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STABILITY ANALYSIS OF TUNGRO DISEASE SPREAD MODEL IN RICE PLANT USING MATRIX METHOD

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Abstract. Rice is one of the staple foods produced from the rice plant. Rice productivity is increased by carrying out efforts to control diseases that usually attack rice plants. Tungro is one of the most destructive diseases of rice plants. Mathematical models can help solve problems in the spread of plant diseases. In this paper, the development of a mathematical model for the spread of tungro disease in rice plants with 6 compartments is developed involving rice in the vegetative and generative phases. Furthermore, stability analysis is carried out on the obtained model by using the Basic Reproduction Number (R_0) search through the matrix method, especially through the search for transition matrices and transmission matrices. The analytical results show that when $R_0 < 1$ the non-endemic equilibrium point is stable. Numerical results showed that rice plants in the generative phase.

Keywords: : stability, basic reproduction number, plant disease, transition matrix, transmission matrix.

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1. INTRODUCTION

One of the goals of the Sustainable Development Goals (SDGs) is to achieve food security and promote sustainable agriculture. To meet food security, one of which is the availability of staple foods, such as rice which is a staple food derived from rice plants. Therefore, efforts need to be made to achieve this target, by controlling the spread of certain diseases in rice plants, one of which is tungro disease. Green leafhopper is a vector that is the primary mediator of the transmission of tungro disease to the rice plant population. If the infection occurs at the nursery stage, tungro symptoms will appear on plants aged 2-3 weeks after planting. Infected young rice plants are a source of primary inoculum after rice is planted in the field. During one period of rice plant growth there were two peaks of infected plants. The first peak of infection was caused by immigrant insects, while the second peak of infection was caused by infection with descendants of immigrant insects.

The field of mathematics can contribute to solving these problems; one way is through mathematical modeling. Mathematical modeling can provide significant in-sight into population behavior and is essential in understanding plant disease dynamics. Research on mathematical modeling of the spread of plant diseases has been carried out, one of which is the development of mathematical models for vector infection dynamics [1]. There are researchers who make epidemic models that describe the dynamics of the spread of plant diseases transmitted by vectors by building the Lyapunov function for global stability [2]. Other researchers have modeled the spread of disease in maize [3].

To see the effectiveness of a model made, a very important step is to analyze the stability of the model. The asymptotically stable equilibrium state of a model is called a particular property of interest [4]. Examples of research that performs model stability analysis include modeling and stability analysis as well as optimal control of vector-borne diseases with nonlinear incidents [5]. Research that creates and analyzes a plant disease epidemic model with monotonic and bilinear cases [6]. In addition, researchers who analyze plant disease models that are transmitted through vectors using fractional derivatives [7]. Researchers who create and analyze mathematical models of insect-borne dispersion diseases with regard to climate change [8], followed by performing numerical simulations to understand the behavior of the mathematical model [9]. Other researchers, carried out mathematical modeling of the spread of diseases specifically on rice plants. They performed an analysis of the insecticidal effect for infected plants and briefly discussed the stability analysis of the model, and presented optimal control via Pontryagin's Maximum Principle [10]. The following year researchers continued to study plant epidemic models involving fungicides with curative treatments [11], and determine the optimal control of the mathematical model of plant diseases to see the effectiveness of the application of fungicides [12]. Furthermore, in 2020, researchers will create and analyze the stability of a mathematical model of the spread of diseases in rice by involving vectors transmitted by green leafhoppers by considering predator-prey interactions between green leafhoppers and ladybugs [13] and researchers who analyzed the mathematical model of the spread of the yellow virus on red chili plants through insect vectors with logistic functions [14].

All of the above plant disease distribution models use stability analysis with various methods. The most widely used method for stability analysis of plant disease distribution models is the method that involves the Jacobian matrix, which is to find the roots of the characteristic equation of the formed Jacobian matrix. This method uses a separate step between stability analysis and finding the Ro value. In addition, for models with a large number of compartments, this method requires the assistance of other methods in determining the roots of the characteristic equations.

For this reason, a method that can directly determine the value of Ro and analyze the stability of the model is needed, without the help of other methods. The basic reproduction number (R_0) is a key parameter in plant disease epidemiology, which largely determines whether or not an epidemic will occur in a plant population. Research on methods for finding Ro in plant disease distribution models has been carried out, namely research that calculates the basic reproduction number of vector-borne plant virus epidemics, using the Next Generation Matrix approach [15]. However, this research has only reached the stage of finding Ro, it has not been used further for model stability analysis.

We developed a mathematical model for the spread of tungro disease in rice plants, which is a development of the previous model [10], by adding rice compartments in the vegetative and generative phases. Furthermore, stability analysis was carried out on the obtained model by using the Basic Reproduction Number (R_0) search through the matrix method. The matrix method referred to in this study is to utilize the properties of the matrix formed, by separating the transition and transmission matrices [16]. Basically, the

matrix method is commonly used in stability analysis of disease distribution models. However, the matrix method that is commonly used is only at the stage of forming the Jacobian matrix, and has not yet separated and used the properties of the transition and transmission matrices. So that the matrix method used in this paper can be an effective alternative to analyze the stability of the model for the spread of tungro disease in rice plants.

