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Optimal Control of Tungro Disease Spread by Considering Growth Phase and Roguing Control

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Abstract

Tungro disease is a serious threat to rice cultivation, caused by a viral infection and spread by green leafhoppers. This study developed a mathematical model to assess the spread of tungro disease, taking into account plant growth phases and control factors, such as roguing. The model is divided into two subpopulations: plants and vectors. Dynamic analysis reveals the existence of two equilibrium points. The non-endemic equilibrium point is stable if $R_0 < 1$; conversely, the endemic point is stable if $R_0 > 1$. Sensitivity analysis using the PRCC method identified that the infectivity level and roguing rate are the most influential parameters on R_0. An optimal control approach was employed to determine the optimal control strategy, considering both the intensity of roguing and vector control. Pontryagin's Maximum Principle was used to obtain optimality conditions. Simulation results showed that roguing applied during the vegetative phase significantly reduced the number of infected plants and the intensity of disease spread. These findings demonstrate that integrating dynamic analysis, sensitivity analysis, and optimal control can provide an effective and efficient strategy for controlling tungro disease.

Keywords: Mathematical models; tungro; roguing; dynamic analysis; sensitivity analysis; optimal control theory.

1 Introduction

Rice (*Oryza sativa* L.) is a significant food commodity that plays a strategic role in the Indonesian economy, particularly as a provider of employment in the agricultural sector. Furthermore, this sector is also a significant source of income for rural communities, contributing to national food security [1]. Rice, as the main crop of the rice plant, is not only widely consumed but also plays

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a vital role in driving socio-economic growth and is a key component in realizing sustainable agricultural development and optimal utilization of natural resources [2].

However, rice productivity in Indonesia is often hampered by various problems, one of which is the attack of plant diseases caused by viruses. One of the most detrimental viral diseases is tungro disease. This disease poses a significant threat to rice production, particularly in South and Southeast Asia [3], [4]. Tungro disease is caused by infection with two types of viruses, namely *Rice Tungro Bacilliform Virus* (RTBV) and *Rice Tungro Spherical Virus* (RTSV), which are transmitted semi-persistently by the insect vector, the green leafhopper (*Nephotettix virescens*) [5]. Infected rice plants typically exhibit symptoms of yellowing leaves, stunted growth, and reduced yields, ultimately resulting in significantly reduced crop yields **pratisno2024detection**.

Various approaches have been developed to understand and control the spread of tungro disease. Amelia et al. [1] studied the characteristics of the virus that causes tungro and explored control strategies, including roguing and the use of pesticides. Anggriani et al. [6] proposed a mathematical optimization approach for a roguing strategy, considering both cost and control effectiveness. Suandi et al. [7] integrated roguing strategies with delayed replanting in analyzing disease spread. Suryaningrat et al. [8] added predator-prey interaction factors to disease spread modeling, while Chikore et al. [9] applied the SEIR model to model interplant transmission via vectors. Pratisno et al. [10] studied disease distribution by considering agronomic aspects, vector populations, and viral genetic variation.

In addition to dispersion modeling, several studies also focus on sensitivity analysis to identify key parameters in system dynamics. Yang and Liu [11] used the Sobol Global Sensitivity Analysis method, while Resmawan and Yahya [12] applied a partial derivative-based local sensitivity method. Anggriani et al. [13] and Ndairou et al. [14] combined Partial Rank Correlation Coefficient (PRCC) and Latin Hypercube Sampling (LHS) in their sensitivity analyses.

Additionally, optimal control theory has also begun to be widely applied in mathematical model-based disease control. Pontryagin's Maximum Principle was used by Madubueze et al. [15] and Adewale et al. [16] in developing control strategies for nonlinear differential equation systems. Amelia et al. [17] used Pontryagin's Minimum Principle and the forward-backward sweep method to solve control models. Barbolosi et al. [18] and Huo et al. [19] applied the optimal control approach to pharmacological and cancer therapy problems, demonstrating the broad potential of this method in dynamic systems. The optimal analysis conducted by Amelia et al. [20] employed Pontryagin's Minimum Principle, considering two variables: pesticide application and natural enemy conservation through refugia.

Although numerous studies have been conducted, no study has specifically developed a mathematical model for tungro disease spread that considers plant growth phases, namely the vegetative and generative phases, and utilizes roguing as the primary control strategy. However, the plant growth phase can influence spread dynamics and the effectiveness of interventions.

Therefore, this study aims to develop and analyze a mathematical model for the spread of tungro disease that explicitly accounts for rice plant growth phases and integrates a roguing-based control strategy. A system dynamics analysis is conducted to examine the model's behavior over time, followed by a sensitivity analysis to identify the key parameters that most influence disease spread. These analyses provide the foundation for formulating and implementing an optimal control strategy to achieve more effective and efficient disease management.

2 Mathematical Models

The model developed in this study is a modification of the model proposed by Maryati et al. [21]. The development focused on implementing a roguing strategy as a control measure to suppress the spread of tungro disease. In this model, the roguing parameter is denoted by the symbol ρ, with the assumption $0 \leq \rho \leq 1$ that represents the proportion of infected plants that are immediately removed from the system. Unlike previous studies by Amelia et al. [1], which

modeled the spread of tungro disease based on general viral characteristics, the model in this study explicitly considers plant growth phases in population dynamics. Furthermore, this model also develops optimal control strategies designed to evaluate the effectiveness of interventions based on each of these growth phases. The model is visually illustrated in Fig. 1, accompanied by a description of each parameter and variable in Table 1.

