

# Mathematical Model of Basal Stem Rot (BSR) Disease Spread in Oil Palm Plants

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**Abstract**—This study discusses the stability analysis of mathematical models for the spread of Basal Stem Rot (BSR) in oil palm plants. In developing this mathematical model, several assumptions are taken to obtain a model that is suitable for the spread of BSR disease. The resulting model is a system of first-order nonlinear differential equations with three variables. This research includes both analytical and numerical analysis. Analytical analysis includes determination of equilibrium points and local stability analysis, while numerical analysis is conducted using Microsoft Excel application. From this study, two equilibrium points were found with stability conditions that depend on the fulfillment of certain conditions. One important result obtained is that the equilibrium point will be locally stable if and only if  $\alpha > \mu$  and  $b > \sqrt{D}$ , where  $D$  is the discriminant of a quadratic equation. After analyzing analytically, the study continued with numerical simulations to illustrate and test the analytical results. Numerical results in the form of graphs show that the solution of the system is stable, which indicates that the disease will be endemic under certain conditions and time. This research provides a deeper understanding of the dynamics of the spread of BSR and the conditions that affect the stability of the spread of the disease. With this analysis, it is expected to contribute to efforts to control BSR disease in oil palm plants. In addition, this research also opens opportunities for the development of similar mathematical models for other plant diseases.

**Keywords**— Oil palm, Basal Stem Rot, Ganoderma sp., equilibrium point, asymptotic local stability.

## I. INTRODUCTION

Oil palm is one of the oil-producing plants with the highest productivity in the world (Barcelos et al., 2015). This is because oil palm is one of the plants with greater oil-producing productivity (tons/hectare) compared to other plants (Salsabila et al., 2022). Palm oil production contributes significantly to the economy in various countries, especially in Indonesia and Malaysia (Purnomo et al., 2020). Cultivation of oil palm plants is carried out because the processed products in the form of palm oil have high economic value with various benefits (Qaim et al., 2020). Among them is palm oil can be used in various

industries such as food, cosmetics, biofuels, and others. Apart from the various benefits mentioned earlier, palm oil is also rich in vitamin E and beta carotene which are beneficial for human health (Szulczewska-Remi et al., 2019).

In oil palm cultivation, one of the most common diseases is Basal Stem Rot (BSR). This disease is the most destructive disease in oil palm crops in Southeast Asia, causing great economic losses (Durand-Gasselin et al., 2005). In Indonesia in particular, this disease causes a potential loss of 500 million USD per year (Zakaria, 2022). BSR disease is caused by the white mold fungus *Ganoderma* sp. Early symptoms of this disease are usually not detected early because it grows very slowly and is difficult to identify (Naher et al., 2013). Initially, BSR only attacked old plants but was later found to attack young plants as well. Therefore, the disease can infect all stages of oil palm growth (Elkhateeb, 2022).

BSR disease causes oil palms to experience a reduction in healthy and productive tree stands. In addition, BSR also causes rotting of the roots and base of the trunk which results in broken, yellowed, and shrunken fronds. When initially infected by the *Ganoderma* sp. fungus, oil palm plants have no symptoms at all, but when the infection has developed to 60-70%, symptoms begin to appear, characterized by rotting at the base of the tree trunk, then causing inhibition of water and nutrient supply to all parts of the tree which causes the tree to gradually die (Aziz et al., 2021)).

Basal Stem Rot disease in oil palms is caused by the fungus *Ganoderma* sp., which is an infectious disease of soil origin (Wong et al., 2021). The disease can spread through spores and contact with roots. If the soil contains inoculum, the spread is very difficult to avoid. The main species that causes the disease is *Ganoderma boninense*, which can grow in the pH range of 3-8.5 with an optimal temperature of around 30°C. Its growth is inhibited at 15 and 35°C, and it is unable to grow at 40°C. Monoculture practices that eliminate other woody plants can increase the risk of infection in oil palms. Infection from *Ganoderma* sp. can also be associated with copper and zinc deficiencies, especially in peat soils with very low pH (Rakib et al., 2017).

