - \mu_d - \mu_h & 0 & 0 \\ 0 & 0 & M & u_3\delta - \Lambda_v + \mu_v & 2u_3\delta + 2\mu_v - \Lambda_v \\ 0 & 0 & -M & 0 & -u_3\delta - \mu_v \end{pmatrix},$$

where

$$D = \frac{\Lambda_h \Lambda_v (-1 + u_1) \beta_h (1 - \xi)}{\mu_h K (-u_3\delta + \Lambda_v - \mu_v)}, \quad M = \frac{K (-u_3\delta + \Lambda_v - \mu_v) \mu_h (-1 + u_1) \beta_v}{\Lambda_h \Lambda_v}.$$

The characteristic equation associated with J_{DFE} is obtained from $|\lambda I - J_{DFE}| = 0$. After simplification, the characteristic equation can be expressed in the form

$$(\lambda + \mu_h)(\lambda - u_3\delta + \Lambda_v - \mu_v)[\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3] = 0,$$

with

$$b_1 = u_3\delta + \mu_v + u_2 + \mu_d + \mu_h + \alpha + \mu_h > 0,$$

$$b_2 = (u_2 + \mu_d + \mu_h + \alpha + \mu_h)(u_3\delta + \mu_v) + (\alpha + \mu_h)(u_2 + \mu_d + \mu_h) > 0,$$

$$b_3 = (\alpha + \mu_h)(u_2 + \mu_d + \mu_h)(u_3\delta + \mu_v)[1 - \mathfrak{R}_0^2] > 0 \text{ if } \mathfrak{R}_0 < 1, \text{ and } b_3 < 0 \text{ if } \mathfrak{R}_0 > 1.$$

Hence, the first two eigenvalues are given by

$$\lambda_1 = -\mu_h < 0, \lambda_2 = -(\Lambda_v - (\mu_v + u_3\delta)) < 0 \text{ if } \Lambda_v > \mu_v + u_3\delta$$

The remaining three eigenvalues are determined from the corresponding characteristic polynomial using the Routh–Hurwitz criterion. If $\mathfrak{R}_0 < 1$, then $b_1 > 0, b_2 > 0, b_3 > 0$, and $b_1 b_2 - b_3 = P + Q + R > 0$ where

$$P = (u_3 \delta + \mu_v)(u_2 + \mu_d + \mu_h + \alpha + \mu_h)(u_3 \delta + \mu_v),$$

$$Q = (u_2 + \mu_d + \mu_h + \alpha + \mu_h)((u_2 + \mu_d + \mu_h + \alpha + \mu_h)(u_3 \delta + \mu_v) + (\alpha + \mu_h)(u_2 + \mu_d + \mu_h)),$$

$$R = (\alpha + \mu_h)(u_2 + \mu_d + \mu_h)(u_3 \delta + \mu_v)\mathfrak{R}_0^2.$$

So, the Routh-Hurwitz criterion for the third order characteristic equation is satisfied. Hence, the disease-free equilibrium solution is locally asymptotically stable if $\mathfrak{R}_0 < 1$ and $\Lambda_v > \mu_v + u_3 \delta$.

For $\mathfrak{R}_0 > 1$, the instability of the disease-free equilibrium is established using Vieta's theorem. Consider the third-order characteristic equation, if the cubic equation has three distinct roots, denoted by $\lambda_3, \lambda_4, \lambda_5$, then, by Vieta's theorem,

$$\lambda_3 + \lambda_4 + \lambda_5 = -b_1 < 0, \quad \lambda_3(\lambda_4 + \lambda_5) + \lambda_4 \lambda_5 = b_2 > 0, \quad \lambda_3 \lambda_4 \lambda_5 = -b_3 > 0.$$

This equation applies if there are two negative eigenvalues or all of them are positive. Thus, there is at least one positive eigenvalue. So, the disease-free equilibrium solution is unstable if $\mathfrak{R}_0 > 1$.

■

3.4. Bifurcation Analysis

Theorem 4. *The system undergoes a transcritical bifurcation at $\mathfrak{R}_0 = 1$. The endemic equilibrium is biologically feasible for $\mathfrak{R}_0 > 1$ and emerges as $\mathfrak{R}_0$ increases through unity. Hence, the bifurcation is a forward transcritical bifurcation.*

Proof. Suppose β_h is chosen as the bifurcation parameter and $x_1 = S_h, x_2 = E_h, x_3 = I_h, x_4 = S_v, x_5 = I_v$. Hence, system (1) becomes

$$\begin{cases} g_1 = \Lambda_h - (1 - u_1)(1 - \xi)\beta_h \frac{x_5}{x_4 + x_5} x_1 - \mu_h x_1, \\ g_2 = (1 - u_1)(1 - \xi)\beta_h \frac{x_5}{x_4 + x_5} x_1 - \alpha x_2 - \mu_h x_2, \\ g_3 = \alpha x_2 - \mu_d x_3 - u_2 x_3 - \mu_h x_3, \\ g_4 = \Lambda_v(x_4 + x_5) \left(1 - \frac{x_4 + x_5}{K}\right) - (1 - u_1)\beta_v \frac{x_3}{x_1 + x_2 + x_3} x_4 - \mu_v x_4 - \delta u_3 x_4, \\ g_5 = (1 - u_1)\beta_v \frac{x_3}{x_1 + x_2 + x_3} x_4 - \mu_v x_5 - \delta u_3 x_5. \end{cases}$$

