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THE EFFECT OF INSECTICIDE IN TUNGRO DISEASE TRANSMISSION MODEL WITH VEGETATIVE AND GENERATIVE PHASE

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Abstract: Tungro disease is one of the obstacles in rice cultivation that affects the achievement of sustainable development goals (SDGs), so it is necessary to control it to minimize losses due to decreased yields. The application of pesticides and natural enemies are techniques that can prevent this disease. To understand the population dynamics of the spread of Tungro disease in rice, modeling was carried out by considering two growth phases (vegetative and generative phases). From the model made, analysis is carried out in the form of dynamic analysis, optimal control, sensitivity, and numerical simulation to illustrate it. The results show that the rice population (both vegetative and generative) and infected green leafhoppers will decrease more quickly due to control by giving insecticides and predators.

Keywords: rice plants, tungro disease; growth phase; dynamic analysis; optimal control theory; sensitivity analysis; insecticide; predator.

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1. INTRODUCTION

One of the Sustainable Development Goals (SDGs) is to achieve food security and promote sustainable agriculture. In achieving this goal, fulfilling basic food needs (i.e., rice from the rice plant) is a must. However, when planting rice plants, they often experience obstacles: tungro virus disease [1]. This disease is caused by Rice Tungro Spherical Virus (RTSV) and Rice tungro Bacilliform Virus (RTBV), which are spread by green leafhopper vector, *Nephotettix virescens* [2-4]. Rice plants are divided into 2 phases: the vegetative phase (0-55 days after planting) and the generative phase (55-120 days after planting). The symptoms of this tungro disease will appear when the plant is 2-3 weeks after planting. The damage caused by this disease will undoubtedly impact reducing crop yields so that it hampers the achievement of SDGs. Therefore, efforts need to be made to control the spread of the disease by preventing the green leafhopper population as the primary mediator of green leafhopper disease transmission to the rice plant population. The use of pesticides and predators as biological agents is one of the most common control techniques [5].

Mathematical researchers have contributed to solving the problem of plant disease spread through mathematical modeling. This solution provides a robust understanding of population behavior and is very important in understanding the dynamics of plant disease spread. Many researchers have developed mathematical models to interpret how disease spreads in plant populations [5]. The often-developed vector-borne disease model, including the vector-borne disease model with direct transmission [6, 7]. The spread of green leafhoppers with insecticides and biological agents [8]. Then the usual vector communicable disease model by considering the plant population exposed before being infected [9-12]. This includes considering the impact of insecticidal and nutritional interventions on plant disease systems [13]. In addition, researchers have also carried out plant disease modeling by assessing the presence of pathogens as an effort to control the disease [14,15,26,27], as well as modeling by considering carrying capacity parameters as limits on the number of plant populations of vectors [16- 19] and disease models. Vector-borne by considering the stages of plant growth, namely vegetative and generative [20]. In contrast,

Suryaningrat et al. make a mathematical model with the intervention of insecticides and the presence of predators to control the disease by controlling the number of vector populations [5].

In this paper, we develop a mathematical model of transmission of tungro disease in rice plants by considering two growth phases (vegetative and generative phases) and provide optimal control treatment in the form of the application of pesticides and insecticides to suppress the spread of green leafhopper disease in rice plants. Not only that, but we also added predators to the model and analyzed them to determine the effect of predators on the green leafhopper population. In addition, we also perform a balanced analysis of the model, including its stability. Then a numerical simulation was carried out to see the population dynamics, and finally, a sensitivity analysis was carried out to see the most sensitive model parameters.

2. MATERIALS AND METHODS

This research uses dynamical system theory, sensitivity analysis, and optimal control theory. First, we use dynamical system theory to determine the equilibrium point and stability. Next, we used sensitivity analysis to find out the most influential parameter in the model. Then, we try to use optimal control theory to consider the cost of the intervention what we do to the system.

2.1 Dynamical System

The dynamical system is an approaching method used to study a system's behavior for the long term. A continuous dynamical system is said to have an equilibrium point if the differential equation $\dot{x} = f(x)$ has solution for $f(x) = 0$. The point of x^* which satisfied $f(x^*) = 0$ is called the equilibrium point.

Definition 1. If a point of x^* is an equilibrium point of (1), then the constant function $x(t) = x^*$ is a solution for a differential equation

$$0 = \frac{dx^*}{dt} = \frac{dx(t)}{dt} = f\big(x(t)\big) = f(x^*) = 0 \qquad (1)$$

A system stable if all the eigenvalues of the Jacobian Matrix of the system has negative value.

2.2 Sensitivity Analysis

Sensitivity analysis is a method we use to determine the correlation between parameters on

each compartment of the system. We use the formulas and theory in the paper was created by Marino et al. [21] and McKay et al. [22]. The result of this method gave us information that some parameters have a strong impact on the system. Then we use the two dominant parameters that we can control to make it possible to control with considering the cost.

2.3 Optimal Control Theory

Optimal control theory is a tool used to obtain the optimal control in a system with the constraints of the problem. Optimal control problem discusses control variables that have an impact on the system. We used the maximum Pontryagin principle to check that our objective function satisfies the necessary and sufficient conditions.