2. RESEARCH METHODS

2.1. Basic of Algebra[16]

In this section, some algebraic concepts will support the discussion of the problem.

Definition 1

Let A be a $p \times p$ real matrix, and $\overline{p} = \{1, 2, ..., p\}$. Then the following properties are satisfied. 1. *A* is the M-matrix if $A_{ii} \ge 0$, $A_{ij} \le 0$; $\forall i, j \neq i \in \bar{p}$.

2. *A* is a Metzler matrix denoted by $A \in M_E^{pxp}$ if $A_{ii} \ge 0$; $\forall i, j \neq i \in \bar{p}$.

Theorem 1

Assume that $A \in \mathbb{R}^{n \times n}$, then the following properties apply.

- (i) A is an M-matrix if and only if $(-A) \in M_E^{n \times n}$
- (ii) A is nonsingular with $A^{-1} > 0$ if and only if A is an M-matrix
- (iii) A is nonsingular with $A^{-1} > 0$ if and only if $(-A) \in M_E^{n \times n}$ is the stability matrix.

Definition 2

Let A be a $p \times q$ real matrix, and $\overline{p} = \{1, 2, ..., p\}$, $\overline{q} = \{1, 2, ..., q\}$. Then A is a nonnegative $(A \ge 0)$ if $A_{ii} \geq 0; \forall_i \in \bar{p}, \forall_i \in \bar{q},$

Definition 3

Let = $(A_{ij}) \in \mathbb{R}^{p \times q}$, and $\overline{p} = \{1, 2, \dots, p\}$, $\overline{q} = \{1, 2, \dots, q\}$. Then A is a positive matrix (A > 0) if $A_{ij} > 0$ 0; $\forall_i \in \bar{p}, \forall_i \in \bar{q}$.

Theorem 2: Local Stability Disease-free equilibrium point

Let F is the transmission matrix and V is the transition matrix at the disease-free equilibrium point. The linearized system around the disease-free equilibrium point is unstable if some of the conditions given below hold:

1) $(F - V) \in M_E^{nxn}$ is not a stability matrix 2) $(F - V) \in M_E^{nxn}$ and $(V - F)^{-1}$ either does not exist or if it exists is not positive. 3) $(-V) \in M_E^{nxn}$ and it exists as V^{-1} which is not positive, $F \ge 0$ and $\rho(FV^{-1}) > 1$.

This theorem is equivalent to the Theorem 3 below

Theorem 3: Local Stability Disease-free equilibrium point

Let $F \in \mathbf{R}^{n \times n}$ is the transmission matrix and $V \in \mathbf{R}^{n \times n}$ is the transition matrix at the disease-free equilibrium point. The linearized system around the disease-free equilibrium point is stable if all the conditions given below apply:

- $(F V) \in M_E^{nxn}$ is a stability matrix 1)
- $(F V) \in M_E^{nxn}$ and $(V F)^{-1}$ exist and positive matrix $(-V) \in M_E^{nxn}$, $V^{-1} > 0$, $F \ge 0$ dan $\rho(FV^{-1}) < 1$. 2)
- 3)

A joint sufficiency-type condition or any of the above conditions (1)-(2) to hold is condition 3.

Theorem 4: Local Stability and Existence of Disease Equilibrium Point

Let $F \in \mathbf{R}^{nxn}$ and $V \in \mathbf{R}^{nxn}$ are transmission matrix and transition matrix at disease-free equilibrium point, respectively. While $F_e \in \mathbf{R}^{nxn}$ and $V_e \in \mathbf{R}^{nxn}$ are respectively transmission matrix and transition matrix at disease equilibrium point, following properties apply:

- 1) Transition matrix at disease equilibrium point and free from disease is (V_e) and (V) identical and does not depend on a reproduction number, and $(-V) \in M_E^{nxn}$ is stability matrix.
- 2) Disease equilibrium points and disease free equilibrium points exist and are unique to all $R_0 \in [0, +\infty]$
- 3) Disease-free transmission matrix and disease-transmission matrix are non-negative, i.e. $F = F(R_0) \ge 0$, $F_e = F_e(R_0) \ge 0$ and $R_0 = \rho(FV^{-1})$ is a reproduction number.

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4) If $R_0 < 1$ then $F < F_e$, if $R_0 > 1$ then $> F_e$, and if $R_0 = 1$ then $F = F_e$. Then asymptotically stable local disease equilibrium point if $R_0 \ge 1$ and unstable if $R_0 < 1$.