According to the obtained basic reproduction number, when $\mathfrak{R}_0 = 1$, then

$$\beta_h^* = \frac{(\alpha + \mu_h)(u_2 + \mu_d + \mu_h)(u_3\delta + \mu_v)}{\alpha\beta_v(-1 + u_1)^2(1 - \xi)}.$$

So, the Jacobian matrix on disease-free equilibrium with the value $\beta_h = \beta_h^*$ will produce three eigenvalues, namely $\lambda_1 = -\mu_h < 0$, $\lambda_2 = -(\Lambda_v - (\mu_h + u_3\delta)) < 0$ if $\Lambda_v > \mu_h + u_3\delta$, $\lambda_3 = 0$ and the characteristic equation $\lambda^2 + b_1\lambda + b_2 = 0$ with b_1, b_2 positive. The roots of the quadratic equation are obtained from

$$\lambda_1, \lambda_2 = \frac{-b_1 \pm \sqrt{b_1^2 - 4b_2}}{2}.$$

If $b_1^2 - 4b_2 > 0$ then $\lambda_1, \lambda_2 < 0$. If $b_1^2 - 4b_2 = 0$ then $\lambda_1 = \lambda_2 = \frac{-b_1}{2} < 0$. And if $b_1^2 - 4b_2 < 0$ then

$$\lambda_1, \lambda_2 = \frac{-b_1}{2} \pm \frac{\sqrt{4b_2 - b_1^2}}{2}i.$$

So, λ_1 and λ_2 have negative real parts. Thus, the first assumption of the Castillo-Chaves and Song theorem is satisfied, there is one zero eigenvalue, while the other eigenvalues have negative real parts.

In addition, the zero eigenvalue is related to the matrix J_{DFE} , which has a right eigenvector and a left eigenvector. The right eigenvector corresponding to the zero eigenvalue is obtained as follows.

$$v = \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \end{pmatrix} = \begin{pmatrix} \frac{\Lambda_h \Lambda_v (-1 + u_1) \beta_h (1 - \xi)}{\mu_h^2 K (\Lambda_v - (u_3\delta + \mu_v))} v_5 \\ -\frac{\Lambda_h \Lambda_v (-1 + u_1) \beta_h (1 - \xi)}{\mu_h K (\alpha + \mu_h) (\Lambda_v - (u_3\delta + \mu_v))} v_5 \\ \frac{\alpha}{u_2 + \mu_d + \mu_h} v_2 \\ \frac{K \mu_h (-1 + u_1) \beta_v}{\Lambda_h \Lambda_v} v_3 - \frac{\Lambda_v - 2(u_3\delta + \mu_v)}{\Lambda_v - (u_3\delta + \mu_v)} v_5 \\ v_5 \end{pmatrix}.$$

Since all parameters are positive and assume $0 < u_1, \xi < 1$, $\Lambda_v > \mu_v + u_3\delta$, $\Lambda_v > 2(\mu_v + u_3\delta)$, and $v_5 > 0$ then $v_1, v_4 < 0$ and $v_2, v_3 > 0$. The left eigenvector corresponding to the zero eigenvalue is obtained as follows.

$$w = \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \end{pmatrix} = \begin{pmatrix} 0 \\ \frac{\alpha}{\alpha + \mu_h} w_3 \\ w_3 \\ 0 \\ -\frac{\Lambda_h \Lambda_v (-1 + u_1) \beta_h (1 - \xi)}{\mu_h K(\Lambda_v - (u_3 \delta + \mu_v))(u_3 \delta + \mu_v)} w_2 \end{pmatrix}.$$

Since all parameters are positive and assume $0 < u_1, \xi < 1, \Lambda_v > \mu_v + u_3 \delta$, and $w_3 < 0$ then $w_2, w_4 < 0$.

Next, under the conditions $0 < u_1, \xi < 1$ and $\Lambda_v > \mu_v + u_3 \delta$, the second-order partial derivatives evaluated at the disease-free equilibrium the critical value of the bifurcation parameter as follows:

$$\begin{aligned} \frac{\partial^2 g_1}{\partial x_1 x_5} &= \frac{\partial^2 g_1}{\partial x_5 x_1} = -\frac{\Lambda_v (-1 + u_1) \beta_h^* (1 - \xi)}{K(\Lambda_v - (u_3 \delta + \mu_v))} > 0, \\ \frac{\partial^2 g_2}{\partial x_1 x_5} &= \frac{\partial^2 g_2}{\partial x_5 x_1} = \frac{\Lambda_v (-1 + u_1) \beta_h^* (1 - \xi)}{K(\Lambda_v - (u_3 \delta + \mu_v))} < 0, \\ \frac{\partial^2 g_4}{\partial x_4 x_5} &= \frac{\partial^2 g_4}{\partial x_5 x_4} = -\frac{2v}{K} < 0, \quad \frac{\partial^2 g_4}{\partial x_3 x_4} = \frac{\partial^2 g_4}{\partial x_4 x_3} = -\frac{\mu_h (-1 + u_1) \beta_v}{\Lambda_h} > 0, \\ \frac{\partial^2 g_5}{\partial x_3 x_4} &= \frac{\partial^2 g_5}{\partial x_4 x_3} = \frac{\mu_h (-1 + u_1) \beta_v}{\Lambda_h} < 0, \quad \frac{\partial^2 g_1}{\partial x_5 \beta_h} = -\frac{\Lambda_h \Lambda_v (-1 + u_1) (1 - \xi)}{K(\Lambda_v - (u_3 \delta + \mu_v)) \mu_h} > 0, \\ \frac{\partial^2 g_2}{\partial x_5 \beta_h} &= \frac{\Lambda_h \Lambda_v (-1 + u_1) (1 - \xi)}{K(\Lambda_v - (u_3 \delta + \mu_v)) \mu_h} < 0. \end{aligned}$$

