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Trianty Putri Blante, Jaharuddin, and Endar H. Nugrahani

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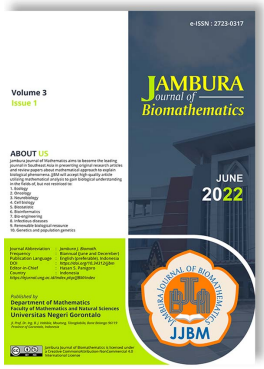

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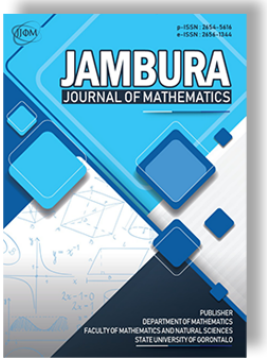

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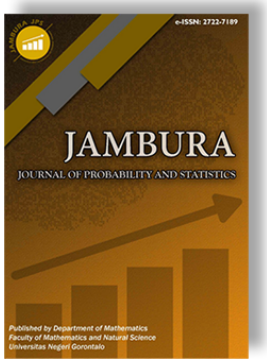

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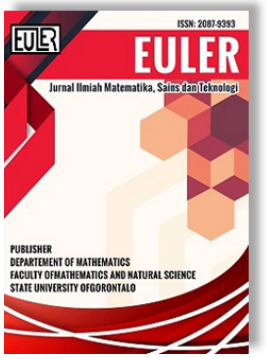

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Research Article

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Sensitivity Analysis of SI_1I_2RS Model for Dengue Fever Transmission

Trianty Putri Blante[1,*], **Jaharuddin**[2], **and Endar H. Nugrahani**[3]

[1,2,3]*Department of Mathematics, IPB University, Bogor 16680, Indonesia*

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ABSTRACT. *Dengue fever is a disease caused by dengue virus transmitted through Aedes aegypti mosquitoes. This study discusses the SI_1I_2RS epidemic model in the spread of dengue fever, assuming that people with this disease can experience severe and mild symptoms. The analysis in this research aims to determine the stability of the equilibrium point, primary reproduction number, parameter sensitivity, and numerical simulation to determine the effect of parameters on the dynamics of the spread of dengue fever. The results of this analysis show two equilibrium points, namely the disease-free equilibrium point, which is locally asymptotically stable when $R_0 < 1$ and the endemic point, which is locally asymptotically stable when $R_0 > 1$. Numerical simulations show that the change in the parameter of the average bite of individual mosquitoes in humans has a significant effect on the primary reproductive number where when the moderate acidity of individual mosquitoes in humans is 0.05 and the contact rate of disease transmission from infected mosquitoes to susceptible humans is 0.025, it can suppress the spread of dengue fever. Therefore, individuals must maintain cleanliness and take precautions against the spread of dengue fever.*

1. Introduction

Indonesia has a tropical climate with two seasons, namely, the rainy and the dry season. This is good for the growth of fauna and flora, so it is also a good place for developing diseases, especially diseases transmitted through vectors. One of the problems that arises at the change of seasons is dengue hemorrhagic fever, caused by mosquito bites [1]. The bite of the Aedes aegypti mosquito is the cause of dengue hemorrhagic fever [2, 3]. Dengue fever is a crucial problem, this is because the number of sufferers and the area where it spreads is increasing along with increasing mobility and population density [4].

Four serotypes from the Flaviviradae group cause dengue fever, which is transmitted by people infected with the Aedes aegypti mosquito through their bites. These four serotypes are DEN-1, DEN-2, DEN-3, DEN-4 [5, 6]. Severe cases of dengue fever in Indonesia are closely related to the most common virus, DEN-3, followed by other viruses such as DEN-2, DEN-1, and DEN-4 [7]. Dengue fever can appear without symptoms of fever of unknown cause, which is known as asymptomatic, namely in the form of dengue fever (DD), dengue hemorrhagic fever (DHF), or plasma leakage, which results in shock (SSD). Dengue fever sometimes has no symptoms, ranging from mild to life-threatening central nervous system disease and shock [8].