2.2. Analysis of Stability Differential Equation Systems using Matrix Method

The flow of the analysis of the stability of the system of differential equations using the matrix method is as follows:

- 1. Determine disease-free equilibrium point and disease equilibrium point from the model.
- 2. Model is constructed into a matrix by performing a linearization process to obtain the Jacobian matrix from the system. This matrix consists of a noninfective (disease-free) Jacobian matrix and an infective Jacobian matrix.
- 3. Matrix $F \in \mathbf{R}^{nxn}$ and matrix $(V) \in \mathbf{R}^{nxn}$ are disease transmission matrix and transition matrix of linearized system around a disease-free equilibrium point, respectively. Matrix $F_e \in \mathbf{R}^{nxn}$ and matrix $(V_e) \in \mathbf{R}^{nxn}$ are disease transmission matrix and transition matrix of linearized system around a disease equilibrium point, respectively. Matrix F, F_e and V, V_e are determined by partitioning matrix.
- 4. Analysis of characteristics of transition matrix with its stability properties is related to properties of Metzler matrix and M-matrix. Analysis of transmission matrix, so that transmission matrix is not negative. This transition matrix and transmission matrix will define an auxiliary matrix (Next Generation Matrix), namely V^{-1} . The largest eigenvalue of the Next Generation Matrix is a parameter relevant to the system's stability and determines the sum of a basic reproduction number of diseases.
- 5. Furthermore, a linearized system around a disease-free equilibrium point is locally stable if this auxiliary matrix (Next Generation Matrix) has a maximum positive eigenvalue and less than one. The basic reproduction number coincides with the spectral radius of the disease spread model.

3. RESULTS AND DISCUSSION

3.1. Formulation of Mathematical Model

Assume that the plant population is divided into four classes. The population of healthy rice plants in the vegetative phase is denoted by S_v , the population of infected rice plants in the vegetative phase is denoted by I_v , the population of susceptible rice plants in the generative phase is denoted S_g and the population of infected rice plants in the generative phase is denoted I_g . Then there is the vector that carries the tungro disease, namely the green leafhopper. The population of green leafhoppers is divided into two classes, susceptible green leafhoppers are denoted by S_{WH} and infected green leafhoppers are denoted by I_{WH} . The rate of recruitment of rice plants is denoted and the rate of vector recruitment is denoted which is constant and each population enters the vulnerable compartment. Healthy plant S_v , S_g infected through an infected by sucking the rice plants affected by the disease. Infected plants and vectors cannot recover. Dimensions of time used in days. Model designed based on host vector model. Another assumption is that there is no influence of environmental factors and climatic factors.

Based on the above assumptions, schematic diagrams and flowcharts of the spread of tungro disease in rice plants can be seen in Figure 1 and Figure 2



Figure 1. Schematic diagram of model spread tungro disease in rice plants with vector transmission



Figure 2. Flowchart of model spread tungro disease in rice plants

From Figure 1 and Figure 2, a model can be constructed in the form of a differential equation as follows:

$$\frac{dS_{v}}{dt} = \lambda - \alpha S_{v} - \beta_{1} S_{v} I_{WH} - \mu_{p} S_{v}
\frac{dI_{v}}{dt} = \beta_{1} S_{v} I_{WH} - \mu_{p} I_{v}
\frac{dS_{g}}{dt} = \alpha S_{v} - \beta_{2} S_{g} I_{WH} - \mu_{p} S_{g}
\frac{dI_{g}}{dt} = \beta_{2} S_{g} I_{WH} - \mu_{p} I_{g}
\frac{dS_{WH}}{dt} = \omega - \gamma_{1} I_{v} S_{WH} - \gamma_{2} I_{g} S_{WH} - \mu_{I} S_{WH}
\frac{dI_{WH}}{dt} = \gamma_{1} I_{v} S_{WH} + \gamma_{2} I_{g} S_{WH} - \mu_{I} I_{WH}$$
(1)

The parameters contained in this model are described in Table 1

Variables/ Parameters	Definition	Unit		
N_p	Rice plant population $(N_p = S_v +$	Individual Plant		
	$I_v + S_g + I_g$			
N _{WH}	Green Leafhopper Population ($N_{wh} = S_{WH} + I_{WH}$)	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 planthopper recruitment rate	$\frac{1}{day}$		
α	Rice plant growth rate from vegetative to generative phase	$\frac{1}{dav}$		
eta_1	The rate of infection of rice plants in the vegetative phase	$\frac{1}{individual \times day}$		

Table 1. Definition of variables and parameters

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Variables/ Parameters	Definition	Unit	
β_2	The infection rate of rice plants in the	1	
	generative phase	individual × day	
γ_1	Green planthopper infection rate	1	
	when taking food from infected rice	individual × dav	
	plants in the vegetative phase	ý	
γ_2	Green planthopper infection rate	1	
	when taking food from infected rice	individual × day	
	plants in the generative phase	r	
μ_P	Rice plant death rate	1	
		day	
μ_I	Green planthopper natural death rate	1	
		day	