2.3.1 Necessary Condition

- Optimal Condition

$$\frac{\partial H}{\partial u} = 0, 0 \leq t \leq T \tag{2}$$

- Adjoint Function

$$\dot{\lambda} = -\frac{\partial H}{\partial x}, 0 \leq t \leq T \tag{3}$$

- Transversal Condition

$$\lambda(T) = 0, (x(T) = x^T \; if \; x^T known) \tag{4}$$

2.3.2 Sufficient Condition

- Minimum Condition

$$\frac{\partial^2 H}{\partial u^2} \geq 0 \tag{5}$$

- Maximum Condition

$$\frac{\partial^2 H}{\partial u^2} \leq 0 \tag{6}$$

2.4 Mathematical Model

We conclude some assumptions in the vector-borne disease model formulated in Amelia et al. [20] and Suryaningrat et al. [5]. We divided the plant population into four subpopulations, which healthy rice plants in the vegetative phase is denoted by S_v,the population of susceptible rice in the generative phase is denoted by S_g, the population of infected rice plants in the vegetative phase

denoted by I_v, and the population of infected rice plants in the vegetative phase denoted by I_g. Then there is the vector that carries the tungro disease, namely the green leafhopper. The population of green leafhoppers are divided into two classes, susceptible green leafhopper is denoted by S_{WH} and infected green leafhopper is denoted by I_{WH}. We also consider the existence of the predator denoted by Pr to suppress the vector. The model developed based on vector-borne model.

Based in the above assumptions, schematic diagrams of the spread of tungro disease in rice plants can be seen in Figure 1.

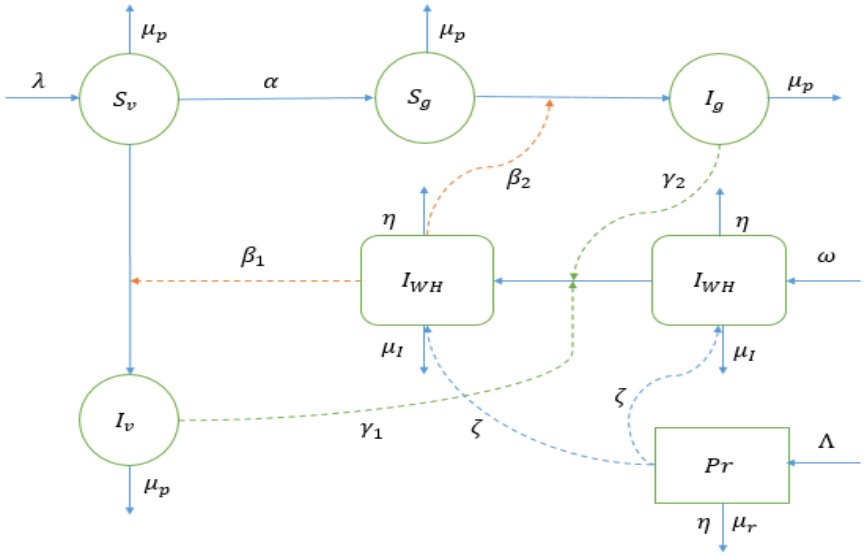


Figure 1. Schematic diagram of model spread tungro disease.

From Figure 1, a model can be constructed in the form of a differential equations as follows as:

$$\begin{aligned}
\frac{dS_v}{dt} &= \lambda - \alpha S_v - \beta_1 S_v I_{WH} - \mu_p S_v \\
\frac{dI_v}{dt} &= \beta_1 S_v I_{WH} - \mu_p I_v \\
\frac{dS_g}{dt} &= \alpha S_v - \beta_2 S_g I_{WH} - \mu_p S_g \\
\frac{dI_g}{dt} &= \beta_2 S_g I_{WH} - \mu_p I_g \\
\frac{dS_{WH}}{dt} &= \omega - \gamma_1 I_v S_{WH} - \gamma_2 I_g S_{WH} - \mu_I S_{WH} - \eta S_{WH} - \zeta Pr S_{WH} \\
\frac{dI_{WH}}{dt} &= \gamma_1 I_v S_{WH} + \gamma_2 I_g S_{WH} - \mu_I I_{WH} - \eta I_{WH} - \zeta Pr I_{WH}
\end{aligned} \tag{7}$$

$$\frac{dPr}{dt} = \Lambda - \mu_r Pr - \eta Pr$$

The parameters contained in this model are described in Table 1.