To be able to predict the spread of BSR, a modeling approach is needed in order to know the extent of the spread of the disease so that steps can be taken to

overcome it. One solution approach to explain the solution of real-world problems is to model real-world problems into mathematical language. Mathematical modeling has a cycle consisting of four main stages, namely, the existence of real problems, making realistic models, making mathematical models, and determining problem solutions (Anderson et al., 2014). Mathematical modeling is widely used to solve problems in various fields such as health, economics, social fields, and agriculture (Inayah et al., 2020).

This study aims to build a mathematical model of the spread of BSR disease in oil palm plants and find its equilibrium point and stability. In developing this mathematical model, several assumptions are taken to obtain a model that is in accordance with the spread of BSR disease. The resulting model is a system of first-order nonlinear differential equations with three variables. This research includes both analytical and numerical analysis. Analytical analysis includes determination of equilibrium point and local stability analysis, while numerical analysis is conducted using *Microsoft Excel* application.

This study provides a deeper understanding of the dynamics of BSR spread and the conditions that affect the stability of the spread of the disease. With this analysis, it is expected to contribute to efforts to control BSR disease in oil palm plants. In addition, this research also opens opportunities for the development of similar mathematical models for other plant diseases, which can help in making more effective decisions and control strategies in the future. The analysis conducted not only provides a theoretical overview, but also empirical evidence through numerical simulations, thus strengthening the validity of the results obtained. Thus, an in-depth understanding of the dynamics of the spread of this disease will be very useful in efforts to improve the productivity and sustainability of oil palm cultivation.

## II. METHODS

### A. Formulation of the model

At this stage, researchers will construct a mathematical model of the spread of Basal Stem Rot disease in oil palm plants. The approach used is a modified SIR (Susceptible - Infectious - Recovered) model approach by considering several additional factors.

### B. Equilibrium Point

After formulating the model, we will find the equilibrium point, which is the point where a system of differential equations does not change over time (Xu & Jiang, 2010).

### C. Analysis of Stability

The equilibrium point that has been found will be sought for stability using Jacobian matrix eigenvalue analysis (Li & Szidarovszky, 1999).

### D. Numerical Simulation

The final step is to conduct numerical simulations of the spread of Basal Stem Rot.

## III. RESULTS AND DISCUSSION

### A. Mathematical Model

The mathematical model of disease spread in this study was developed from a modified SIR model by considering several additional factors. The assumptions for the formation of the mathematical model of the spread of Basal Stem Rot disease are:

1. Oil palm plants affected by BSR disease caused by *Ganoderma* sp. cannot be cured, but only reduce infection or increase resistance (Sujarit et al., 2020). Thus, no R (Recovered) subpopulation is used in this model.
2. Oil palms infected with BSR will always be able to infect other palms, so no subpopulation E (Exposed) is used in this mathematical model of disease spread.
3. The population of oil palms is homogeneous in terms of susceptibility to infection. That is, all plants have a similar level of risk of being infected by *Ganoderma* sp.
4. The total number of oil palm plants in the system remains constant over the observed time period. This means that no new plants were planted or removed from the population.
5. Parameters such as infection rate ( $\beta$ ), intrinsic growth rate of the fungus ( $\alpha$ ), fungal mortality rate ( $\mu$ ), natural mortality rate of oil palms ( $d$ ), and mortality rate of oil palms due to fungal infection ( $\delta$ ) are assumed to remain constant over the observed period. This ignores the natural fluctuations in these parameters over time.
6. There is no significant spatial variation in the distribution of oil palms or *Ganoderma* sp. fungi in the observed region.
7. The models are based on the assumptions of linearity and determinism, meaning that the response of the plant or fungus to infection or environment is assumed to be proportional and predictable.