3.2. Equilibrium Point

Based on model (1), we have two equilibrium point there are the disease-free equilibrium point (E_{df}) and the endemic equilibrium point (E_{end})

$$E_{df} = \left((S_{V})_{df}, (I_{V})_{df}, (S_{g})_{df}, (I_{g})_{df}, (S_{WH})_{df}, (I_{WH})_{df} \right) = \left(\frac{\lambda}{\alpha + \mu_{p}}, 0, \frac{\lambda \alpha}{\mu_{p}(\alpha + \mu_{p})}, 0, \frac{\omega}{\mu_{l}}, 0 \right)$$
(2)

$$(S_{V})_{df} = \frac{\lambda}{\alpha + \mu_{p}}, (I_{V})_{df} = 0, (S_{g})_{df} = \frac{\lambda \alpha}{\mu_{p}(\alpha + \mu_{p})}, (I_{g})_{df} = 0, (S_{WH})_{df} = \frac{\omega}{\mu_{l}}, (I_{WH})_{df} = 0$$

$$E_{end} = \left((S_{V})_{end}, (I_{V})_{end}, (S_{g})_{end}, (I_{g})_{end}, (S_{WH})_{end}, (I_{WH})_{end} \right)$$
(3)

$$(S_{V})_{end} = \frac{\lambda}{\alpha + \beta_{1}I_{WH} + \mu_{p}}, (I_{V})_{end} = \frac{\lambda \beta_{1}I_{WH}}{\mu_{p}(\alpha + \beta_{1}I_{WH} + \mu_{p})}, (S_{g})_{end} = \frac{\lambda \alpha}{A}, (I_{g})_{end} = \frac{\lambda \alpha \beta_{2}I_{WH}}{\mu_{p}A}$$
(3)

$$(S_{WH})_{end} = \frac{\omega \mu_{p}A}{A\mu_{l}\mu_{p} + \lambda I_{WH}(I_{WH}\beta_{1}\beta_{2}\gamma_{1} + \alpha\beta_{2}\gamma_{2} + \mu_{p}\beta_{1}\gamma_{1})}$$
with $A = I_{WH}^{2}\beta_{1}\beta_{2} + \alpha\beta_{2}I_{WH} + I_{WH}\beta_{1}\mu_{p} + I_{WH}\mu_{p}\beta_{2} + \alpha\mu_{p} + \mu_{p}^{2}$ (4)

3.3. The Construction of Matrix Model

The first step in constructing the model to be a matrix is the linearization process. The linearization process is the process of forming the Jacobian matrix of the system. The Jacobian matrix was divided into a non-infectious (a disease-free) Jacobian matrix and an infective Jacobian matrix.

The noninfective Jacobian matrix of the tungro disease spread model is as follows.

$$J(df) = \begin{pmatrix} -\alpha - \mu_p & 0 & 0 & 0 & 0 & -\beta_1(S_V)_{df} \\ 0 & -\mu_p & 0 & 0 & 0 & \beta_1(S_V)_{df} \\ \alpha & 0 & -\mu_p & 0 & 0 & -\beta_2(S_g)_{df} \\ 0 & 0 & 0 & -\mu_p & 0 & \beta_2(S_g)_{df} \\ 0 & -\gamma_1(S_{WH})_{df} & 0 & -\gamma_2(S_{WH})_{df} & -\mu_I & 0 \\ 0 & \gamma_1(S_{WH})_{df} & 0 & \gamma_2(S_{WH})_{df} & \gamma_1(I_V)_{df} + \gamma_2(I_g)_{df} & -\mu_I \end{pmatrix}.$$
 (5)

The infective Jacobian matrix of the tungro disease spread model is as follows.

$$J(end) = \begin{pmatrix} -\alpha - \beta_1 I_{WH} - \mu_p & 0 & 0 & 0 & 0 & -\beta_1 (S_V)_{end} \\ \beta_1 I_{WH} & -\mu_p & 0 & 0 & 0 & \beta_1 (S_V)_{end} \\ \alpha & 0 & -\beta_2 I_{WH} - \mu_p & 0 & 0 & -\beta_2 (S_g)_{end} \\ 0 & 0 & \beta_2 I_{WH} & -\mu_p & 0 & \beta_2 (S_g)_{end} \\ 0 & -\gamma_1 (S_{WH})_{end} & 0 & -\gamma_2 (S_{WH})_{end} -\gamma_1 (I_V)_{end} - \gamma_2 (I_g)_{nd} - \mu_I & 0 \\ 0 & \gamma_1 (S_{WH})_{end} & 0 & \gamma_2 (S_{WH})_{end} & \gamma_1 (I_V)_{end} + \gamma_2 (I_g)_{end} & -\mu_I \end{pmatrix}.$$
(6)

From matrik (5) we partition of matrix the matrix to be F - V as follows.

$$F - V = \begin{bmatrix} -\mu_P & 0 & \beta_1 (S_V)_{df} \\ 0 & -\mu_P & \beta_2 (S_g)_{df} \\ \gamma_1 (S_{WH})_{df} & \gamma_2 (S_{WH})_{df} & -\mu_I \end{bmatrix}$$
(7)

$$F = \begin{bmatrix} 0 & 0 & \beta_1(S_V)_{df} \\ 0 & 0 & \beta_2(S_g)_{df} \\ \gamma_1(S_{WH})_{df} & \gamma_2(S_{WH})_{df} & 0 \end{bmatrix} and -V = \begin{bmatrix} -\mu_p & 0 & 0 \\ 0 & -\mu_p & 0 \\ 0 & 0 & -\mu_l \end{bmatrix}.$$
 (8)

$$F = \begin{bmatrix} 0 & 0 & \frac{\beta_1 \lambda}{\alpha + \mu_p} \\ 0 & 0 & \frac{\beta_2 \lambda \alpha}{(\alpha + \mu_p)\mu_p} \\ \frac{\gamma_1 \omega}{\mu_I} & \frac{\gamma_2 \omega}{\mu_I} & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \mu_p & 0 & 0 \\ 0 & \mu_p & 0 \\ 0 & 0 & \mu_I \end{bmatrix}$$
(9)