Next, the values of a and b obtained and can be written as

$$a = \sum_{k,i,j=1}^5 w_k v_i v_j \frac{\partial^2 g_k}{\partial x_i x_j} = -w_2 v_1 v_5 \frac{\Lambda_v (-1 + u_1) \beta_h^* (1 - \xi)}{K(\Lambda_v - (u_3 \delta + \mu_v))} + w_5 v_3 v_4 \frac{\mu_h (-1 + u_1) \beta_v}{\Lambda_h} < 0,$$

and

$$b = \sum_{k,i,j=1}^5 w_k v_i \frac{\partial^2 g_k}{\partial x_i \beta_h} = w_2 v_5 \frac{\Lambda_h \Lambda_v (-1 + u_1) (1 - \xi)}{K(\Lambda_v - (u_3 \delta + \mu_v)) \mu_h} > 0.$$

The obtained values of a and b correspond to one of the cases in the Castillo-Chavez and Song theorem. The bifurcation analysis confirms that the system exhibits a forward transcritical bifurcation at $\mathfrak{R}_0 = 1$. The endemic equilibrium becomes biologically feasible as $\mathfrak{R}_0$ increases

beyond unity. ■

3.5. Sensitivity Analysis

This section presents the sensitivity analysis for the SMD model. The Latin Hypercube Sampling-Partial Rank Correlation Coefficients (LHS-PRCC) with a sample size of 5000 are employed to identify the most influential parameter. First, the sensitivity analysis was conducted for $\mathcal{R}_0$, and the results are provided in Figure 2.

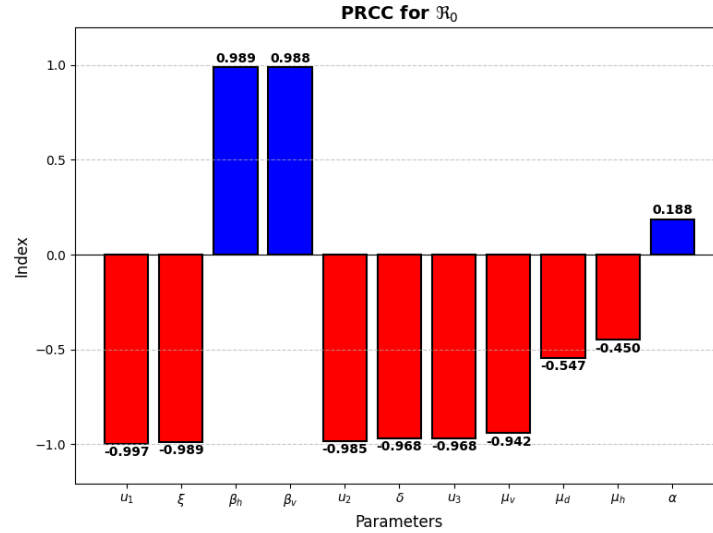


Figure 2. Sensitivity analysis on $\mathcal{R}_0$.

Figure 2 shows that both transmission rates, β_h and β_v , have the strongest positive correlation with $\mathcal{R}_0$. This justifies that cross-species transmission is highly sensitive to contact rates between host plants and vectors. Meanwhile, α , which is the latency transition, exhibits the weakest positive correlation. This indicates that faster latency progression increases transmission potential. Overall disease persistence is governed by vector-host encounter rates rather than the internal incubation period.

All control measures show a strong effect, statistically dominant in providing a suppression effect on $\mathcal{R}_0$. First, the prevention effort u_1 and resistance level ξ have the largest magnitude among all parameters. Suppressing the potential of the biting/feeding rate of vectors directly reduces transmission in both plant-to-vector and vector-to-plant. Next, bioinsecticide application intensity u_3 and its death rate effect δ significantly truncate vector lifespan. This prevents them from surviving the extrinsic incubation period required to transmit the SMV. Last, the Roguing

intensity u_2 serves as an essential third control, which substantially reduces the residence time of infectious plants.

Those findings indicate that an integrated pest management (IPM) system emphasizes conducting preventive efforts (u_1) first, alongside roguing (u_2) and spraying bioinsecticides (u_3), to achieve an impactful effect that forces $\mathfrak{R}_0 < 1$ and eradicates infection cases from cultivation. The combination of these control measures yields eight scenarios. Hence, to determine the most cost-effective strategy, we formulate an optimal control problem and will elaborate on it next.