Table 1. Definition of variables and parameters

Variables/ Parameters	Definition	Unit
N_p	Rice plant population $(N_p = S_v + I_v + S_g + I_g)$	Individual Plant
N_{WH}	Green Leafhopper Population ($N_{wh} = S_{WH} + 3I_{WH}$)	Individual Vector
S_V	Healthy rice plant population in the vegetative phase	Individual Plant
I_V	Infected rice plant population in the vegetative phase	Individual Plant
S_g	Healthy rice plant population in the generative phase	Individual Plant
I_g	Infected rice plant population in the generative phase	Individual Plant
S_{WH}	Healthy Green Leafhopper Population	Individual Vector
I_{WH}	Infected Green Leafhopper Population	Individual Vector
λ	Rice plant recruitment rate	$\frac{1}{day}$
ω	Green leafhopper recruitment rate	$\frac{1}{day}$
α	Rice plant growth rate from vegetative to generative phase	$\frac{1}{day}$
β_1	The rate of infection of rice plants in the vegetative phase	$\frac{1}{individual \times day}$
β_2	The infection rate of rice plants in the generative phase	$\frac{1}{individual \times day}$
γ_1	Green leafhopper infection rate when taking food from infected rice plants in the vegetative phase	$\frac{1}{individual \times day}$
γ_2	Green leafhopper infection rate when taking food from infected rice plants in the generative phase	$\frac{1}{individual \times day}$
μ_P	Rice plant death rate	$\frac{1}{day}$
μ_I	Green leafhopper natural death rate	$\frac{1}{day}$
Λ	Predator recruitment	Individual Predator
μ_r	Natural death of a predator	$\frac{1}{day}$
η	Death due to insecticide application	$\frac{1}{day}$
ζ	Death due to predation of green leafhoppers by predators	$\frac{1}{day}$

3. RESULTS AND DISCUSSION

3.1 Dynamical System

3.1.1 Non-endemic Equilibrium Point

Based on model (7), we have three equilibrium point as follows:

1. Non-endemic Equilibrium Point

$$\{S_v, I_v, S_g, I_g, S_{WH}, I_{WH}, Pr\} = \left\{\frac{\lambda}{\mu_p+\alpha}, 0, \frac{\lambda\alpha}{\mu_p(\mu_p+\alpha)}, 0, \frac{\omega(\mu_r+\eta)}{\eta^2+(\mu_i+\mu_r)\eta+\Lambda\zeta+\mu_i\mu_r}, 0, \frac{\Lambda}{\mu_r+\eta}\right\}$$

2. Endemic Equilibrium Point 1

$$\{S_v, I_v, S_g, I_g, S_{WH}, I_{WH}, Pr\} = \{\overline{S_V}, \overline{I_v}, \overline{S_g}, \overline{I_g}, \overline{S_{WH}}, \overline{I_{WH}}, \overline{Pr}\}$$

3. Endemic Equilibrium Point 2

$$\{S_v, I_v, S_g, I_g, S_{WH}, I_{WH}, Pr\} = \{\overline{\overline{S_V}}, \overline{\overline{I_v}}, \overline{\overline{S_g}}, \overline{\overline{I_g}}, \overline{\overline{S_{WH}}}, \overline{\overline{I_{WH}}}, \overline{\overline{Pr}}\}.$$

3.1.2 Basic Reproduction Ratio

The basic reproduction ratio $(\mathcal{R}_0)$ is an important parameter in epidemiology. This parameter is used to determine the number of secondary infections caused by primary infections in susceptible populations. The next-generation method is used for determining the basic reproduction ratio $(\mathcal{R}_0)$, where v is the transfer matrix and f is the new infection matrix.

$$f = \begin{bmatrix} \beta_1 S_v I_{WH} \\ \beta_2 S_g I_{WH} \\ \gamma_1 I_v S_{WH} + \gamma_2 I_g S_{WH} \end{bmatrix} \text{ and } v = \begin{bmatrix} \mu_p I_v \\ \mu_p I_g \\ \mu_i I_{WH} + \eta I_{WH} + \zeta Pr I_{WH} \end{bmatrix}$$

The basic reproduction ratio is obtained from the dominant eigenvalue commonly called the spectral radius of (FV^{-1}), where F and V are the Jacobian matrices of f and v obtained at non-endemic equilibrium points as follows:

$$\mathcal{R}_0 = (\eta+\mu_r)\sqrt{\frac{\omega\lambda(\mu_p\beta_1\gamma_1+\alpha\beta_2\gamma_2)}{\mu_p^2(\mu_p+\alpha)((\eta+\mu_r)(\eta+\mu_i)+\Lambda\zeta)^2}} \tag{8}$$

3.1.3 Stability Analysis

Theorem 1. The non-endemic equilibrium point of model (2) is locally asymptotically stable if

$\mathcal{R}_0 < 1$.