Where

 F_e is the transmission matrix at the disease-free equilibrium point V_e is the transition matrix at the disease-free equilibrium point. We define that |V| is determinant of V, where :

$$|V| = \mu_p \begin{vmatrix} \mu_p & 0 \\ 0 & \mu_I \end{vmatrix} = \mu_p (\mu_p \mu_I) = \mu_p^2 \mu_I$$
(10)

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu_{P}} & 0 & 0\\ 0 & \frac{1}{\mu_{P}} & 0\\ 0 & 0 & \frac{1}{\mu_{I}} \end{bmatrix}$$
(11)

From matrix (6) we partition the matrix into a matrix $F_e - V_{e_e}$ as follows

$$F_{e} - V_{e} = \begin{vmatrix} -\mu_{P} & 0 & \beta_{1}(S_{V})_{end} \\ 0 & -\mu_{P} & \beta_{2}(S_{g})_{end} \\ \gamma_{1}(S_{WH})_{nd} & \gamma_{2}(S_{WH})_{end} & -\mu_{I} \end{vmatrix}$$
(12)

$$F_{e} = \begin{bmatrix} 0 & 0 & \beta_{1}(S_{V})_{end} \\ 0 & 0 & \beta_{2}(S_{g})_{end} \\ \gamma_{1}(S_{WH})_{end} & \gamma_{2}(S_{WH})_{end} & 0 \end{bmatrix} \text{and } V_{e} = \begin{bmatrix} \mu_{p} & 0 & 0 \\ 0 & \mu_{p} & 0 \\ 0 & 0 & \mu_{I} \end{bmatrix}.$$
(13)

Where

 F_e is the transmission matrix at the disease equilibrium point

 V_e is the transition matrix at the disease equilibrium point

The transition matrix of the disease equilibrium equal to the transition matrix the disease free equilibrium point, as in equation (14)

$$V_{e} = \begin{bmatrix} \mu_{P} & 0 & 0\\ 0 & \mu_{P} & 0\\ 0 & 0 & \mu_{I} \end{bmatrix} \operatorname{dan} V = \begin{bmatrix} \mu_{P} & 0 & 0\\ 0 & \mu_{P} & 0\\ 0 & 0 & \mu_{I} \end{bmatrix}$$
(14)

3.4. Matrix Analysis

Based on definition 1, from the matrix (F - V) obtained $(F - V) \in M_E^{3x3}$ with the following process.

$$(F - V)_{ij} \ge 0; \forall i, j (\ne i) \in \bar{p} \quad \bar{p} = \{1, 2\}$$

$$(F - V)_{12} = 0, \quad (F - V)_{13} = \frac{\beta_1 \lambda}{\alpha + \mu_P} > 0,$$

$$(F - V)_{21} = 0 \quad (F - V)_{23} = \frac{\beta_2 \lambda}{(\alpha + \mu_P)\mu_P} > 0, \quad (F - V)_{31} = \frac{\gamma_1 \omega}{\mu_I} > 0, \quad (F - V)_{32} = \frac{\gamma_2 \omega}{\mu_I} > 0$$

Based on definition 1, from the matrix V obtained $-V \in M_E^{3\times 3}$ with the following process.

$$(-V)_{ij} \ge 0; \forall i, j (\ne i) \in \bar{p} \quad \bar{p} = \{1,3\}$$
$$(-V)_{12} = 0, \ (-V)_{13} = 0 \quad (-V)_{21} = 0 \quad (-V)_{23} = 0 \quad (-V)_{31} = 0 \quad (-V)_{32} = 0$$

Based on definition 2, from the matrix V^{-1} obtained $V^{-1} > 0$, with the following process.

$$V^{-1}_{ij} \ge 0; \forall_i \in p, \forall_j \in q. \quad p = q = \{1,3\}$$
$$V^{-1}_{11} = \frac{1}{\mu_P} > 0, V^{-1}_{12} = 0, V^{-1}_{13} = 0 \quad V^{-1}_{21} = 0, V^{-1}_{22} = \frac{1}{\mu_P} > 0, V^{-1}_{23} = 0,$$
$$V^{-1}_{31} = 0, \quad V^{-1}_{32} = 0, V^{-1}_{33} = \frac{1}{\mu_I} > 0,$$

Based on definition 3 from the matrix *F* obtained $F \ge 0$, with the following process.