Table 1: Description of Parameters and Variables [21]

Variables/ Parameters	**Description**	**Value** $R_0 < 1$	**Value** $R_0 > 1$
S_v	Rice plants are susceptible (vegetative phase)	500	500
I_v	Rice plants infected (vegetative phase)	100	100
S_g	Rice plants are susceptible (generative phase)	300	300
I_g	Rice plants infected (generative phase)	100	100
S_{wh}	Susceptible vectors	450	450
I_{wh}	Infected vector	150	150
Λ	Recrutment rate of rice plant	100	100
μ_p	Rice plant death rate	0.3	0.3
α	The growth rate of plants from I_v to I_g	0.7	0.7
ρ	Roguing level	0.1	0.1
β_1	Rate of I_v	0.0005	0.0005
β_2	Rate of I_g	0.0005	0.0005
ω	Recrutment rate of S_v	100	100
γ_1	The rate of vector infection when taking food from I_v	0.007	0.025
γ_2	Vector infection when taking food from I_g	0.005	0.002
μ_t	Vector mortality rate	0.7	0.2
A_1	Cost coefficient for I_v	1	1
A_2	Cost coefficient for I_g	1	1
C	Cost coefficient for roguing	1	1

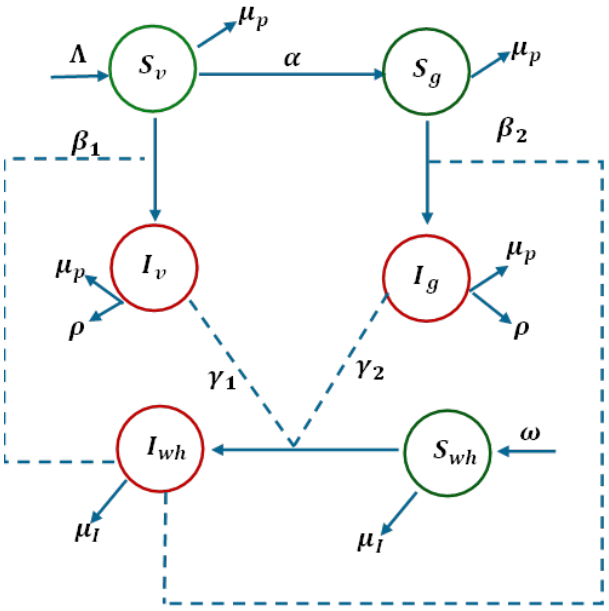


Figure 1: Schematic diagram of the spread of tungro disease

From Fig. 1 and Table 1, the resulting development model is stated in Eq. 1 to Eq. 6.

$$\frac{dS_v}{dt} = \Lambda - \alpha S_v - \beta_1 S_v I_{wh} - \mu_p S_v \tag{1}$$

$$\frac{dI_v}{dt} = \beta_1 S_v I_{wh} - \mu_p I_v - \rho I_v \tag{2}$$

$$\frac{dS_g}{dt} = \alpha S_v - \beta_2 S_g I_{wh} - \mu_p S_g \tag{3}$$

$$\frac{dI_g}{dt} = \beta_2 S_g I_{wh} - \mu_p I_g - \rho I_g \tag{4}$$

$$\frac{dS_{wh}}{dt} = \omega - \gamma_1 I_v S_{wh} - \gamma_2 I_g S_{wh} - \mu_I S_{wh} \tag{5}$$

$$\frac{dI_{wh}}{dt} = \gamma_1 I_v S_{wh} + \gamma_2 I_g S_{wh} - \mu_I I_{wh} \tag{6}$$

With $S_v, I_v, S_g, I_g, S_{wh}, I_{wh} \geq 0$.

3 Results and Discussion

This section presents an analysis of the dynamics of tungro disease spread, starting from the identification of the equilibrium point and the calculation of the basic reproduction number (R_0), as well as the analysis of endemic and non-endemic stability, to the sensitivity of key parameters that form the basis for designing optimal control strategies. Next, numerical simulations are used to illustrate population dynamics and the implementation of control strategies, thereby linking theoretical findings to practical applications in a logical, structured manner.

3.1 Result

3.1.1 Dynamic Analysis

Theorem 1
If the tungro disease spread model (Eq. 1 to Eq. 6) with non-negative initial conditions

$$S_v(0), \quad I_v(0), \quad S_g(0), \quad I_g(0), \quad S_{wh}(0), \quad I_{wh}(0) \geq 0$$

has a solution, then the solution obtained is unique, namely:

$$L(t) = \begin{pmatrix} S_v(t) \\ I_v(t) \\ S_g(t) \\ I_g(t) \\ S_{wh}(t) \\ I_{wh}(t) \end{pmatrix} \in \Omega_L \quad \text{for} \quad t > 0.$$

Based on [22], the existence and uniqueness of the solution of the system of Eq. 1 to Eq. 6 are investigated in the region $[0, \infty) \times \Omega_L$, where

$$\Omega_L = \left\{ (S_v, I_v, S_g, I_g, S_{wh}, I_{wh})^T \in \mathbb{R}_+^6 \mid \max\{|S_v|, |I_v|, |S_g|, |I_g|, |S_{wh}|, |I_{wh}|\} \leq L \right\}.$$

For L sufficiently large.