Proof. Through the method in [23], to confirm the non-endemic equilibrium point stability in a model, it can be known by subtitute the non-endemic equilibrium point E_{df} into the Jacobian matrix of model (2). Then, we obtain:

$$J = \begin{bmatrix} -\alpha-\mu_p & 0 & 0 & 0 & 0 & -\beta_1(S_v)_{df} & 0 \\ 0 & -\mu_p & 0 & 0 & 0 & \beta_1(S_v)_{df} & 0 \\ \alpha & 0 & -\mu_p & 0 & 0 & -\beta_2(S_g)_{df} & 0 \\ 0 & 0 & 0 & -\mu_p & 0 & \beta_2(S_g)_{df} & 0 \\ 0 & -(S_{WH})_{df}\gamma_1 & 0 & -(S_{WH})_{df}\gamma_2 & -\eta-\mu_i-(Pr)_{df}\zeta & 0 & -\zeta(S_{WH})_{df} \\ 0 & (S_{WH})_{df}\gamma_1 & 0 & (S_{WH})_{df}\gamma_2 & 0 & -\eta-\mu_i-Pr\zeta & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\eta-\mu_r \end{bmatrix}.$$

The characteristic polynomial of J is

$$P(x) = \left(x+\mu_p\right)^2\left(x+\alpha+\mu_p\right)(x+\mu_r+\eta)\left((\eta+\mu_r)x+(\eta+\mu_r)(\eta+\mu_i)+\Lambda\zeta\right)(ax^2+bx+c)$$

where

$$a = \mu_p\left(\mu_p+\alpha\right)\left(\eta^3+(\mu_i+2\mu_r)\eta^2+(\Lambda\zeta+2\mu_i\mu_r+\mu_r^2)\eta+\mu_r(\Lambda\zeta+\mu_i\mu_r)\right) > 0$$

$$b = \mu_p\left(\mu_p+\alpha\right)\left((\eta+\mu_r)(\eta+\mu_i)+\Lambda\zeta\right)\left((\mu_r+\eta)\left(\mu_i+\mu_p+\eta\right)+\Lambda\zeta\right) > 0$$

$$c = (\eta^4+2(\mu_i+\mu_r)\eta^3+(2\Lambda\zeta+\mu_i^2+4\mu_i\mu_r+\mu_r^2)\eta^2+2(\mu_i+\mu_r)(\Lambda\zeta+\mu_i\mu_r)\eta+\Lambda^2\zeta^2+\mu_i^2\mu_r^2)\mu_p^3+\alpha(\eta^4+2(\mu_i+\mu_r)\eta^3+(2\Lambda\zeta+\mu_i^2+4\mu_i\mu_r+\mu_r^2)\eta^2+2(\mu_i+\mu_r)(\Lambda\zeta+\mu_i\mu_r)\eta+(\Lambda\zeta+\mu_i\mu_r)^2)\mu_p-\omega\lambda(\mu_r+\eta)^2\left(\mu_p\beta_1\gamma_1+\alpha\beta_2\gamma_2\right) > 0$$

$$c = (\eta^4+2(\mu_i+\mu_r)\eta^3+(2\Lambda\zeta+\mu_i^2+4\mu_i\mu_r+\mu_r^2)\eta^2+2(\mu_i+\mu_r)(\Lambda\zeta+\mu_i\mu_r)\eta+\Lambda^2\zeta^2+\mu_i^2\mu_r^2)\mu_p^3+\alpha(\eta^4+2(\mu_i+\mu_r)\eta^3+(2\Lambda\zeta+\mu_i^2+4\mu_i\mu_r+\mu_r^2)\eta^2+2(\mu_i+\mu_r)(\Lambda\zeta+\mu_i\mu_r)\eta+(\Lambda\zeta+\mu_i\mu_r)^2)\mu_p^2-\omega\lambda(\mu_r+\eta)^2\left(\mu_p\beta_1\gamma_1+\alpha\beta_2\gamma_2\right) > 0$$

$$c = \mu_p^3(\eta^4+2(\mu_i+\mu_r)\eta^3+(2\Lambda\zeta+\mu_i^2+4\mu_i\mu_r+\mu_r^2)\eta^2+2(\mu_i+\mu_r)(\Lambda\zeta+\mu_i\mu_r)\eta+\Lambda^2\zeta^2+\mu_i^2\mu_r^2)+\alpha\mu_p^2\,(\eta^4+2(\mu_i+\mu_r)\eta^3+(2\Lambda\zeta+\mu_i^2+4\mu_i\mu_r+\mu_r^2)\eta^2+2(\mu_i+\mu_r)(\Lambda\zeta+\mu_i\mu_r)\eta+(\Lambda\zeta+\mu_i\mu_r)^2)-R^2\mu_p^2\left(\mu_p+\alpha\right)\left((\eta+\mu_r)(\eta+\mu_i)+\Lambda\zeta\right)^2 > 0$$