Based on the assumptions that have been built, the phenomenon of BPB disease spread in oil palm plants can be made in the transfer diagram as in Figure 1.

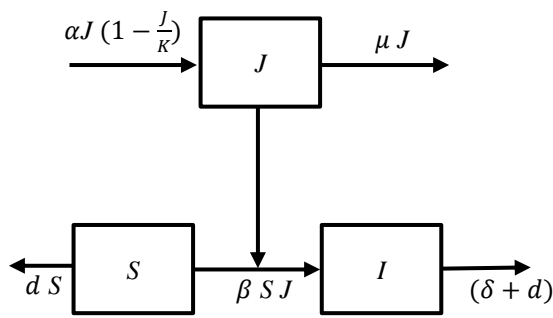


Figure 1. Transfer diagram of BSR disease spread in oil palm plants

There are three main compartments representing different individual conditions and environmental factors within the fungus-affected oil palm population.

Compartment J represents environmental factors that affect the growth and spread of fungi. Fungal growth in environmental factors is influenced by the growth rate ( $J(1 - \frac{J}{K})$ ) and fungal mortality ( $\mu J$ ). Compartment J may affect the inflow to compartments S and I due to the susceptible level of exposure to the fungus in the oil palm.

In compartment S, the influence of compartment J is included through the parameter  $\beta$ . This indicates that susceptible individuals (S) have a higher chance of being infected if the exposure to fungi in the environment (J) is higher and the number of infected individuals (I) is also high. Thus, the outflow from compartment S will depend on the level of mold exposure represented by compartment J and the number of infected individuals, as well as compartment S itself.

In compartment I, the influence of compartment J is also included through the parameter  $\beta$ . This indicates that the fungal transmission rate from an infected individual to a susceptible individual (S) depends on the level of fungal exposure in the environment (J) and the number of susceptible individuals (S). Thus, the outflow from compartment S and the inflow to compartment I will be affected by the level of fungal exposure represented by compartment J and the number of susceptible individuals (S), as well as compartment I itself.

In the transfer diagram, there are three important mortality rate parameters to consider. First, the  $d$  parameter represents the natural mortality rate of the palm, such as due to aging or environmental factors. Second, the  $\delta$  parameter reflects the mortality rate of susceptible plants due to fungal attack, indicating the direct impact of the infection. Third, the  $\mu$  parameter indicates the fungal mortality rate under environmental factors, which affect the spread and sustainability of the fungal attack.

Based on the transfer diagram in Figure 1, differential equations are derived that become a system of differential equations that represent the rate of change of each subpopulation. The differential equation system of BPB disease spread in oil palm plants can be seen in system (1):

$$\begin{aligned} \frac{dS}{dt} &= -\beta \cdot S \cdot J \cdot I - d \cdot S \\ \frac{dI}{dt} &= \beta \cdot S \cdot J \cdot I - (d + \delta) \cdot I \\ \frac{dJ}{dt} &= \alpha \cdot J \left(1 - \frac{J}{K}\right) - \mu \cdot J \end{aligned} \tag{1}$$

The variables and parameters for the mathematical model of Basal Stem Rot spread can be seen in Table 1:

Table 1. List of variables and parameters of the BSR disease spread model in oil palm plants

Variable/Parameter	Description	Units	Terms
$S(t)$	Number of oil palm plants susceptible to infection	Number of plants	$S(t) \geq 0$
$I(t)$	Number of oil palm plants infected by <i>Ganoderma</i> sp. spores.	Number of plants	$I(t) \geq 0$
$J(t)$	<i>Ganoderma</i> sp. fungus population in soil	Number of plants	$J(t) \geq 0$
$K$	Maximum environmental capacity that can sustain a fungal population	Number of plants	$K > 0$
$\beta$	Infection rate or contact rate between oil palm plants and the fungus	Individual/d ay	$\beta > 0$
$\alpha$	Intrinsic growth rate of fungi	Individual/d ay	$\alpha > 0$
$\mu$	Fungal mortality rate	Individual/d ay	$\mu > 0$
$d$	Natural mortality rate of palms	Individual/d ay	$d > 0$
$\delta$	Mortality rate of infected oil palm plants due to fungal attack	Individual/d ay	$\delta > 0$