Proof:

Let

$$L = (S_v, I_v, S_g, I_g, S_{wh}, I_{wh})^T, \quad \bar{L} = (\bar{S}_v, \bar{I}_v, \bar{S}_g, \bar{I}_g, \bar{S}_{wh}, \bar{I}_{wh})^T$$

and define

$$F(L) = \left(\frac{dS_v}{dt}, \frac{dI_v}{dt}, \frac{dS_g}{dt}, \frac{dI_g}{dt}, \frac{dS_{wh}}{dt}, \frac{dI_{wh}}{dt}\right)^T$$

For any $L, \bar{L} \in \Omega_L$, we obtain:

$$\begin{aligned}\|F(L) - F(\bar{L})\| &\leq M(|S_v - \bar{S}_v| + |I_v - \bar{I}_v| + |S_g - \bar{S}_g| + |I_g - \bar{I}_g| + |S_{wh} - \bar{S}_{wh}| + |I_{wh} - \bar{I}_{wh}|) \\ &= M\|L - \bar{L}\|,\end{aligned}$$

where

$$M_1 = \mu_p, \quad M_2 = \mu_p + \rho, \quad M_3 = \mu_I, \quad \text{and} \quad M = \max\{M_1, M_2, M_3\}.$$

Biologically, the fulfillment of the Lipschitz condition and the existence of a unique solution indicate that the model is stable and can evolve predictably and consistently, without producing unrealistic or deviant behavior. ■

Theorem 2

If every initial condition $S_v(0), I_v(0), S_g(0), I_g(0), S_{wh}(0), I_{wh}(0) \geq 0$, then the solution of Eq. 1 to Eq. 6 is non-negative for all $t > 0$.

Proof:

It is shown that all solutions of equations Eq. 1 to Eq. 6 are non-negative for all $t > 0$, given the initial conditions $S_v(0), I_v(0), S_g(0), I_g(0), S_{wh}(0), I_{wh}(0) \geq 0$. Obtained:

$$\frac{dS_v}{dt} = \Lambda - \alpha S_v - \beta_1 S_v I_{wh} - \mu_p S_v,$$

$$\frac{dS_v}{dt} = -(\alpha + \beta_1 I_{wh} + \mu_p) S_v + \Lambda.$$

$$S_v(0) = \exp\left(-\int_0^t (\alpha + \beta_1 I_{wh}(s) + \mu_p) ds\right) \geq 0.$$

By using the same method, it follows that $S_v(0)$, $I_v(0)$, $S_g(0)$, $I_g(0)$, $S_{wh}(0)$, $I_{wh}(0) \geq 0$ for each $t > 0$. Thus, it is proven that Eq. 1 to Eq. 6 have non-negative solutions. ■

Biologically, these results indicate that the model's solutions will consistently remain non-negative over time. This implies that the model will not produce negative population numbers, thereby ensuring that the system remains biologically meaningful and realistic.

Theorem 3

If Eq. 1 to Eq. 6 have solutions, then the obtained solutions are finite for all $t \in [0, t_0]$.

Proof:

From Eq. 1 to Eq. 6, we obtain: $N = N_P + N_V$, $N_P = S_v + I_v + S_g + I_g$, and $N_V = S_{wh} + I_{wh}$. By assuming that $\mu_p = \mu_I = \mu_p + \rho = \mu$, we have $\frac{dN}{dt} = \Lambda + \omega - \mu N$.By using the variable separation method, we obtain $0 \leq N(t) \leq \frac{\Lambda+\omega}{\mu}$. Thus, it is proven that Eq. 1 to Eq. 6 are bounded for all $t \in [0, t_0]$. ■

Biologically, this model demonstrates realism by showing that plant populations have a reasonable and controlled upper limit. In other words, the model reflects natural conditions in which plant populations do not experience uncontrolled growth or sudden extinction within a given time span.

3.1.2 Non-Endemic Equilibrium Point

The non-endemic equilibrium point is obtained by settingEq. 1 to Eq. 6 equal to zero [23]. Then, by substituting the non-endemic condition $I_v = I_g = I_{wh} = 0$ into equations Eq. 1 to Eq. 6, the non-endemic equilibrium point is obtained as written in Eq. 7.

$$E_0 = \{S_v, I_v, S_g, I_g, S_{wh}, I_{wh}\} = \left\{\frac{\Lambda}{\mu_p + \alpha}, 0, \frac{\alpha\Lambda}{\mu_p(\mu_p + \alpha)}, 0, \frac{\omega}{\mu_I}, 0\right\}. \tag{7}$$

3.1.3 Basic Reproduction Number

The Basic Reproduction Number is defined as

$$R_0 = \rho(FV^{-1}),$$

where ρ is the spectral radius (dominant eigenvalue) of the matrix FV^{-1} [24], [25], [26].