$$c = \left(1-{\mathcal{R}_0}^2\right)\mu_p^2\left(\mu_p+\alpha\right) > 0.$$

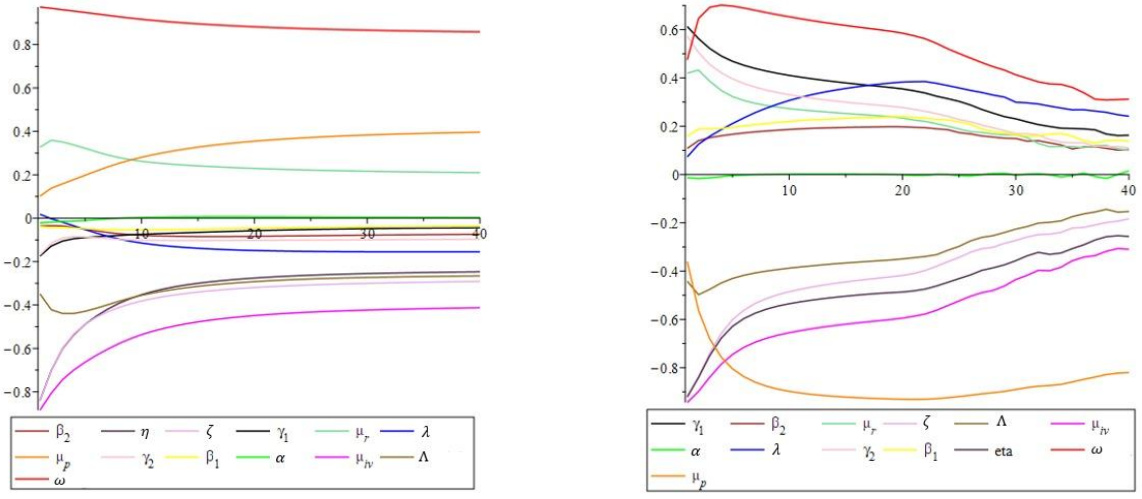
Based on equation $P(x)$, it shows that all eigenvalues has a negative value. It means that model (2) is locally asymptotically stable at the non-endemic equilibrium point E_{df} if $\mathcal{R}_0 < 1$.

3.2 Sensitivity Analysis

We used sensitivity analysis to find out the most crucial parameter of the model. First, we take 5000 samples through the Latin Hypercube Sampling (LHS) and then use the Partial Rank Correlation Coefficient (PRCC) to discover the correlation of the parameter. Finally, We compute the vector, both susceptible and infectious, and the result is:

Figure 2.a shows that the most dominant parameter is μ_I which has a negative relationship. It describes that when the value r increases, so the susceptible vector decreased. Meanwhile, ζ and η have a significant effect on the number of susceptible vector population but it all decrease over the time.

Figure 2.b shows that the most dominant parameter μ_p. This parameter has negative relationship, it describes that when the value μ_p increase, so the number of infectious vector decrease. Meanwhile, parameters like ζ and η has the significant effect on the number of susceptible vector population but it all decrease over the time.



a. Compartment S_{WH} b. Compartment I_{WH}

Figure 2. Sensitivity Analysis on S_{WH} and I_{WH}.

3.3 Optimal Control Model

This section is devoted to investigating the optimal intervention to reduce the population of

green leafhoppers that can spread tungro disease, so it is expected that the rice population infected with tungro disease can be reduced. We combine the following two controls into a complete model (7)

u_1: Control of the use of insecticide

u_2: Adding more predators to the ecosystem.

The model system now reads

$$\begin{aligned}
\frac{dS_v}{dt} &= \lambda - \alpha S_v - \beta_1 S_v I_{WH} - \mu_p S_v \\
\frac{dI_v}{dt} &= \beta_1 S_v I_{WH} - \mu_p I_v \\
\frac{dS_g}{dt} &= \alpha S_v - \beta_2 S_g I_{WH} - \mu_p S_g \\
\frac{dI_g}{dt} &= \beta_2 S_g I_{WH} - \mu_p I_g \\
\frac{dS_{WH}}{dt} &= \omega - \gamma_1 I_v S_{WH} - \gamma_2 I_g S_{WH} - \mu_I S_{WH} - (1+u_1)\eta S_{WH} - \zeta Pr S_{WH} \\
\frac{dI_{WH}}{dt} &= \gamma_1 I_v S_{WH} + \gamma_2 I_g S_{WH} - \mu_I I_{WH} - (1+u_1)\eta I_{WH} - \zeta Pr I_{WH} \\
\frac{dPr}{dt} &= (1+u_2)\Lambda - \mu_r Pr - (1+u_1)\eta Pr
\end{aligned} \tag{9}$$

with initial conditions

$$S_v(0), I_v(0), S_g(0), I_g(0), S_{WH}(0), I_{WH}(0), Pr \geq 0.$$

A combination of the population of healthy and infected green leafhoppers, together with the cost of insecticide use and adding more predators, is considered as the objective function to be minimized, namely,

$$J(u_1, u_2) = \int_0^T [A\, I_{WH}(t) + B\, S_{WH}(t) + C_1 u_1^2(t) + C_2 u_2^2(t)]\, dt \tag{10}$$

where T is the final time, A, B are positive weight constant, and C_1, C_2 is the weight constant for the cost of the tungro disease reduction strategy.