Based on the consequences resulting from the transmission of dengue fever, it is necessary to anticipate, including carrying out, mathematical studies in the form of mathematical modeling. Mathematical modeling is the process of representing and interpreting real-world problems in mathematical expressions [9–11]. One research to solve the problem of the spread of dengue fever is to study mathematical modeling and several assumptions regarding its spread. Mathematical models were used to determine the dynamics of the spread of dengue fever in previous research, namely mathematical research on the transmission of dengue fever, which was carried out by [12]. This study studied the SIR model based on the transmission rate to humans in the spread of dengue fever. [13] research assumes a decrease in human immunity to disease in the space of dengue fever. This research analyzes a model for the spread of dengue fever, a modification of the model developed by [13]. In Indonesia, dengue fever can have mild symptoms. According to [14], in West Java province, dengue fever itself has mild symptoms that sometimes cannot be seen like dengue fever. Therefore, a model emerged that involved adding subpopulations by considering the number of infected human populations with mild symptoms, namely fever/common flu. This research aims to modify the model for the spread of dengue fever by adding a compartment for the number of human populations infected with mild symptoms.

The steps in conducting this research are to determine the real-world problem to be studied, make assumptions used to construct the model, construct a modified compartmental diagram of the spread of dengue fever, construct a mathematical model of the spread of dengue fever in the form of a nonlinear differential equation, determine the disease-free equilibrium point and the disease-endemic equilibrium point, analyze the stability of the equilibrium point, determine the basic reproduction number, and simulate a modified mathematical model of the spread of dengue fever. Determine the disease-free equilibrium point

*Corresponding Author.

Email : *triantyputriblante0@gmail.com* (T. P. Blante)
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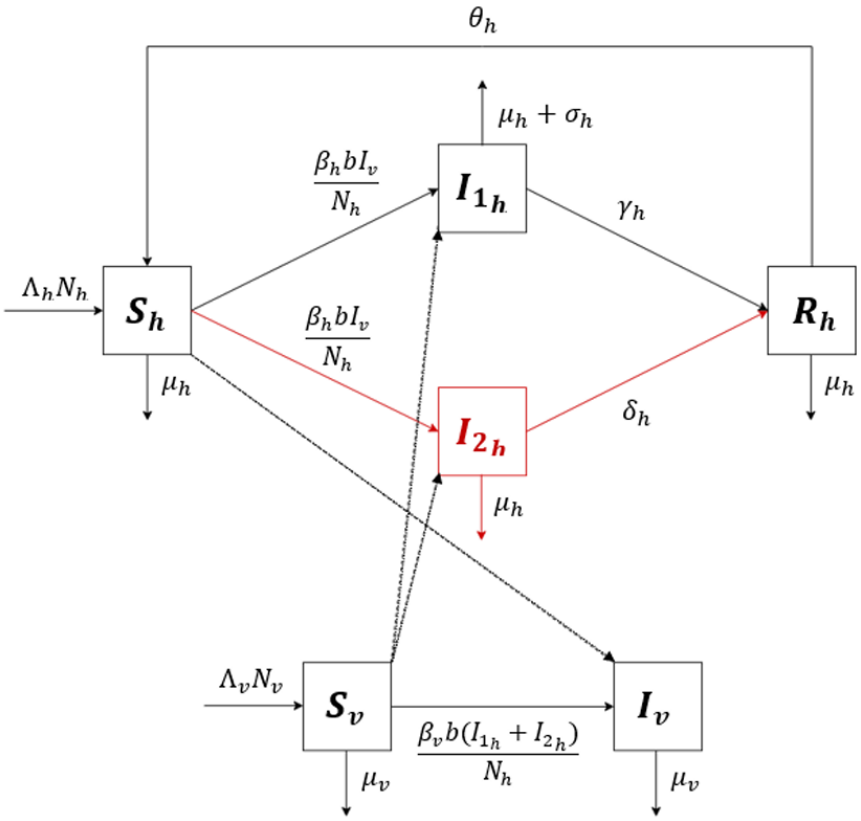

Figure 1. Compartment Diagram of The Spread of Dengue Fever

and the disease-endemic equilibrium point, analyze the stability of the equilibrium point, determine the basic reproduction number, and simulate a modified mathematical model of the spread of dengue fever.

2. Model Formulation

In this section. The SI_1I_2RS model is a modification of the SIRS model by adding the following assumptions:

1. There is no migration in each population.
2. Infected humans can be infected individuals with severe symptoms or mild symptoms.
3. The rate of infection in humans infected with severe and mild symptoms is the same.