### B. Equilibrium Point

To analyze the equilibrium, point of the model, the system (1) must be equal to zero, so it can be written as (2).

$$\begin{aligned} -\beta \cdot S \cdot J \cdot I - d \cdot S &= 0 \\ \beta \cdot S \cdot J \cdot I - (d + \delta) \cdot I &= 0 \\ \alpha \cdot J \left(1 - \frac{J}{K}\right) - \mu \cdot J &= 0 \end{aligned} \tag{2}$$

The disease-free equilibrium point is the equilibrium point when there is no disease in the population (Soares & Bassanezi, 2020). This situation will be fulfilled if none of the individuals are affected by the disease, so that  $I = 0$ . By substituting  $I = 0$  into system (2), the disease-free equilibrium point is obtained.  $E_1 = \left(0, 0, \frac{(\alpha - \mu)K}{\alpha}\right)$ .

Next, we will look for the endemic equilibrium point, which is the equilibrium point when the subpopulation of infected and dead is not equal to zero or when the disease spreads in the population. Endemic means that in the subpopulation there are always individuals infected with the disease, so we get I at the endemic equilibrium point of the disease, namely  $I^* > 0$ . The equilibrium point of the system (2) is obtained  $E_2 = (S^*, I^*, J^*)$ , with (3)

$$\begin{aligned} S^* &= \frac{d + \delta}{\beta K} \\ I^* &= \frac{d}{\beta} \\ J^* &= \frac{(\alpha - \mu)K}{\alpha} \end{aligned} \tag{3}$$

C. Analysis of Stability

To evaluate the stability of the equilibrium, point in the model, the first step is to linearize the system of equations around the equilibrium point. This process involves the use of the Jacobian matrix (J) (Iljin, 2015), which is 3x3 in size according to the given system of equations. By using the Jacobian matrix, the linear behavior of the system around the equilibrium point will be obtained. Then, by substituting the calculated equilibrium point into the linearized system of equations, the local stability evaluation is obtained. This procedure provides a better understanding of how the system will respond to small perturbations or fluctuations around the equilibrium point. The Jacobian matrix of system (4) is:

$$J = \begin{bmatrix} -\beta JI - d & -\beta S J & -\beta S I \\ \beta J I & \beta S J - (d + \delta) & -\beta S I \\ 0 & 0 & \alpha \left(1 - \frac{2J}{K}\right) - \mu \end{bmatrix} \tag{4}$$

Next, the disease-free equilibrium point  $(0, 0, \frac{(\alpha - \mu)K}{\alpha})$  is substituted into the Jacobian matrix (4), thus obtained (5)

$$J = \begin{bmatrix} -d & 0 & 0 \\ 0 & -(d + \delta) & 0 \\ 0 & 0 & -\alpha + \mu \end{bmatrix} \tag{5}$$

By forming the characteristic equation of the Jacobian Matrix (5), namely  $\det(\lambda I - J) = 0$  The eigenvalue obtained is  $\lambda_1 = -d, \lambda_2 = -(d + \delta), \lambda_3 = -\alpha + \mu$ . An equilibrium point is said to be locally asymptotically stable, if all real parts of the eigenvalues of its Jacobian matrix are negative (Stephen Wiggins, 1991). Therefore, the disease-free equilibrium point will be locally asymptotically stable if  $\alpha > \mu$ .