F, V, and R_0 are obtained for the tungro disease model as in Eq. 8:

$$F = \begin{bmatrix} \beta_1 S_v I_{wh} \\ \beta_2 S_g I_{wh} \\ \gamma_1 I_v S_{wh} + \gamma_2 I_g S_{wh} \end{bmatrix}, \quad V = \begin{bmatrix} \mu_p I_v + I_v \rho \\ \mu_p I_g + I_g \rho \\ \mu_I I_{wh} \end{bmatrix}, R_0 = \sqrt{\frac{\Lambda\omega(\mu_p\beta_1\gamma_1 + \alpha\beta_2\gamma_2)}{\mu_p(\mu_p + \alpha)(\mu_p + \rho)\mu_I^2}}. \tag{8}$$

Here, F represents a matrix of new infection levels that appear in the compartment, and V represents the levels that leave the compartments. In this case, it represents the average number of new infections produced by a single tungro-infected plant in a fully susceptible rice population.

3.1.4 Stability Analysis

Theorem 4
The non-endemic equilibrium point in Eq. 7 is locally stable if $R_0 < 1$.

Proof:
The stability of the non-endemic equilibrium point for Eq. 7 is determined by the eigenvalues obtained from the characteristic Eq. 9 obtained from the Jacobian matrix.

$$\frac{1}{\mu_I\mu_p(\mu_p + \alpha)}(\mu_p + \lambda)(\mu_p + \lambda + \rho)(\mu_p + \alpha + \lambda)(\mu_I + \lambda)(a_0\lambda^2 + a_1\lambda + a_2) = 0, \tag{9}$$

with

$$\begin{aligned} a_0 &= 1 > 0, \\ a_1 &= \rho + \mu_I + \mu_p > 0, \\ a_2 &= \mu_p(\mu_p + \alpha)(\mu_p + \rho)\mu_I^2 - \Lambda\omega(\mu_p\beta_1\gamma_1 + \alpha\beta_2\gamma_2) \\ &= 1 - R_0^2 > 0. \end{aligned}$$

From Eq. 9, the eigenvalues are:

$$\begin{aligned} \lambda_1 &= -\mu_p \\ \lambda_2 &= -(\mu_p + \rho) \\ \lambda_3 &= -(\mu_p + \alpha) \\ \lambda_4 &= -\mu_I \\ \lambda_{5,6} &= \frac{-a_1 \pm \sqrt{a_1^2 - 4a_0a_2}}{2a_0}. \end{aligned}$$

Based on these eigenvalues, $\lambda_1, \ldots, \lambda_4$ are clearly negative. To ensure local stability, the eigenvalues λ_5 and λ_6 must also be negative. Using the Routh–Hurwitz criterion, the polynomial $a_0\lambda^2 + a_1\lambda + a_2 = 0$ has roots with negative real parts if all its coefficients have the same sign. This implies λ_5 and λ_6 are negative if $R_0 < 1$.

Because all eigenvalues are negative, it is proven that the non-endemic equilibrium point in Eq. 7 is locally stable if $R_0 < 1$. These results indicate that tungro disease will not spread in a plant population if $R_0 < 1$. ■

3.1.5 Existence of Endemic Equilibrium Points

Theorem 6
Eq. 1 to Eq. 6 have an endemic equilibrium point if $R_0 > 1$.

Proof:
The existence of an endemic equilibrium point is obtained by solving Eq. 1 to Eq. 5, yielding the point as shown in Eq. 10 to Eq. 14:

$$I_g^* = \frac{I_{wh}^* \beta_2 \alpha \Lambda}{(I_{wh}^* \beta_2 + \mu_p)(I_{wh}^* \beta_1 + \mu_p + \alpha)(\mu_p + \rho)}, \tag{10}$$

$$I_v^* = \frac{\beta_1 \Lambda I_{wh}^*}{(I_{wh}^* \beta_1 + \mu_p + \alpha)(\mu_p + \rho)}, \tag{11}$$

$$S_g^* = \frac{\alpha \Lambda}{(I_{wh}^* \beta_2 + \mu_p)(I_{wh}^* \beta_1 + \mu_p + \alpha)}, \tag{12}$$

$$S_v^* = \frac{\Lambda}{I_{wh}^* \beta_1 + \mu_p + \alpha}, \tag{13}$$

$$S_{wh}^* = \frac{\omega(I_{wh}^* \beta_2 + \mu_p)(I_{wh}^* \beta_1 + \mu_p + \alpha)(\mu_p + \rho)}{(I_{wh}^* \beta_2 + \mu_p)(I_{wh}^* \beta_1 + \mu_p + \alpha)(\mu_p + \rho)\mu_I + (\gamma_1 \beta_1 \mu_p + \beta_2 (I_{wh}^* \beta_1 \gamma_1 + \alpha \gamma_2)) I_{wh}^* \Lambda}. \tag{14}$$