3.6. Optimal Control Problem

In order to control disease transmission and reduce the risk of harvesting loss, we aim to suppress the number of both infected plants and infectious vectors. Procedures to suppress the infected population can be conducted both directly and indirectly, such as seed treatment, roguing, and bioinsecticide spraying. To achieve a cost-effective strategy, we constructed the dynamical model in system (1) by reformulating parameters u_1, u_2 , and u_3 for seed treatment, roguing, and spraying bioinsecticides into control functions, which involve t . Then, we obtained

$$\begin{aligned}
 \frac{dS_h(t)}{dt} &= \Lambda_h - (1 - u_1(t))(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} S_h(t) - \mu_h S_h(t), \\
 \frac{dE_h(t)}{dt} &= (1 - u_1(t))(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} S_h(t) - \alpha E_h(t) - \mu_h E_h(t), \\
 \frac{dI_h(t)}{dt} &= \alpha E_h(t) - \mu_d I_h(t) - u_2(t)I_h(t) - \mu_h I_h(t), \\
 \frac{dS_v(t)}{dt} &= \Lambda_v N_v(t) \left(1 - \frac{N_v(t)}{K}\right) - (1 - u_1(t))\beta_v \frac{I_h(t)}{N_h(t)} S_v(t) - \mu_v S_v(t) \\
 &\quad - \delta u_3(t)S_v(t), \\
 \frac{dI_v(t)}{dt} &= (1 - u_1(t))\beta_v \frac{I_h(t)}{N_h(t)} S_v(t) - \mu_v I_v(t) - \delta u_3(t)I_v(t),
 \end{aligned} \tag{7}$$

Mathematically, the goal of optimal control is to calculate an optimal rate of applying three controls by minimizing the objective function of

$$\mathcal{J}(u_1, u_2, u_3) = \int_0^{tf} (P_1 I_h(t) + P_2 I_v(t) + Q_1 u_1(t)^2 + Q_2 u_2(t)^2 + Q_3 u_3(t)^2) dt.$$

The first two parts of the integrand are the costs related to the high number of both infected

plants and vectors. At the same time, the last three parts represent the rate of conducting each intervention. Note that P_1, P_2, Q_1, Q_2 , and Q_3 are the weight constants that will balance each component in the function $J(u_1, u_2, u_3)$. Here, the optimal solution (u_1^*, u_2^*, u_3^*) is determined such that

$$J(u_1^*, u_2^*, u_3^*) = \min\{\max\{(u_1^*, u_2^*, u_3^*), 0\}, 1\}.$$

Pontryagin's Maximum Principle is used to govern the necessary conditions of the optimal control problem to investigate its existence. The Hamiltonian function for the optimal control system (7) can be written as

$$\begin{aligned} H(t) = & P_1 I_h(t) + P_2 I_v(t) + Q_1 u_1(t)^2 + Q_2 u_2(t)^2 + Q_3 u_3(t)^2 \\ & + \left(\Lambda_h - (1 - u_1(t))(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} S_h(t) - \mu_h S_h(t) \right) L_{S_h}(t) \\ & + \left((1 - u_1(t))(1 - \xi)\beta_h \frac{I_v(t)}{N_v(t)} S_h(t) - \alpha E_h(t) - \mu_h E_h(t) \right) L_{E_h}(t) \\ & + (\alpha E_h(t) - \mu_d I_h(t) - u_2(t) I_h(t) - \mu_h I_h(t)) L_{I_h}(t) \\ & + \left(\Lambda_v N_v(t) \left(1 - \frac{N_v(t)}{K} \right) - \left((1 - u_1(t))\beta_v \frac{I_h(t)}{N_h(t)} - \mu_v \right) S_v(t) - \delta u_3(t) S_v(t) \right) L_{S_v}(t) \\ & + \left((1 - u_1(t))\beta_v \frac{I_h(t)}{N_h(t)} S_v(t) - \mu_v I_v(t) - \delta u_3(t) I_v(t) \right) L_{I_v}(t), \end{aligned}$$

where $L_{S_h}(t), L_{E_h}(t), L_{I_h}(t), L_{S_v}(t)$, and $L_{I_v}(t)$ are the co-state variables in the optimal control problem. Next, it was noted that it must adhere to Pontryagin's Maximum Principle, which means that it must satisfy the necessary conditions for the optimal control problem as follows.

- State equation

$$S_h(t) \geq 0, \quad E_h(t) \geq 0, \quad I_h(t) \geq 0, \quad S_v(t) \geq 0, \quad I_v(t) \geq 0.$$

- Co-state variables

$$\begin{aligned} \frac{dL_{S_h}(t)}{dt} = & \left(-\frac{\beta_h(1 - \xi)(1 - u_1(t))I_v(t)}{I_v(t) + S_v(t)} - \mu_h \right) L_{S_h}(t) - \frac{\beta_h(1 - \xi)(1 - u_1(t))I_v(t)}{I_v(t) + S_v(t)} L_{E_h}(t) \\ & - \frac{\beta_v(1 - u_1(t))S_v(t)I_h(t)}{(S_h(t) + E_h(t) + I_h(t))^2} L_{S_v}(t) + \frac{\beta_v(1 - u_1(t))S_v(t)I_h(t)}{(S_h(t) + E_h(t) + I_h(t))^2} L_{I_v}(t), \end{aligned}$$