Next, the equilibrium point of endemic disease  $(\frac{d + \delta}{\beta K}, \frac{d}{\beta}, \frac{(\alpha - \mu)K}{\alpha})$  is substituted into the Jacobian matrix (4), thus obtained (6).

$$J = \begin{bmatrix} -\left(\frac{(\alpha - \mu)Kd}{\alpha}\right) - d & -\left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) & -\left(\frac{d(d + \delta)}{\beta K}\right) \\ \frac{(\alpha - \mu)Kd}{\alpha} & \frac{(d + \delta)(\alpha - \mu)}{\alpha} - (d + \delta) & \frac{d(d + \delta)}{\beta K} \\ 0 & 0 & -\alpha + \mu \end{bmatrix} \tag{6}$$

The eigenvalues of matrix (6) can be found by finding  $\det(\lambda I - J) = 0$ . Where:

$$\det(\lambda I - J) = \begin{vmatrix} \lambda + \left(\frac{(\alpha - \mu)Kd}{\alpha}\right) + d & -\left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) & -\left(\frac{d(d + \delta)}{\beta K}\right) \\ \frac{(\alpha - \mu)Kd}{\alpha} & \lambda - \left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) - (d + \delta) & \frac{d(d + \delta)}{\beta K} \\ 0 & 0 & \lambda - (-\alpha + \mu) \end{vmatrix}$$

$$\det(\lambda I - J) = \begin{vmatrix} \lambda + \left(\frac{(\alpha - \mu)Kd}{\alpha}\right) + d & -\left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) & -\left(\frac{d(d + \delta)}{\beta K}\right) \\ \frac{(\alpha - \mu)Kd}{\alpha} & \lambda - \left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) - (d + \delta) & \frac{d(d + \delta)}{\beta K} \\ 0 & 0 & \lambda - (-\alpha + \mu) \end{vmatrix}$$

$$\begin{aligned} &= \lambda - (-\alpha + \mu) \cdot \begin{vmatrix} \lambda + \left(\frac{(\alpha - \mu)Kd}{\alpha}\right) + d & -\left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) \\ \frac{(\alpha - \mu)Kd}{\alpha} & \lambda - \left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) - (d + \delta) \end{vmatrix} \\ &= (\lambda - (-\alpha + \mu)) \cdot \left( \left( \lambda + \left(\frac{(\alpha - \mu)Kd}{\alpha}\right) + d \right) \left( \lambda - \left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) - (d + \delta) \right) \right) + \left(\frac{(\alpha - \mu)^2(d + \delta)Kd}{\alpha^2}\right) \end{aligned}$$

One of the eigenvalues is  $\lambda_3 = -\alpha + \mu$ . The other two eigenvalues are obtained by solving the quadratic equation (7)

$$\begin{aligned} &\left( \lambda + \left(\frac{(\alpha - \mu)Kd}{\alpha}\right) + d \right) \left( \lambda - \left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) - (d + \delta) \right) \\ &\quad + \left(\frac{(\alpha - \mu)^2(d + \delta)Kd}{\alpha^2}\right) \\ &= \left( \lambda^2 + \left(\frac{(\alpha - \mu)Kd}{\alpha}\right) + d - \left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) - (d + \delta) \right) \lambda \\ &\quad - \left(\frac{(\alpha - \mu)Kd}{\alpha}\right) + d \left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) - (d + \delta) \left(\frac{(\alpha - \mu)^2(d + \delta)Kd}{\alpha^2}\right) \\ &= \lambda^2 + \left(\frac{(\alpha - \mu)Kd}{\alpha}\right) + d - \left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) - (d + \delta) \left(\frac{(\alpha - \mu)Kd}{\alpha}\right) + d \left(\frac{(d + \delta)(\alpha - \mu)}{\alpha}\right) - (d + \delta) \left(\frac{(\alpha - \mu)^2(d + \delta)Kd}{\alpha^2}\right) \\ &= 0 \end{aligned} \tag{7}$$