Substituting Eq. 10 to Eq. 14 into Eq. 6, we obtain Eq. 15:

$$-\frac{I_{wh}^*(a_4 I_{wh}^{*2} + a_5 I_{wh}^* + a_6)}{(I_{wh}^* \beta_2 + \mu_p)(I_{wh}^* \beta_1 + \mu_p + \alpha)(\mu_p + \rho)\mu_I + I_{wh}^* \Lambda (I_{wh}^* \beta_1 \beta_2 \gamma_1 + \mu_p \beta_1 \gamma_1 + \alpha \beta_2 \gamma_2)} = 0, \tag{15}$$

where the coefficients are

$$\begin{aligned}
a_4 &= ((\mu_p + \rho)\mu_I + \Lambda\gamma_1)\beta_2 \mu_I \beta_1, \\
a_5 &= ((\alpha + \mu_p)\beta_2 + \beta_1 \mu_p)(\mu_p + \rho)\mu_I^2 + \Lambda(\mu_p \beta_1 \gamma_1 + \alpha \beta_2 \gamma_2)\mu_I - \Lambda \omega \beta_1 \beta_2 \gamma_1, \\
a_6 &= \mu_I^2 \mu_p (\alpha + \mu_p)(\mu_p + \rho) - \omega \Lambda (\mu_p \beta_1 \gamma_1 + \alpha \beta_2 \gamma_2).
\end{aligned}$$

Based on the Routh–Hurwitz criterion, the characteristic equation polynomial $(a_4 I_{wh}^2 + a_5 I_{wh} + a_6) = 0$, will have at least one positive root if one of the polynomial coefficients has a different sign. Therefore, it can be concluded that Eq. 11 has a positive root if $R_0 > 1$.

This indicates that when $R_0 > 1$, tungro disease has the potential to spread and persist within the plant population, thus allowing the existence of an endemic equilibrium point. ■

3.1.6 Sensitivity Analysis

Sensitivity analysis was conducted using the Latin Hypercube Sampling (LHS) method and the Partial Rank Correlation Coefficient (PRCC) method [27]. A total of 5.000 samples were used to determine the parameters that influence the basic reproduction number, as written in Eq. 8, where each parameter is assumed to have a value between 0 and 1. The results obtained are presented in Table 2.

Table 2: Sensitivity Analysis Results

Parameter	Sensitivity Index to R_0	Parameter	Sensitivity Index to R_0
μ_p	-0.288313328930483	β_2	0.00254836677353819
α	-0.643749923339776	γ_1	0.00245239481037207
ρ	0.000614852963961030	γ_2	0.00134398671948831
β_1	0.00218526675365787	μ_I	0.00161285354250586

The PRCC sensitivity analysis results, presented in Table 2, indicate that several parameters have a significant influence on the basic reproduction number R_0. Specifically, the parameter α exhibits a strong negative correlation with R_0, suggesting that increasing the value of α can reduce the potential for disease transmission. Furthermore, five other parameters, namely ρ, β_1, β_2, γ_1, γ_2, and μ_I, show a positive effect, where increasing their values contributes to a decrease in the R_0 value. Conversely, the parameters μ_p and α have a negative relationship, where increasing their values can actually lead to a reduction in R_0. Among all the parameters analyzed, only the parameter ρ (roguing rate) can be directly controlled in field practice. Therefore, this parameter was selected as the primary control variable in formulating the optimal control strategy.

3.1.7 Optimal Control

The objective of optimal control in this section is to minimize the costs incurred while controlling the population of infected plants and vectors; thus, the objective function of this optimal control model is as given in Eq. 16.

$$J(u) = \min \left\{ \int_{t_0}^{t_1} \left(A_1 I_v + A_2 I_g + C u^2 \right) \, dt \right\} \tag{16}$$

With constraint functions as in Eq. 17 to Eq. 22:

$$\frac{dS_v}{dt} = \Lambda - \alpha S_v - \beta_1 S_v I_{wh} - \mu_p S_v, \tag{17}$$

$$\frac{dI_v}{dt} = \beta_1 S_v I_{wh} - \mu_p I_v - u I_v, \tag{18}$$

$$\frac{dS_g}{dt} = \alpha S_v - \beta_2 S_g I_{wh} - \mu_p S_g, \tag{19}$$

$$\frac{dI_g}{dt} = \beta_2 S_g I_{wh} - \mu_p I_g - u I_g, \tag{20}$$

$$\frac{dS_{wh}}{dt} = \omega - \gamma_1 I_v S_{wh} - \gamma_2 I_g S_{wh} - \mu_I S_{wh}, \tag{21}$$

$$\frac{dI_{wh}}{dt} = \gamma_1 I_v S_{wh} + \gamma_2 I_g S_{wh} - \mu_I I_{wh}. \tag{22}$$

Boundary conditions:

$$t_0 < t < t_1, \quad 0 \le u(t) \le 1, \quad S_v(0), I_v(0), S_g(0), I_g(0), S_{wh}(0), I_{wh}(0) \ge 0$$

The optimal control theory method is used to solve the optimal control model using the Pontryagin minimum principle, where u is the optimal control [28] [29]. From the objective function and constraints in Eq. 16 to Eq. 22, the Hamiltonian function is obtained as in Eq. 23.

The Hamiltonian function H is given by:

$$H = A_1 \cdot I_v + A_2 \cdot I_g + C \cdot u^2 + \lambda_1 \frac{dS_v}{dt} + \lambda_2 \frac{dI_v}{dt} + \lambda_3 \frac{dS_g}{dt} + \lambda_4 \frac{dI_g}{dt} + \lambda_5 \frac{dS_{wh}}{dt} + \lambda_6 \frac{dI_{wh}}{dt}. \tag{23}$$

With λ_i for is $i = 1, \ldots, 6$, a co-state variable, then the Hamiltonian function must satisfy: co-state equations, adjoint equations, and stationary conditions.