The control problem formed from the constraint equation as the equation of each compartment (9) and the objective function (10), it is possible to form a Hamiltonian equation related to the optimal control problem. Therefore, we look for, using Pontryagin's maximum principle [24], the

optimal control $(u_1^*, u_2^*) \in U$ satisfying (9), such that

$$J(u_1^*, u_2^*) = \min\{J(u_1, u_2) \mid (u_1, u_2) \in U\}. \tag{11}$$

The associated pseudo-Hamiltonian is

$$\begin{aligned} H = {} & A\, I_{WH} + B\, S_{WH} + C_1 u_1^2 + C_2 u_2^2 + L_1\left(\lambda - \alpha S_v - \beta_1 S_v I_{WH} - \mu_p S_v\right) \\ & + L_2\left(\beta_1 S_v I_{WH} - \mu_p I_v\right) + L_3\left(\alpha S_v - \beta_2 S_g I_{WH} - \mu_p S_g\right) + L_4\left(\beta_2 S_g I_{WH} - \mu_p I_g\right) \\ & + L_5\left(\omega - \gamma_1 I_v S_{WH} - \gamma_2 I_g S_{WH} - \mu_I S_{WH} - (1+u_1)\eta S_{WH} - \zeta Pr S_{WH}\right) \\ & + L_6\left(\gamma_1 I_v S_{WH} + \gamma_2 I_g S_{WH} - \mu_I I_{WH} - (1+u_1)\eta I_{WH} - \zeta Pr I_{WH}\right) \\ & + L_7((1+u_2)\Lambda - \mu_r Pr - (1+u_1)\eta Pr) \end{aligned} \tag{12}$$

where $L_1, i = 1, \ldots, 7$ are adjoint variables satisfying (3) and (4), i.e

$$\begin{aligned} \dot{L}_1 &= -\alpha L_3 - \beta_1 I_{WH} L_2 - \left(-\alpha - \beta I_{WH} - \mu_p\right) L_1 \\ \dot{L}_2 &= \gamma_1 L_5 S_{WH} - \gamma_1 L_6 S_{WH} + \mu_p L_2 \\ \dot{L}_3 &- \beta_2 I_{WH} L_4 - \left(-\beta_2 I_{WH} - \mu_p\right) L_3 \\ \dot{L}_4 &= \gamma_2 L_5 S_{WH} - \gamma_2 L_6 S_{WH} + \mu_p L_4 \\ \dot{L}_5 &= -B - \left(\gamma_1 I_v + \gamma_2 I_g\right) L_6 - (-\eta(u_1+1) - \gamma_1 I_v - \gamma_2 I_g - \mu_1 - \zeta \mathrm{Pr}) L_5 \\ \dot{L}_6 &= -A + \beta_1 L_1 S_v - \beta_1 L_2 S_v + \beta_2 L_3 S_g - \beta_2 L_4 S_g - (-\eta(u_1+1) - \mu_1 - \zeta \mathrm{Pr}) L_6 \\ \dot{L}_7 &= \zeta I_{WH} L_6 + \zeta L_5 S_{WH} - (-\eta(u_1+1) - \mu_r) L_7 \end{aligned} \tag{13}$$

with the final condition $L_i(T) = 0$ for $i = 1, \ldots, 7$. The necessary and sufficient optimal condition satisfying (2) and (5), which in turn give the optimal control

$$\begin{aligned} u_1^* &= \max\left\{0, \min\left(1, \frac{\eta(I_{WH} L_6 + L_5 S_{WH} + L_7 Pr)}{2C_1}\right)\right\} \\ u_2^* &= \max\left\{0, min\left(1, \frac{\Lambda L_7}{2C_2}\right)\right\}. \end{aligned} \tag{14}$$

3.5 Numerical Simulation

In this section, we shall carry out numerical simulations of the model to assess the impact of various control strategies on the dynamics of the disease. First, we will perform a numerical

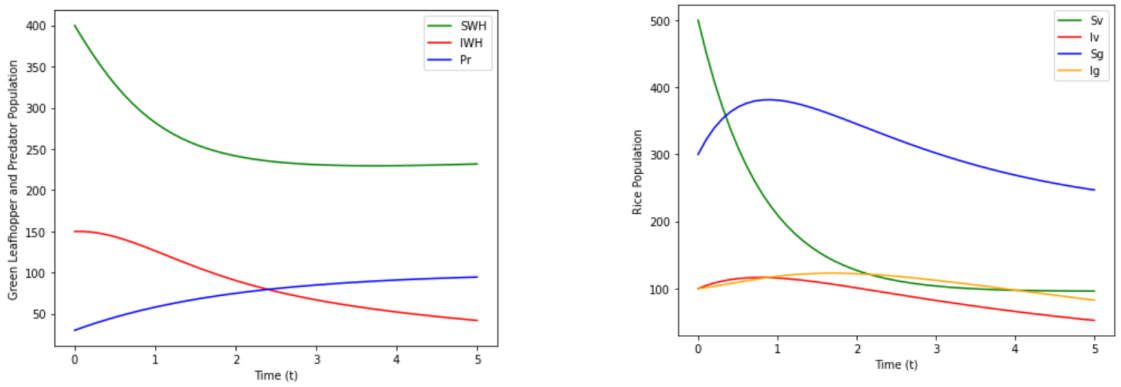
simulation on the model without control. The equations of the model (7) are solved numerically using Python ode solver in *scipy* library, which is based on the Explicit fourth-order Runge-Kutta method. The dynamics of the spread of tungro disease are shown in Figures. 2 with initial values and parameter values in Table 2.