$$\begin{aligned} F_{ij} &\geq 0; \forall_i \in \bar{p}, \forall_j \in \bar{q}. \ \bar{p} = \bar{q} = \{1,3\} \\ F_{11} &= 0, \ F_{12} = 0 \ F_{13} = \frac{\beta_1 \lambda}{\alpha + \mu_P} > 0 \ F_{21} = \frac{\beta_2 \nu}{\mu} > 0, \\ F_{22} &= 0 \ F_{23} = \frac{\beta_1 \lambda}{(\alpha + \mu_P)\mu_P} > 0, \\ F_{31} &= \frac{\gamma_1 \omega}{\mu_I} > 0, \\ F_{32} &= 0 \end{aligned}$$

Based on definition 3 from the matrix F_e obtained $F_e \ge 0$, with the following process $(F_e)_{ii} \ge 0; \forall_i \in \overline{p}, \forall_i \in \overline{q}, \overline{p} = \overline{q} = \{1,3\}.$

$$(F_{e})_{11} = 0, \quad (F_{e})_{12} = 0 \quad (F_{e})_{13} = \beta_{1}(S_{v})_{end} > 0 \quad (F_{e})_{21} = 0, \quad (F_{e})_{22} = 0 \quad (F_{e})_{23} = \beta_{2}(S_{g})_{end} > 0, \quad (F_{e})_{31} = \gamma_{1}(S_{wh})_{end} > 0, \quad (F_{e})_{32} = \gamma_{2}(S_{wh})_{end} > 0, \quad (F_{e})_{33} = 0$$

$$\frac{(S_{v})_{end}}{(S_{v})_{df}} = \frac{\alpha + \mu_{p}}{(\alpha + \mu_{p}) + \beta_{1}I_{wH}} \Leftrightarrow (S_{v})_{nd} = (S_{v})_{df} \quad \frac{\alpha + \mu_{p}}{(\alpha + \mu_{p}) + \beta_{1}I_{wH}} > 0$$

$$\frac{(S_{g})_{end}}{(S_{g})_{df}} = \frac{(\alpha + \mu_{p})\mu_{p}}{A} \Leftrightarrow (S_{g})_{end} = (S_{g})_{df} \cdot \frac{(\alpha + \mu_{p})\mu_{p}}{A} > 0$$

$$\frac{(S_{wH})_{end}}{(S_{wH})_{df}} = \frac{\mu_{I}\mu_{p}A}{\mu_{I}\mu_{p}A + \lambda I_{wH}(I_{wH}\beta_{1}\beta_{2}\gamma_{1} + \alpha\beta_{2}\gamma_{2} + \mu_{p}\beta_{1}\gamma_{1})} \Leftrightarrow (S_{wH})_{end} = (S_{wH})_{df} \cdot \frac{\mu_{I}\mu_{p}A + \lambda I_{wH}(I_{wH}\beta_{1}\beta_{2}\gamma_{1} + \alpha\beta_{2}\gamma_{2} + \mu_{p}\beta_{1}\gamma_{1})}{(S_{wH})_{end}} > 0$$

3.5. Basic Reproduction Number (R_0)

Matrix F and matrix (V) define the next generation matrix, i.e matrix FV^{-1} . The maximum modulus FV^{-1} is a relevant parameter to characterize the stability of the infective compartment and determine the disease reproduction number or basic reproduction number (R_0). The basic reproduction number (R_0) associated with the maximum eigenvalues of the auxiliary matrix (FV^{-1}), which coincides with its spectral radius. Next written:

$$R_0 = \rho \left(FV^{-1} \right) \tag{15}$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \beta_1 \frac{\lambda}{(\alpha + \mu_P)\mu_I} \\ 0 & 0 & \beta_2 \frac{\lambda\alpha}{(\alpha + \mu_P)\mu_I\mu_P} \\ \frac{\gamma_1 \omega}{\mu_I \mu_P} & \frac{\gamma_2 \omega}{\mu_I \mu_P} & 0 \end{bmatrix}$$
(16)

The eigenvalues of the matrix (FV^{-1}) are

$$q_1 = 0 \text{ and } q_{2,3} = \pm \frac{\sqrt{(\alpha + \mu_p)\lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_l)}}{(\alpha + \mu_p)\mu_l\mu_p}.$$
(17)

Basic reproduction number (R_0) is obtained from the spectral radius of the FV^{-1} matrix, so that

$$R_0 = \rho(FV^{-1}) = \frac{\sqrt{(\alpha + \mu_p)\lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_l)}}{(\alpha + \mu_p)\mu_l\mu_p}.$$
(18)

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3.6. The Construction Matrix $F(R_0)$ and Matrix $F_e(R_0)$