Co-state Equation:

$$\begin{aligned}
\frac{\partial H}{\partial \lambda_1} &= \frac{dS_v}{dt} = \Lambda - \alpha S_v - \beta_1 S_v I_{wh} - \mu_p S_v, \\
\frac{\partial H}{\partial \lambda_2} &= \frac{dI_v}{dt} = \beta_1 S_v I_{wh} - \mu_p I_v - u I_v, \\
\frac{\partial H}{\partial \lambda_3} &= \frac{dS_g}{dt} = \alpha S_v - \beta_2 S_g I_{wh} - \mu_p S_g, \\
\frac{\partial H}{\partial \lambda_4} &= \frac{dI_g}{dt} = \beta_2 S_g I_{wh} - \mu_p I_g - u I_g, \\
\frac{\partial H}{\partial \lambda_5} &= \frac{dS_{wh}}{dt} = \omega - \gamma_1 I_v S_{wh} - \gamma_2 I_g S_{wh} - \mu_I S_{wh}, \\
\frac{\partial H}{\partial \lambda_6} &= \frac{dI_{wh}}{dt} = \gamma_1 I_v S_{wh} + \gamma_2 I_g S_{wh} - \mu_I I_{wh}.
\end{aligned}$$

Adjoint Equation:

$$\dot{\lambda}_1 = -\frac{\partial H}{\partial S_v} = -\lambda_1(-\beta_1 I_{wh} - \mu_p - \alpha) - \lambda_2 I_{wh}\beta_1 - \lambda_3\alpha,$$

$$\dot{\lambda}_2 = -\frac{\partial H}{\partial I_v} = -\lambda_2(-\mu_p - u) + \lambda_5\gamma_1 S_{wh} - \lambda_6\gamma_1 S_{wh},$$

$$\dot{\lambda}_3 = -\frac{\partial H}{\partial S_g} = -\lambda_3(-\beta_2 I_{wh} - \mu_p) - \lambda_4\beta_2 I_{wh},$$

$$\dot{\lambda}_4 = -\frac{\partial H}{\partial I_g} = -A_2 - \lambda_4(-\mu_p - u) + \lambda_5\gamma_2 S_{wh} - \lambda_6\gamma_2 S_{wh},$$

$$\dot{\lambda}_5 = -\frac{\partial H}{\partial S_{wh}} = -\lambda_5(-\gamma_2 I_g - \gamma_1 I_v - \mu_I) - \lambda_6(-\gamma_2 I_g + \gamma_1 I_v),$$

$$\dot{\lambda}_6 = -\frac{\partial H}{\partial I_{wh}} = \lambda_1 S_v\beta_1 - \lambda_2 S_v\beta_1 + \lambda_3\beta_2 S_g - \lambda_4\beta_2 S_g + \lambda_6\mu_I.$$

Stationary conditions:

$$u^* = \frac{\lambda_4 I_g + \lambda_2 I_v}{2C}.$$

Control function with bounds:

$$u^* = \max\left\{\min\left[\frac{\lambda_4 I_g + \lambda_2 I_v}{2C}, 1\right], 0\right\}.$$

3.2 Discussion

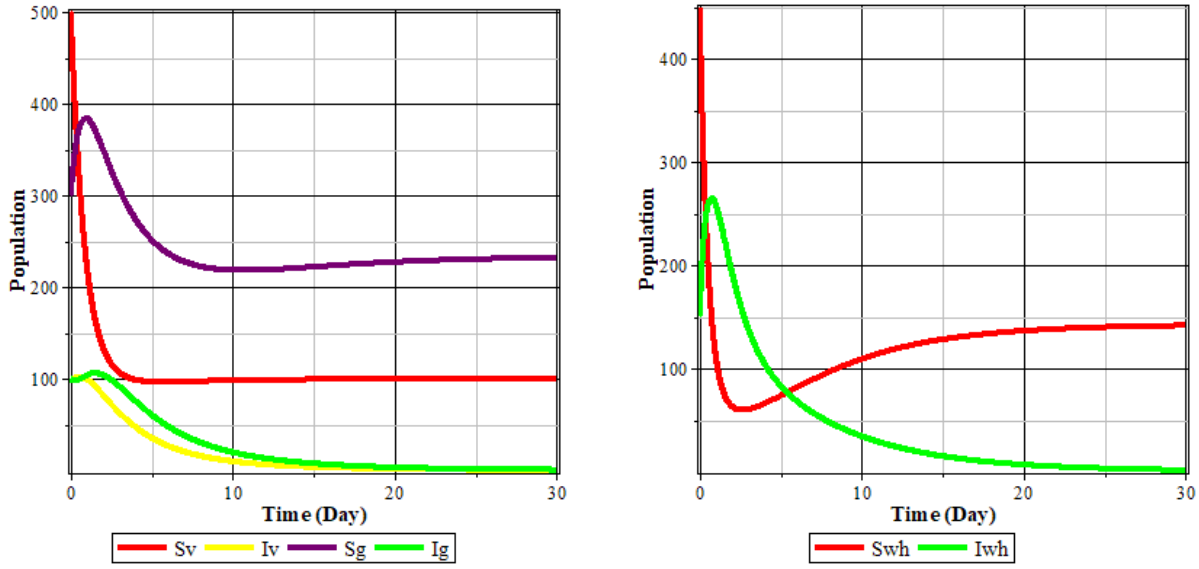
The simulations presented in this section, both for population dynamics and optimal control, are performed using the parameters explicitly described in Table 1.