Table 2. Parameter and Initial value for the model

Parameters /Variables	**Value**
λ	100
ω	500
α	0.7
β_1	0.001
β_1	0.001
γ_1	0.0025
γ_2	0.002
μ_p	0.3
μ_1	0.7
μ_r	0.3
η	0.2
Λ	50
ζ	0.01
S_v	500
I_v	100
S_g	300
I_g	100
S_{WH}	400
I_{WH}	150
Pr	30

Figure 3.a shows that the population of green leafhoppers, both susceptible and infected populations, will decrease. This is due to the presence of green leafhoppers by predators. While the predator population over time will increase. This is due to the recruitment rate of predators.

Figure 3.b it can be seen that all plant populations decreased for both susceptible rice plant populations (vegetative and generative phases) and infected rice plant populations (vegetative and generative phases), but the susceptible plant population in the generative phase increased. This is because of the growth of susceptible plants in the vegetative phase to susceptible plants in the generative phase.



(a) **(b)**

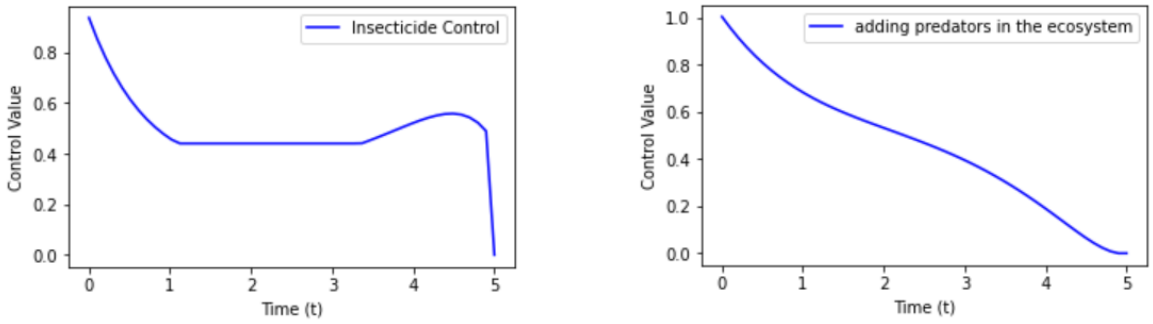
Figure 3. Dynamics population of green leafhoppers and their predators (a) and rice (b).

We proceed with numerical simulations for the optimal control problem model system (9) using the initial values of the compartments and model parameters in Table 2, then the constants on the objective function are positive weight constants $A = B = 1$ and control strategy costs $C_1 = 5$, $C_2 = 7$. In order to investigate the impact of various control strategies, the following three scenarios are considered

1. Strategy A: Insecticide control only $(u_2 = 0)$,
2. Strategy B: With the addition of predators in the ecosystem only $(u_1 = 0)$,
3. Strategy C: Both insecticide control and adding predators in the ecosystem are carried out $(u_1, u_2 \neq 0)$.

With a forward-backward sweep algorithm that is done in python programming, obtained control values for each non-zero control are as follows. Figure 4.a and Figure 4.b show that controlling using insecticides and adding predators in the ecosystem is sufficient only until the fifth week, but

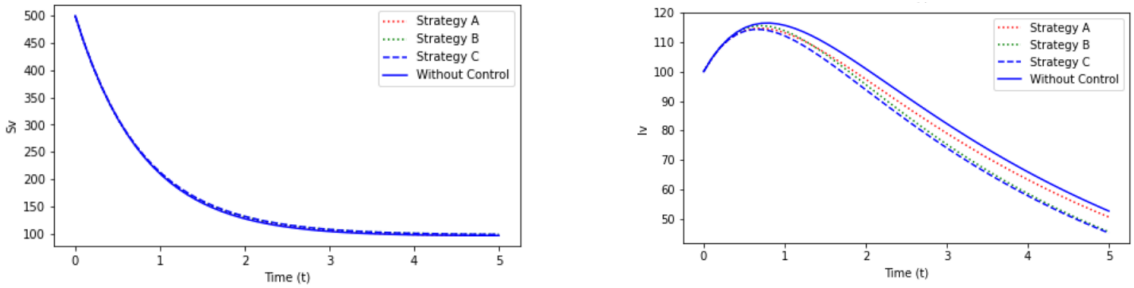
for control using insecticides in the fourth week, more insecticides must be given (adding it as much as 10% of last week).



(**a**) (**b**)

Figure 4. Control values u_1 (a) and u_2 (b) against time.