The quadratic equation (7) can be solved by the ABC formula, with the values of A, B, and C as:

$$\begin{aligned}
 a &= 1 \\
 b &= \frac{(\alpha - \mu)Kd}{\alpha} + d - \left( \frac{(d + \delta)(\alpha - \mu)}{\alpha} - (d + \delta) \right) \\
 &= - \left( \frac{(\alpha - \mu)Kd - (d + \delta)}{\alpha} + 2d + \delta \right) \\
 c &= - \left( \frac{(\alpha - \mu)Kd}{\alpha} + d \right) \left( \frac{(d + \delta)(\alpha - \mu)}{\alpha} - (d + \delta) \right) \\
 &\quad + \left( \frac{(\alpha - \mu)^2(d + \delta)Kd}{\alpha^2} \right) \\
 &= \frac{(d + \delta)(\mu((\alpha - \mu)Kd + ad) + (\alpha - \mu)^2Kd)}{\alpha^2}
 \end{aligned}$$

Obtained (8)

$$\lambda_{12} = \frac{- \left( \frac{(\alpha - \mu)Kd - (d + \delta)}{\alpha} + 2d + \delta \right) \pm \sqrt{D}}{2} \tag{8}$$

By  $D = \left( \frac{(\alpha - \mu)Kd - (d + \delta)}{\alpha} + 2d + \delta \right)^2 - 4 \left( \frac{(d + \delta)(\mu((\alpha - \mu)Kd + ad) + (\alpha - \mu)^2Kd)}{\alpha^2} \right)$ . The endemic disease equilibrium point is locally asymptotically stable if  $\alpha > \mu$  and  $b > \sqrt{D}$ .

**D. Model Simulation**

Simulation of the spread of BSR disease in oil palm plants is carried out using parameter values determined based on the endemic analysis of the BSR disease model. This analysis generates existence conditions and stability conditions for certain critical points. This simulation utilizes variable and parameter values, where *S* represents the number of susceptible oil palm plants, *I* represent the number of oil palm plants infected by *Ganoderma* sp. spores, and *J* is the population size of *Ganoderma* sp. fungi. Based on these parameter values and variables, simulations will be conducted on the subpopulations of *S*(*t*), *I*(*t*), and *R*(*t*).

Figure 2 shows the condition of the disease-free population. The simulation for this disease-free critical point is performed with the initial value of *S*(0) = 1000, *I*(0) = 0, and *J*(0) = 100, also parameters  $\beta = 0,005$ ,  $\alpha = 0,05$ ,  $\mu = 0,004$ ,  $d = 0,006$ ,  $\delta = 0,03$  and  $N = 1000$  which is determined based on the existence and stability conditions. Figure 2 shows that the population of susceptible plants remains constant for one hundred days because none of the plants are infected. Meanwhile, Figure 3 illustrates the population dynamics under endemic conditions. The simulation for this endemic

critical point uses the initial value of *S*(0)=999, *I*(0)=1, *J*(0)=100 with parameters  $\beta = 0,005$ ,  $\alpha = 0,05$ ,  $\mu = 0,004$ ,  $d = 0,006$ ,  $\delta = 0,03$  and  $N = 1000$  which is also determined based on the existence and stability conditions. From Figure 3, the susceptible subpopulation decreases when the infected subpopulation starts to rise. Meanwhile, fungal subpopulation in the environment tends to experience a constant increase.