3.2.1 Population Dynamics

Based on Fig. 2, the population dynamics of rice plants during the vegetative and generative phases indicate that tungro disease infection did not develop into an endemic state. This is evident from the significant downward trend in the number of infected plants in both phases, which approaches zero by the end of the observation period (day 30). Meanwhile, the population of susceptible plants reached a stable state, indicating that the infection would not continue to spread. This phenomenon is consistent with the basic reproduction number ($R_0 < 1$), which

epidemiologically means that each infected plant transmits the disease to, on average, fewer than one healthy plant. Therefore, the infection cannot persist and tends to disappear over time.

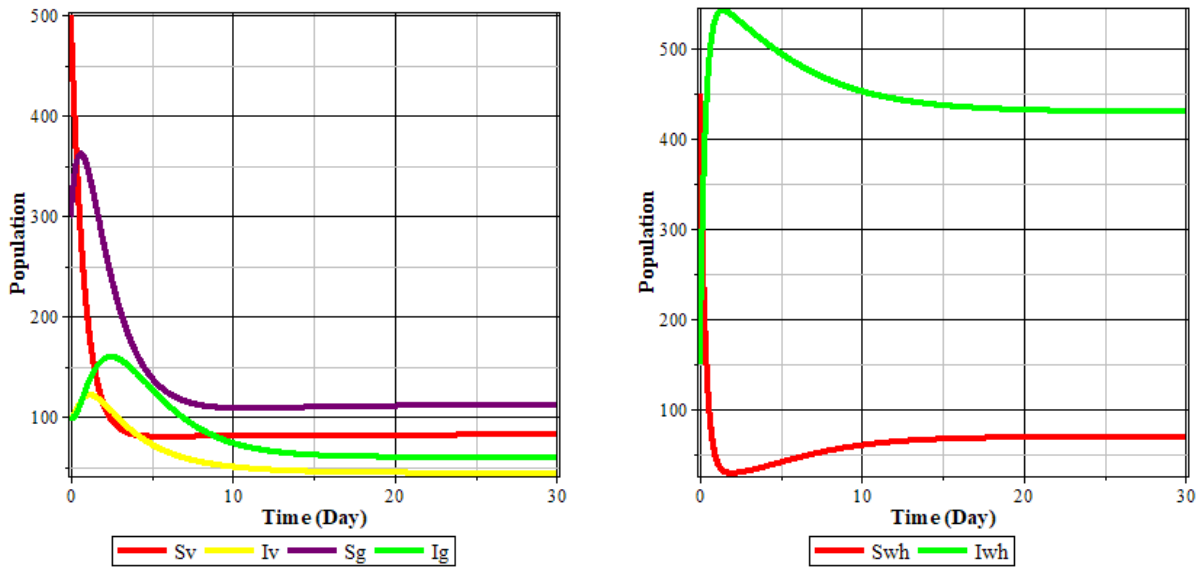
Analysis of the insect vector population also shows a similar pattern. Initially, the number of infected insects increases and reaches a peak, but then decreases significantly to approach zero. In contrast, the population of healthy insects, which had previously declined, gradually increases again. This indicates that infection in the vector population is not sustainable and the outbreak does not last long, which is consistent with the condition $R_0 < 1$.



(a) Rice Plant (b) Vector

Figure 2: Population dynamics when $R_0 < 1$

Conversely, in Fig. 3, the population dynamics of rice plants indicate that the infection persisted throughout the observation period. The number of infected plants remained relatively high, with no significant decline, and the population of infected insects persisted for 30 days. This indicates that the disease can maintain its chain of transmission, creating an endemic state consistent with the condition $R_0 > 1$.