$$\begin{aligned}
\frac{dL_{E_h}(t)}{dt} &= -(-\alpha - \mu_h)L_{E_h}(t) - \alpha L_{I_h}(t) - \frac{\beta_v(1 - u_1(t))S_v(t)I_h(t)}{(S_h(t) + E_h(t) + I_h(t))^2}L_{S_v}(t) \\
&\quad + \frac{\beta_v(1 - u_1(t))S_v(t)I_h(t)}{(S_h(t) + E_h(t) + I_h(t))^2}L_{I_v}(t), \\
\frac{dL_{I_h}(t)}{dt} &= -P_1 - (-\mu_d - \mu_h - u_2(t))L_{I_h}(t) \\
&\quad - \left(-\frac{\beta_v(1 - u_1(t))S_v(t)}{S_h(t) + E_h(t) + I_h(t)} + \frac{\beta_v(1 - u_1(t))S_v(t)I_h(t)}{(S_h(t) + E_h(t) + I_h(t))^2} \right) L_{S_v}(t) \\
&\quad - \left(\frac{\beta_v(1 - u_1(t))S_v(t)}{S_h(t) + E_h(t) + I_h(t)} - \frac{\beta_v(1 - u_1(t))S_v(t)I_h(t)}{(S_h(t) + E_h(t) + I_h(t))^2} \right) L_{I_v}(t), \\
\frac{dL_{S_v}(t)}{dt} &= -\frac{\beta_h(1 - \xi)(1 - u_1(t))S_h(t)I_v(t)}{(S_v(t) + I_v(t))^2}L_{S_h}(t) + \frac{\beta_h(1 - \xi)(1 - u_1(t))S_h(t)I_v(t)}{(S_v(t) + I_v(t))^2}L_{E_h}(t) \\
&\quad - \left(\Lambda_v \left(1 - \frac{S_v(t) + I_v(t)}{K} \right) - \frac{\Lambda_v(S_v(t) + I_v(t))}{K} - \frac{\beta_v(1 - u_1(t))I_h(t)}{S_h(t) + E_h(t) + I_h(t)} \right. \\
&\quad \left. - \delta u_3(t) - \mu_v \right) L_{S_v}(t) - \frac{\beta_v(1 - u_1(t))I_h(t)}{S_h(t) + I_h(t) + E_h(t)}L_{I_v}(t), \\
\frac{dL_{I_v}(t)}{dt} &= -P_2 - \left(-\frac{\beta_h(1 - \xi)(1 - u_1(t))S_h(t)}{S_v(t) + I_v(t)} + \frac{\beta_h(1 - \xi)(1 - u_1(t))S_h(t)I_v(t)}{(S_v(t) + I_v(t))^2} \right) L_{S_h}(t) \\
&\quad - \left(\frac{\beta_h(1 - \xi)(1 - u_1(t))S_h(t)}{S_v(t) + I_v(t)} - \frac{\beta_h(1 - \xi)(1 - u_1(t))S_h(t)I_v(t)}{(S_v(t) + I_v(t))^2} \right) L_{E_h}(t) \\
&\quad - \left(\Lambda_v \left(1 - \frac{S_v(t) + I_v(t)}{K} \right) - \frac{\Lambda_v(S_v(t) + I_v(t))}{K} \right) L_{S_v}(t) \\
&\quad - (-\delta u_3(t) - \mu_v)L_{I_v}(t).
\end{aligned}$$

- Stationary conditions

Stationary conditions of the problem (7) are $\partial H/\partial u_1 = 0, \partial H/\partial u_2 = 0$ and $\partial H/\partial u_3 = 0$. Since $0 \leq u_1^*, u_2^*, u_3^* \leq 1$, we get

$$\begin{aligned}
u_1^* &= \min \left\{ \max \left\{ 0, \left(\frac{\beta_h(1 - \xi)S_h(t)I_v(t)}{S_v(t) + I_v(t)} (L_{E_h}(t) - L_{S_h}(t)) \right. \right. \right. \\
&\quad \left. \left. \left. + \frac{\beta_v S_v(t)I_h(t)}{S_h(t) + E_h(t) + I_h(t)} (L_{I_v}(t) - L_{S_v}(t)) \right) / 2Q_1 \right\}, 1 \right\},
\end{aligned}$$

$$u_2^* = \min \left\{ \max \left\{ 0, \frac{I_h(t)}{2Q_2} L_{I_h}(t) \right\}, 1 \right\},$$

$$u_3^* = \min \left\{ \max \left\{ 0, \frac{\delta \left(S_v(t) L_{S_v}(t) + I_v(t) L_{I_v}(t) \right)}{2Q_3} \right\}, 1 \right\}.$$

Since $\partial^2 H / \partial u_1^2 = 2Q_1 > 0$, $\partial^2 H / \partial u_2^2 = 2Q_2 > 0$, and $\partial^2 H / \partial u_3^2 = 2Q_3 > 0$, satisfy the minimum criterion of the Pontryagin Maximum Principle, u_1^* , u_2^* and u_3^* are the optimal solution of control system (7).

4. NUMERICAL SIMULATION AND ITS DISCUSSION

Numerical simulations were performed to confirm the results of mathematical analysis and provide a dynamic projection of SMD transmission under the control strategies. The values of each parameter in Table 1 were used. First, we confirm Theorem 4 numerically as a bifurcation diagram in Subsection 4.1. Next, we discuss the findings of the optimal control problem numerically in Subsections 4.2 and 4.3.

4.1. Bifurcation

To further illustrate the bifurcation behaviour obtained analytically, a numerical bifurcation diagram is constructed by plotting the endemic infected population (I_h^*) against the basic reproduction number ($\mathcal{R}_0$). The resulting bifurcation diagram is presented in Figure 3.