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The construction matrix $F = F(R_0)$, $F_e = F_e(R_0)$, with $F = F(R_0) \ge 0$ and $F_e = F_e(R_0) \ge 0$

$$F = F(R_0) = (1 - R_0).B.\begin{bmatrix} 0 & 0 & \beta_1 \lambda \mu_1 \mu_p \\ 0 & 0 & \beta_2 \lambda \mu_1 \\ \gamma_1 \omega \mu_p (\alpha + \mu_p) & \gamma_2 \omega \mu_p (\alpha + \mu_p) & 0 \end{bmatrix}$$
(19)

with
$$B = \frac{\left[(\alpha + \mu_p)\mu_I\mu_p + \sqrt{(\alpha + \mu_p)\lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_P)}\right]}{(\alpha + \mu_p)\left[(\mu_I\mu_p)^2(\alpha + \mu_p) - \lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_P)\right]}$$
(20)

$$F = F(R_0) \geq 0$$

so that obtained $R_0 = \rho(FV^{-1}) < 1$ with $\frac{\lambda \omega(\alpha \beta_2 \gamma_2 + \beta_1 \gamma_1 \mu_P)}{(\alpha + \mu_p)(\mu_I \mu_p)^2} < 1$ (21)

And we have

$$F_{e}(R_{0}) = (R_{0} - 1).C.\begin{bmatrix} 0 & 0 & \frac{(S_{v})_{end}}{(S_{v})_{df}}\beta_{1}\lambda\mu_{I}\mu_{P} \\ 0 & 0 & \frac{(S_{g})_{end}}{(S_{g})_{df}}\beta_{2}\lambda\mu_{I} \\ \frac{(S_{WH})_{end}}{(S_{WH})_{df}}\gamma_{1}\omega\mu_{P}(\alpha + \mu_{P}) & \frac{(S_{WH})_{end}}{(S_{WH})_{df}}\gamma_{2}\omega\mu_{P}(\alpha + \mu_{P}) & 0 \end{bmatrix}$$
(22)

with
$$C = \frac{\left[(\alpha + \mu_p)\mu_I\mu_p + \sqrt{(\alpha + \mu_p)\lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_p)}\right]}{\left[\lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_p) - (\alpha + \mu_p)(\mu_I\mu_p)^2\right](\alpha + \mu_p)}$$

$$F_e \ge 0, \text{ then } F_e(R_0) \ge 0$$
(23)

so that obtained
$$R_0 = \rho(FV^{-1}) > 1$$
 with $\frac{\lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_I)}{(\alpha + \mu_p)(\mu_I\mu_p)^2} > 1$ (24)

3.7. Stability Analysis

3.7.1 Stability at the disease-free equilibrium point

Based on theorem 3 concerning the local stability of the disease-free equilibrium point, the system (1) is linearized around the disease-free equilibrium point giving several conditions that fulfill the necessary and sufficient conditions for the asymptotically stable local disease-free equilibrium point, namely: $(F - V) \in M_F^{3x3}, (-V) \in M_F^{3x3}, V^{-1} > 0, F \ge 0$

and
$$\rho(FV^{-1}) < 1$$
 with $F = F(R_0) \ge 0$, where $R_0 = R_0(F) = \rho(FV^{-1})$ is the reproduction number, and $\rho(FV^{-1}) < 1$ with $(\mu_p + \alpha)(\mu_I\mu_p)^2 > \lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_P)$ or $\frac{\lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_P)}{(\mu_p + \alpha)(\mu_I\mu_p)^2} < 1$.

Proof: Based on the results of the analysis of the transition matrix and the transmission matrix at the diseasefree equilibrium point and forming the transmission matrix at the disease-free equilibrium point in the Basic Reproduction Number (R_0) , namely $F = F(R_0)$ th following results are obtained

$$F - V = \begin{bmatrix} -\mu_{p} & 0 & \frac{\beta_{1}\lambda}{\alpha + \mu_{p}} \\ 0 & -\mu_{p} & \frac{\beta_{2}\lambda}{(\alpha + \mu_{p})\mu_{p}} \\ \frac{\gamma_{1}\omega}{\mu_{l}} & \frac{\gamma_{2}\omega}{\mu_{l}} & -\mu_{l} \end{bmatrix} \in M_{E}^{3x3}, \quad -V = \begin{bmatrix} -\mu_{p} & 0 & 0 \\ 0 & -\mu_{p} & 0 \\ 0 & 0 & -\mu_{l} \end{bmatrix} \in M_{E}^{3x3}, \quad V^{-1} = \begin{bmatrix} \frac{1}{\mu_{p}} & 0 & 0 \\ 0 & \frac{1}{\mu_{p}} & 0 \\ 0 & 0 & \frac{1}{\mu_{l}} \end{bmatrix} > 0$$

$$F = \begin{bmatrix} 0 & 0 & \frac{\beta_{1}\lambda}{\alpha + \mu_{p}} \\ 0 & 0 & \frac{\beta_{2}\lambda\alpha}{(\alpha + \mu_{p})\mu_{p}} \\ \frac{\gamma_{1}\omega}{\mu_{l}} & \frac{\gamma_{2}\omega}{\mu_{l}} & 0 \end{bmatrix} \ge 0 \text{ and } R_{0} = \rho(FV^{-1}) < 1 \text{ with } \frac{\lambda\omega(\alpha\beta_{2}\gamma_{2} + \beta_{1}\gamma_{1}\mu_{p})}{(\alpha + \mu_{p})(\mu_{l}\mu_{p})^{2}} < 1$$

It is evident that the disease-free equilibrium point is locally stable asymptotic.