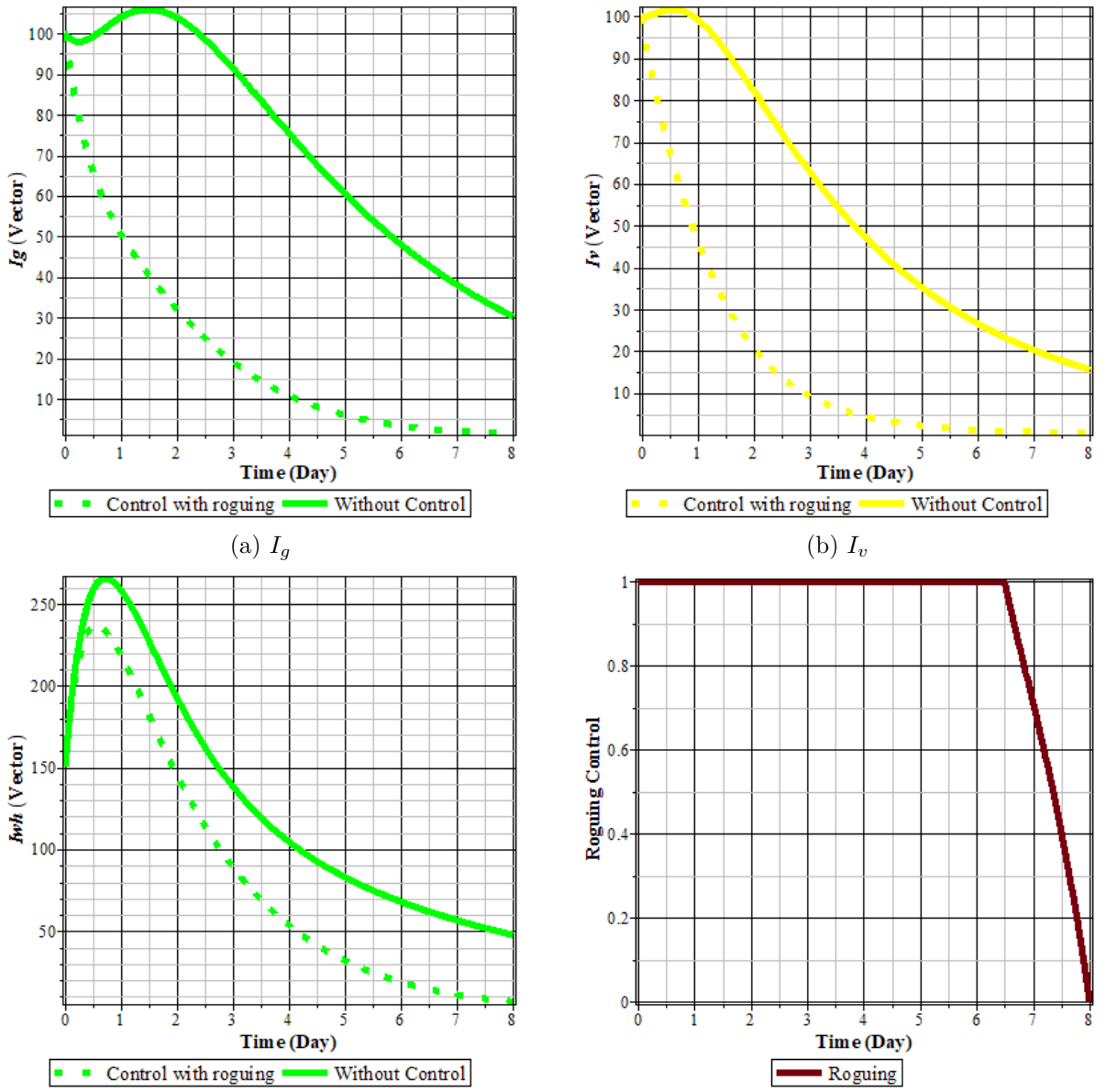
(a) Rice Plant (b) Vector

Figure 3: Population dynamics when $R_0 > 1$

3.2.2 Optimal Control

Fig. 4 shows the effectiveness of optimal control through roguing, which significantly reduced the spread of tungro disease in rice plants. The population of infected plants decreased consistently in both growth phases in the control scenario compared to the uncontrolled scenario. A significant decrease was also observed in the population of infected vectors, confirming that reducing the inoculum source by removing infected plants can directly suppress infection levels in both plants and vectors.

An optimal roguing strategy applied intensively, especially in the first seven days after initial infection detection, is crucial for breaking the chain of transmission and preventing a surge in the number of infected plants. This approach has the potential to lead the system toward a disease-free state and reduce the basic reproductive number, thereby preventing the formation of an endemic state and supporting sustainable rice production



(a) I_g (b) I_v

(c) I_{wh} (d) $u(t)$

Figure 4: Population dynamics when $R_0 > 1$

4 Conclusion

This study successfully developed a mathematical model that considers the growth phase of rice plants and control strategies, including roguing, to analyze the spread of tungro disease. Simulations showed that the application of roguing during the vegetative phase significantly reduced the number of infected plants and decreased the transmission rate. Dynamic analysis identified two equilibrium points, while sensitivity analysis using the Partial Rank Correlation Coefficient (PRCC) method confirmed that the level of infectivity and the roguing rate were the most influential parameters on the R_0 value. Using an optimal control approach based on Pontryagin's Maximum Principle, the best strategy combining roguing intensity and vector control can be determined to suppress infection and prevent endemic conditions. Thus, this optimal control strategy has been proven effective in reducing the infection rate and has the potential to lead the system towards a tungro-free plant condition.

CRediT Authorship Contribution Statement

Rika Amelia: Conceptualization, methodology, formal analysis, investigation, writing—review and editing, funding acquisition. **Nursanti Anggriani:** Conceptualization, methodology, formal analysis, writing—original draft preparation. **Rosiman:** Investigation, data curation, validation. **Abdul Gazir Syarifudin:** Writing—review and editing, project administration. Nadine **Zahra Chairunnisa:** Project administration, writing. **Angellyca Leoni Manuela:** Project administration, writing.

Declaration of Generative AI and AI-assisted technologies

In preparing this manuscript, the author utilized Grammarly to refine the language and verify the grammar.

Declaration of Competing Interest

The authors declare no competing interest.

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Data and Code Availability

Not applicable.

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