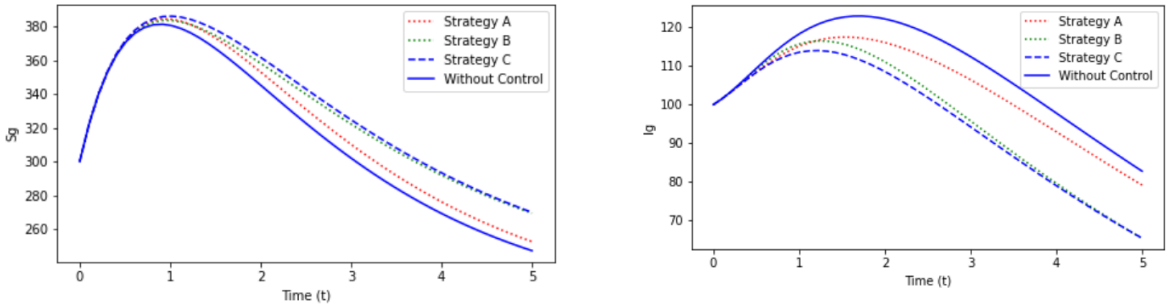
The following is a comparison chart of strategies in each compartment



(**a**) (**b**)

Figure 5. Effect of each strategy on S_v (a) and I_v (b) compartments.

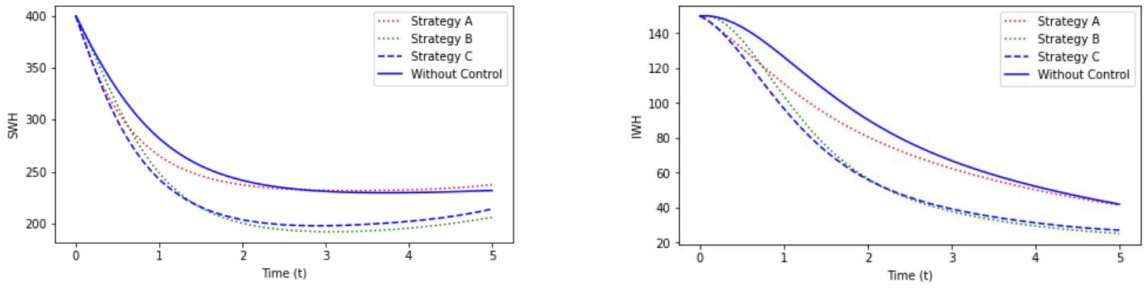
Based on Figure 5, the S_v compartment does not have much effect due to control either by adding predators or controlling insecticides because the population continues to move to the generative phase compartment over time. At the same time, the population I_v with control was lower than without control due to the fewer number of infected vectors after control.



(**a**) (**b**)

Figure 6. Effect of each strategy on S_g (a) and I_g (b) compartments.

Based on Figure 6, the population of healthy rice in the generative phase (S_g) with control is higher than the population without control. The number of infected rice is less, resulting in more healthy rice. And the infected rice population during the generative period (I_g) will be lower after being controlled because the number of vectors is fewer.



(a) **(b)**

Figure 6. Effect of each strategy on S_{WH} (a) and I_{WH} (b) compartments.

Based on Figure 6, green leafhopper populations, both infected and healthy will decrease due to increased mortality by predators and insecticides.

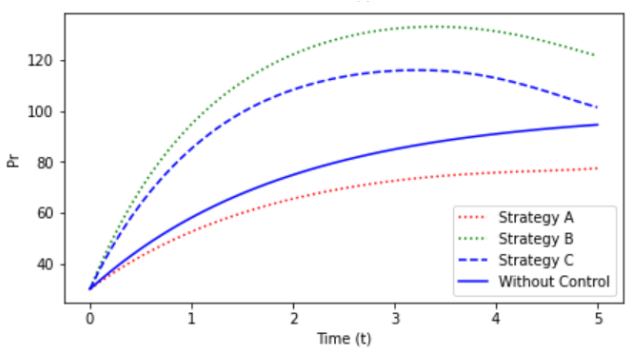


Figure 7. Effect of each strategy on Pr compartments.

Based on Figure 7, The Pr compartment with Strategy B saw a much larger increase in the number of predators. However, with the addition of insecticide control (Strategy C), predators did not increase too much. Meanwhile, with insecticide control alone (Strategy A) the number of predators will be lower.

4. DISCUSSION

Previous research on tungro disease by developing a model with roguing to control the spread of the tungro virus was then applied to numerical simulations [3-4] and the optimal control of tungro with insecticides [25] and biological agents [5]. The results show that we can see the rice

population's dynamics by dividing the rice plant into generative and vegetative phases. Finally, optimal control is applied to this model in the form of a predator. This model can be developed in the future by adding the persistence of green leafhoppers.

5. CONCLUSION

The constructed model of tungro disease transmission in the vegetative and generative phases has three equilibrium points, one non-endemic point, and two endemic points. Non-endemic point stability indicates that the system will be stable if $\mathcal{R}_0 < 1$. Based on the sensitivity analysis, it can be concluded that the death of the vector strongly influences the population of susceptible rice plants. In contrast, the population of infected green leafhoppers is strongly influenced by the death rate of the rice plant. At the same time, the simulation results show that the infected and uninfected green leafhopper population decreases. As a result, the infected rice population during the vegetative and generative periods will decrease, while the healthy productive rice population will be more. Likewise, if controlled using insecticides and predators, the people of rice (both vegetative and generative) and infected green leafhoppers will decrease more quickly.

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CONFLICT OF INTERESTS

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

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