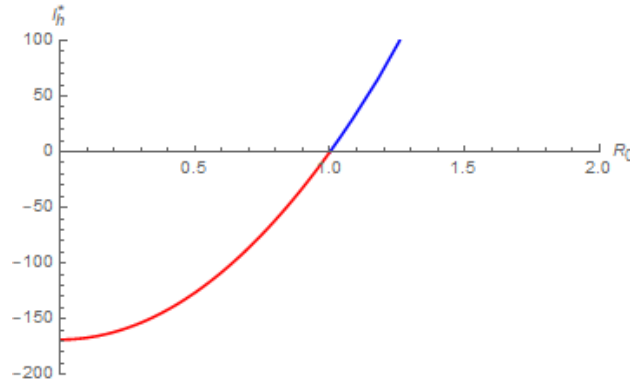


Figure 3. Bifurcation diagram of I_h^* versus $\mathcal{R}_0$.

Based on Figure 3, the bifurcation diagram illustrates the relationship between the basic reproduction number ($\mathcal{R}_0$) and the endemic infected population (I_h^*). The red branch represents the equilibrium solution for $\mathcal{R}_0 < 1$, where $I_h^* < 0$. Although this branch is mathematically

obtained from the endemic equilibrium equations, it is not biologically feasible because the infected population cannot take negative values. As $\mathfrak{R}_0$ increases toward unity, the value of I_h^* increases and approaches zero. At the critical value $\mathfrak{R}_0 = 1$, the endemic equilibrium satisfies $I_h^* = 0$, indicating the bifurcation solution. When $\mathfrak{R}_0$ increases beyond unity, the equilibrium branch moves into the positive region, as shown by the blue branch, with $I_h^* > 0$. Thus, the endemic equilibrium becomes biologically feasible for $\mathfrak{R}_0 > 1$. The exchange of the equilibrium branch through $I_h^* = 0$ at $\mathfrak{R}_0 = 1$ is consistent with Theorem 4, confirming that the system exhibits a forward transcritical bifurcation.

The results of the sensitivity analysis indicate that u_1, u_2 , and u_3 are significant in controlling the SMD transmission and yield eight control scenarios. We defined each control scenario in Table 2.

Table 2. Control scenario.

Scenario	Preventive effort (u_1)	Roguing effort (u_2)	Spraying effort (u_3)
I	0	0	0
II	u_1^*	0	0
III	0	u_2^*	0
IV	0	0	u_3^*
V	u_1^*	u_2^*	0
VI	u_1^*	0	u_3^*
VII	0	u_2^*	u_3^*
VIII	u_1^*	u_2^*	u_3^*

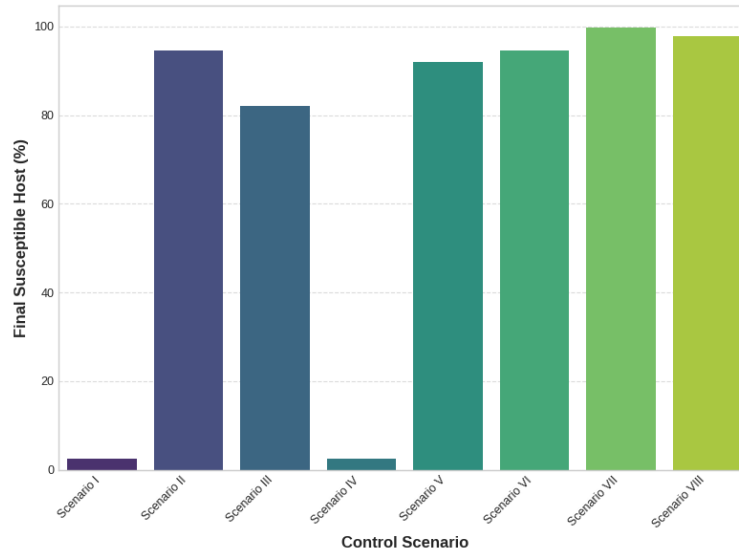
To get straight to the insights from the results, we provide the levels of both susceptible plants and vectors during the harvesting period, as well as the cost control, in a bar chart in Subsection 4.2.

4.2. Numerical Results of Optimal Control

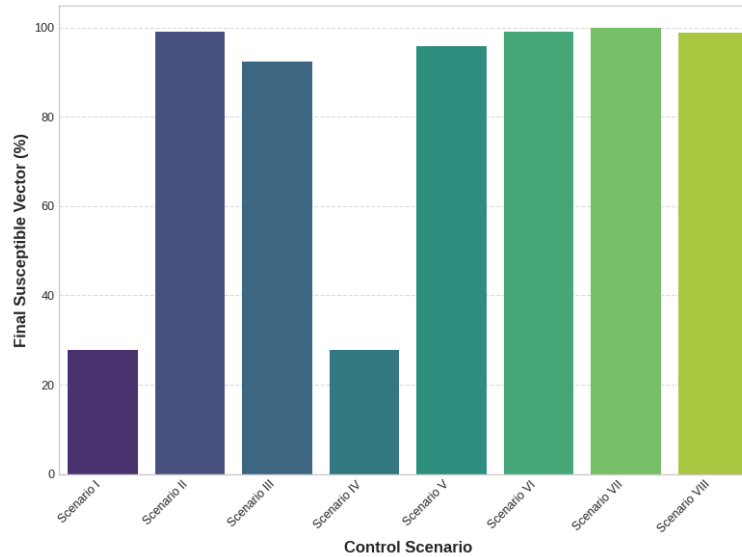
To evaluate the practical and economic viability of each plausible control scenario, comprehensive numerical simulations across eight control scenarios were conducted over the complete soybean cultivation period until the harvesting stage. In our computational workflow, the cost-effectiveness of each scenario is quantitatively assessed using two primary performance measures. First, we provide the state of both susceptible plants and susceptible vectors, which are aimed to be maximized when entering the harvesting period. The results are shown in Figure 4 as

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a bar chart to ease the comparison of each scenario.