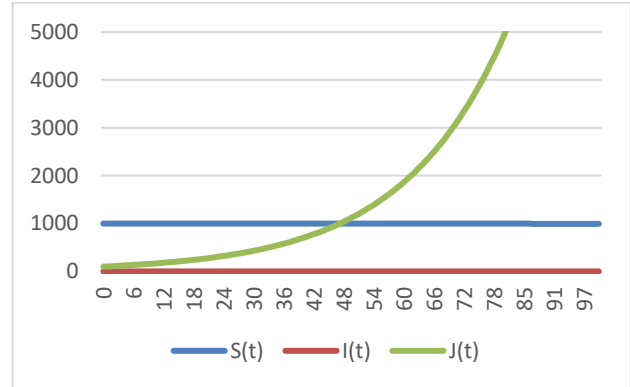


Figure 2. Disease-free state simulation curve

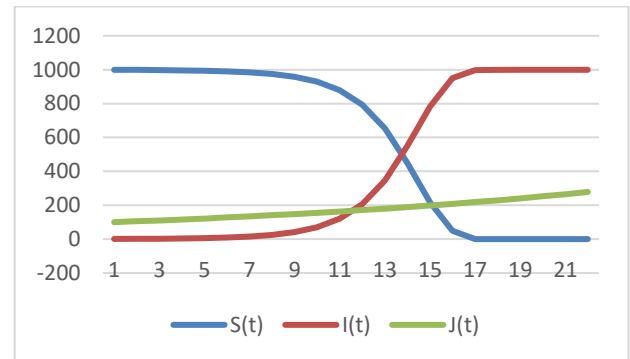


Figure 3. Simulation curve of disease endemic condition

**E. Implications of the Findings**

Based on the model simulations that have been carried out, there are findings and implications in controlling basal stem rot disease in oil palm plants. This can be seen in table 2.

Table 2. Findings and Practical Applications of Basal Stem Rot disease control

Findings	Practical Applications
<b>Early Detection and Prevention</b>	
Simulations show that if there are no infected plants ( $I(0) = 0$ ), the susceptible plants (S) remains constant.	This emphasizes the importance of measures to keep the oil palm population of population healthy. Regular monitoring and inspections can help detect early infections before they spread, so that preventive measures such as quarantine and culling of infected plants can be taken immediately.
<b>Plant Population Management</b>	
Under endemic conditions, susceptible population decreases while infected population increases.	Management strategies should include the crop rotation and planting BPB-susceptible population resistant varieties to reduce susceptible populations. Good management can stabilize populations and prevent major outbreaks.
<b>Use of Fungicides</b>	
The fungal population increases constantly under endemic conditions.	Effective fungicide applications can help control fungal populations in the environment. However, it is important to use fungicides judiciously to avoid fungal resistance and negative impacts on the environment.
<b>Policy and Protocol Development</b>	
Model parameters provide the existence and stability of the population.	This data can be used to develop evidence-based disease control policies and protocols. For example, setting the thresholds for intervention measures based on parameter values such as transmission rate ( $\beta$ ) and recovery rate ( $\alpha$ ).

#### IV. CONCLUSION

This study introduces an approach using mathematical models to understand the long-term behavior of the spread of Basal Stem Rot (BSR) in oil palm. The mathematical model developed in this study identified two important equilibrium points. The first equilibrium point describes a disease-free condition, where the population does not experience infection from the fungus *Ganoderma* sp. despite being in the oil palm environment. The second critical point describes an endemic condition, where the disease persists and transmits in the population continuously.

This study not only identifies these two equilibrium points but also ensures their existence and stability. In this context, stability means that if the population is near one of the critical points, it will tend to move towards the critical point despite controls and changes in treatment. The first equilibrium point,  $E_1$ , is the desired disease-free condition, while the second equilibrium point,  $E_2$ , is the endemic condition that reflects the continuous disease transmission in the population.

The results of the model simulation show that the susceptible subpopulation decreases as the infected subpopulation starts to rise. Meanwhile, the fungal subpopulation in the environment tends to experience a constant increase.

With this in-depth understanding of the transmission dynamics and stability of equilibrium points, this study provides important insights for BSR control and prevention strategies in oil palm populations. Such strategies can be focused on more effective interventions by considering the equilibrium points. With this analysis, it is expected to contribute to efforts to control BSR disease in oil palm plants. In addition, this research also opens opportunities for the development of similar mathematical models for other plant diseases.

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