3.7.2 Stability at the endemic equilibrium point

Based on theorem 4 regarding the local stability of the endemic equilibrium point, the system (1) is linearized around the endemic equilibrium point giving several conditions that meet the necessary and sufficient conditions for asymptotically stable local endemic equilibrium points, namely:

 $(V_e) \in M_E^{3x3}$, is said to be a transition matrix at the endemic equilibrium point, where $(V_e) = (V) \in M_E^{3x3}$ is the stability matrix, $F_e \ge 0$, and $\rho(FV^{-1}) > 1$ with $(\mu_p + \alpha)(\mu_1\mu_p)^2 < \lambda\omega(\alpha\beta_2\gamma_2 + \beta_1\gamma_1\mu_P)$ or

 $\frac{\lambda\omega(\alpha\beta_2\gamma_2+\beta_1\gamma_1\mu_P)}{(\mu_p+\alpha)(\mu_I\mu_p)^2} > 1.$

Proof: Based on the results of the analysis of the transition matrix and the transmission matrix at the disease equilibrium point and forming the transmission matrix at the disease equilibrium point in the Basic Reproduction Number (R_0), namely $F_e = F_e(R_0)$, the following results are obtained

$$\begin{split} V_e &= V = \begin{bmatrix} \mu_P & 0 & 0\\ 0 & \mu_P & 0\\ 0 & 0 & \mu_I \end{bmatrix} \in M_E^{3x3}, F_e = \begin{bmatrix} 0 & 0 & \beta_1(S_V)_{end} \\ 0 & 0 & \beta_2(S_g)_{end} \\ \gamma_1(S_{WH})_{nd} & \gamma_2(S_{WH})_{end} & 0 \end{bmatrix} \succeq 0 \\ \text{dan } R_0 &= \rho(FV^{-1}) > 1 \text{ with } \frac{\lambda \omega(\alpha \beta_2 \gamma_2 + \beta_1 \gamma_1 \mu_I)}{(\alpha + \mu_P)(\mu_I \mu_P)^2} > 1 \end{split}$$

It is evident that the disease equilibrium point is locally stable asymptotically.

3.8. Numerical Simulation

Numerical simulations were carried out to show the population dynamics in the model (1). The population dynamics that are made include model simulation when the value of $\Re_0 < 1$ and model simulation when the value of $\Re_0 > 1$.

The dynamics of the spread of tungro disease are shown in Figures 3 and 4 with initial values and parameter values in Table 3.

Table 3 Initial Values and Parameters

	Table 5. Initial values and Latameters						
Variables/	Value		Variables/	Value			
Parameters	$(\mathfrak{R}_0 < 1)$	$(\Re_0 > 1)$	Parameters	$(\mathfrak{R}_0 < 1)$	$(\Re_0 > 1)$		
λ	100	100	α	0.7	0.7		
ω	100	100	β_1	0.0005	0.001		
S_V	500	500	β_2	0.0005	0.001		
I_V	100	100	γ_1	0.007	0.025		
S_{g}	300	300	γ_2	0.005	0.02		
I_g	100	100	μ_p	0.3	0.3		
S_{WH}	400	400	μ_I	0.7	0.7		
I_{WH}	150	150					



Figure 3. Dynamics Population of Green Leafhoppers (a) and (b) rice when Ro= 0.7968 < 1



Figure 4. Dynamics Population of Green Leafhoppers (a) and (b) rice when $R_0 = 2.208 > 1$

From Figure 3(a), it can be seen that the Green Leafhopper population is vulnerable when $R_o = 0.7968 < 1$. Therefore, from the beginning, it will continue to decline, then the next day, it will increase and tend to be constant. As for the Green Leafhopper population that was infected initially, the population increased but then continued to decline and experience extinction.

From Figure 3(b), Also, we can see the plant population in the vegetative and generative phases when $R_o = 0.7968 < 1$. Therefore, the population of susceptible rice plants in the vegetative and generative phases will decrease and become constant. The rice plants infected by the vegetative phase decrease from the beginning until it was extinct. Meanwhile, the rice plants infected by the generative phase are increasing at the beginning and are also extinct at some point. This occurs because the reduced number of infected plants causes a reduction of infection levels in susceptible rice plants.

From Figure 4(a) and Figure 4(b), it can be seen that the Green Leafhopper population as a vector didn't become extinct. It means, when 2,208 > 1, the spread of Green Leafhopper in rice plants will continue to occur.

Furthermore, the results showed that the population of rice plants infected by the generative phase was higher than the vegetative phase.

4. CONCLUSIONS

The matrix method can be used to determine the stability of the system of differential equations and the Basic Reproduction Number (R_0) on the model of the spread of tungro disease in rice plants with the vector of migrating green leafhoppers. In the analysis of the system's stability, it was found that the non-endemic and endemic equilibrium points were asymptotically stable. Numerical simulation results can strengthen the analytical results. The population of rice plants infected by the generative phase is more than by the vegetative phase.

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