(a)



(b)

Figure 4. Percentage of susceptible (a) plants and (b) vectors in harvesting period.

As shown in Figure 4(a) and (b), the final condition of both plants and vectors demonstrates the decisive role of the multi-tiered control scenario. Under Scenario I, no control is implemented to reduce the risk of disease transmission, leading to catastrophic plant collapse, where only around 2.5% of plants are harvestable, and 28% of vectors remain susceptible. This result is similar to the condition under Scenario IV, which is the worst scenario under a single control scenario compared

to Scenarios II and III. Meanwhile, Scenario VII emerges as the most effective scenario compared to the two other double-control scenarios and is also better than Scenario VIII. Scenarios VII and VIII provide near-complete field protection, preserving around 98% and 100% of susceptible plants and keeping the susceptible vectors around 99% and 100%. Furthermore, the intermediate partial scenarios, which are numbers II, III, V, and VI, yield substantial susceptibility for S_h and S_v around 82% to 95% and 92% to 99%. Yet, they leave residual infection reservoirs in the field that can threaten the next cultivation cycle. Therefore, Scenarios VII and VIII are considered the two most effective scenarios in controlling SMD transmission and yield the maximum harvestable plants.

The cumulative objective function (J) of each scenario is calculated to balance the cost of control implementation with the economic penalties induced by plant loss and disease prevalence. The economic perspective resulting from this simulation is shown in Figure 5.

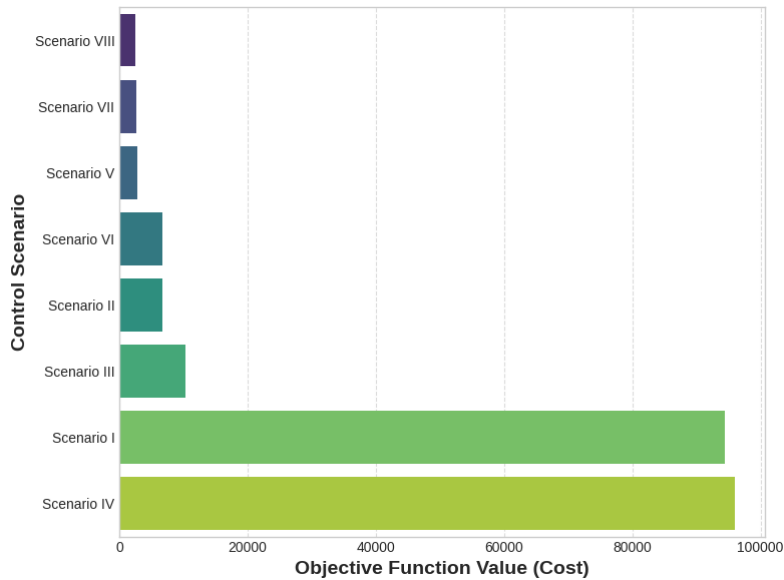


Figure 5. Cumulative objective function of each scenario.

Based on economic evaluation, the cumulative objective function value reveals a notable inverse relationship between intervention effectiveness and cumulative cost. Scenarios I and IV emerge as the highest objective values ($J > 94,000$), which demonstrate that omitting control or relying on indirect plant interventions imposes immense economic burdens driven almost entirely

by the severe penalties associated with plant destruction. In contrast, Scenarios VII and VIII have the lowest cumulative objective values ($J \approx 2,500 - 2,800$), followed by Scenarios V, VI, and II. By employing synergistic controls at the early transmission phase, these scenarios rapidly suppress the possibility of SMD transmission. This significantly reduces the time to implement the control and minimizes overall economic losses over the cultivation timeline, yielding a lower objective function value.

An overview of the results shown in Figures 4 and 5 and their interpretation raises an interesting issue. Figure 4 shows that Scenario VII yields a better condition, with more harvestable plants, than Scenario VIII. Meanwhile, Scenario VIII is more efficient than VII. We study this by conducting a numerical sensitivity analysis based on the seed resistance level, as highlighted in this study. The analysis will provide a number as a threshold to determine whether Scenario VII or VIII should be implemented according to the seed variety. This is elaborated on in Subsection 4.3.

4.3. Investigating the Threshold Number of Seed Quality

To determine the practical trade-offs between the formulated intervention scenarios under variable conditions, a sensitivity analysis of the objective function value was conducted across varying levels of seed quality (ξ). The results are shown in Figure 6.

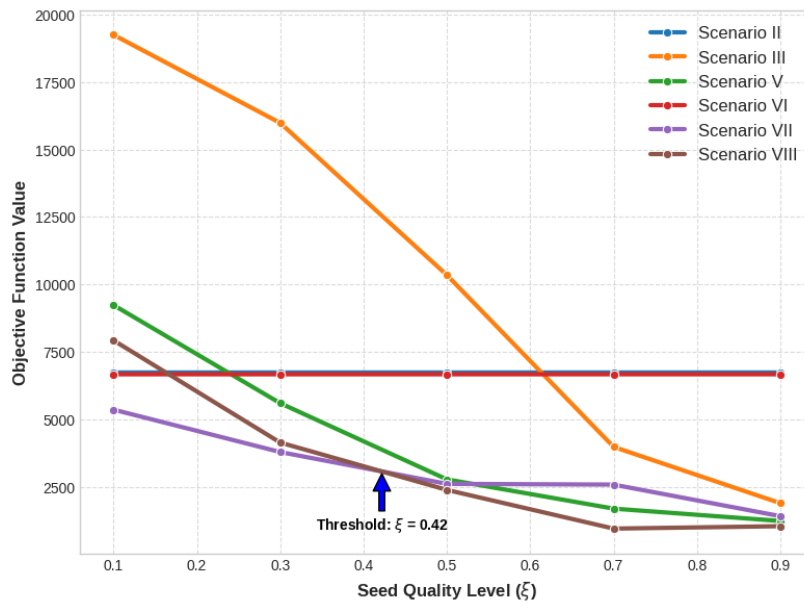


Figure 6. Sensitivity analysis of the objective function due to the seed variety.

Figure 6 shows that the baseline quality of the planting material strongly governs the economic viability of each control scenario. Control configurations that lack coupling with resistance-level enhancements (such as Scenarios II and VI) exhibit constant objective costs across all values of ξ . Yet, the strategies incorporating adaptive host-level mechanisms (such as Scenarios III, V, VII, and VIII) indicate cost reductions by using better seed quality.

A critical number of phenomena, called a threshold, emerges at $\xi \approx 0.42$, delineating the optimal operational boundary between Scenario VII and VIII. For low-quality seed ($\xi < 0.42$), Scenario VII has the lowest objective function value, indicating that under poor seed resistance, the control structure of this scenario is more cost-effective in mitigating harvesting losses without incurring excessive implementation costs. In contrast, once we use a seed such that the resistance level surpasses the threshold ($\xi > 0.42$), Scenario VIII becomes the decisively superior strategy, generating the global minimum objective function value across the entire parameter range of ξ , which drops to its lowest around $\xi \approx 0.7$.

These findings provide guidance for integrated pest management and agricultural policy. When farmers are only able to access low-quality or uncertified seed varieties ($\xi < 0.42$), they should prioritize the deployment of Scenario VII, which combines roguing and spraying bioinsecticides, to prevent cost escalation. In contrast, if they use high-quality or certified seed varieties ($\xi > 0.42$), Scenario VIII, which involves prevention, roguing, and spraying bioinsecticides, yields a maximum economic return by capitalizing on high baseline plant resilience to achieve complete epidemiological suppression while the cumulative operational cost is lower.

5. CONCLUSION

In this study, an SMD transmission model was formulated and analyzed to investigate its long-term behavior and evaluate the efficacy of synergistic control strategies. Theoretical analysis using dynamical systems theory established that the model solution will always be non-negative and bounded within the biologically feasible region. By using qualitative dynamical analysis, the basic reproduction number ($\mathcal{R}_0$) was determined as the governing parameter dictating the stability of

both disease-free and endemic equilibria, as well as determining the emergence of bifurcations.

Global sensitivity analysis using LHS-PRCC methods identifies the decisive drivers of disease transmission. Both transmission factors, β_h and β_v , exhibited the strongest positive influence on $\mathcal{R}_0$, highlighting the critical role of plant-vector contact rates. In contrast, intervention parameters show overwhelming negative correlations with $\mathcal{R}_0$, while the level of seed quality (ξ) and preventive effort (u_1) have the highest reduction magnitude, followed by spraying bioinsecticide (u_3) and its effective death rate (δ), as well as roguing effort (u_2).

Numerical simulations across eight different control configurations comprehensively indicated the decisive superiority of integrated pest management (IPM). The baseline scenario with no control and inadequate single-target interventions resulted in near-total crop loss, preserving only around 2.5% of harvestable plants and incurring prohibitive economic penalties, which is $J > 94,000$. Conversely, multi-tiered intervention scenarios, as demonstrated by Scenarios VII and VIII, yield near-complete epidemiological protection. These scenarios maintain around 98% to 100% harvestable plants and prevent the formation of an endemic vector reservoir, as well as achieving the lowest cumulative objective cost, which is $J \approx 2,500 - 2,800$.

A key practical insight from the numerical sensitivity analysis is the identification of a critical seed quality threshold. The threshold of ξ indicates the optimal deployment between Scenarios VII and VIII. For low-quality or uncertified seed varieties ($\xi < 0.42$), Scenario VII arises as the most viable choice economically, effective in buffering the high baseline susceptibility without extra intervention cost. Once seed quality crosses the threshold ($\xi \geq 0.42$), Scenario VIII yields the globally optimal cost-effective strategy, capitalizing on high innate plant resistance to eliminate the infection using a minimum cost of control implementation.

Collectively, these findings are supported by clear mathematical validation and provide actionable guidance for sustainable crop protection. Rather than relying on single-target reactive intervention, farmers and agricultural authorities are pushed to adopt integrated control that combines certified disease-resistant seed, mechanical plant-vector contact barriers, roguing, and bioinsecticide spraying. Future research may extend this study by incorporating seasonal vector

reproduction or stochastic environmental effects to refine the field-level decision support system further.

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CONFLICT OF INTERESTS

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

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