The rate of infection in humans infected with severe and mild symptoms is the same. In this model, there are two total populations, namely, the human population and the mosquito population. The human population is divided into four subpopulations, namely the susceptible population (S_h), the infected human population with severe symptoms (I_{1_h}), the infected human population with mild symptoms (I_{1_h}), and the population that has recovered from dengue fever (R_h). The mosquito population consists of two subpopulations: the susceptible population (S_v) and the infected population (I_v). In the human population, the number of susceptible human people increases due to births at a rate $\wedge_h$ and individuals who recover from dengue fever and lose immunity at a rate θ_h, the number of infected human people with mild symptoms increases due to mosquito bites on humans and contact transmission. Disease from infected mosquitoes to susceptible humans at a rate $(\beta_h * b)$, the number of infected human populations with severe symptoms increases with mosquito bites on humans and contact transmission of disease from infected mosquitoes to susceptible humans at a rate $(\beta_h * b)$, the number of human populations that recover from dengue fever increases because there are humans who recover from dengue fever with mild symptoms at a rate of γ_h and humans who recover from dengue fever with severe symptoms at a rate of δ_h, and all subpopulations experience natural death at a rate of μ_h. The diagram of the model is given in Figure 1.

Based on the assumptions and compartment diagram from Figure 1, the following system of nonlinear differential equations is obtained:

$$\begin{aligned}
\frac{dS_h}{dt} &= \wedge_h N_h - 2\frac{\beta_h b}{N_h} I_v S_h - \mu_h S_h + \theta_h R_h,\\
\frac{dI_{1_h}}{dt} &= \frac{\beta_h b}{N_h} I_v S_h - (\mu_h + \gamma_h + \sigma_h) I_{1_h},\\
\frac{dI_{2_h}}{dt} &= \frac{\beta_h b}{N_h} I_v S_h - (\mu_h + \delta_h) I_{2_h},\\
\frac{dR_h}{dt} &= \gamma_h I_{1_h} + \delta_h I_{2_h} - (\mu_h + \theta_h) R_h,\\
\frac{dS_v}{dt} &= \wedge_v N_v - \frac{\beta_v b(I_{1_h} + I_{2_h})}{N_v} S_v - \mu_v S_v,\\
\frac{dI_v}{dt} &= \frac{\beta_v b(I_{1_h} + I_{2_h})}{N_h} S_v - \mu_v I_v.
\end{aligned} \tag{1}$$

The system of eq. (1) can be simplified by defining the following variables: $s_h = \frac{S_h}{N_h}, i_{1_h} = \frac{I_{1_h}}{N_h}, i_{2_h} = \frac{I_{1_h}}{N_h}, s_v = \frac{S_v}{N_v}, i_v = \frac{I_v}{N_v}, n = \frac{N_v}{N_h}$.

Based on these new variables, eq. (1) becomes:

$$\begin{aligned}
\frac{ds_h}{dt} &= \wedge_h - 2\beta_h b n i_v s_h - \mu_h s_h + \theta_h r_h,\\
\frac{di_{1_h}}{dt} &= \beta_h b n i_v s_h - (\mu_h + \gamma_h + \sigma_h) i_{1_h},\\
\frac{di_{2_h}}{dt} &= \beta_h b n i_v s_h - (\mu_h + \delta_h) i_{2_h},\\
\frac{dr_h}{dt} &= \gamma_h i_1 h + \delta_h i_2 h - (\mu_h + \theta_h) r_h,\\
\frac{ds_v}{dt} &= \wedge_v - \beta_v b (i_{1_h} + i_{2_h}) s_v - \mu_v s_v,\\
\frac{di_v}{dt} &= \beta_v b (i_{1_h} + i_{2_h}) s_v - \mu_v i_v.
\end{aligned} \tag{2}$$

3. Results and Discussion

3.1. Equilibrium Point

The dynamic system of the spread of dengue fever is expressed in the system of eq. (2) has two equilibrium points. They are the disease-free equilibrium point and the endemic equilibrium point. The disease-free equilibrium point is obtained when $I_h = 0$, $I_v = 0$, and the others are not equal to zero, so the obtained is $T^0 = (s_h^0, 0, 0, 0, 0, s_v^0, 0) = \left(\frac{\wedge_h}{\mu_h}, 0, 0, 0, \frac{\wedge_v}{\mu_v}, 0\right)$ and the endemic equilibrium point is obtained when all subpopulations are not equal to zero, so the obtained is $T^* = (s_h^*, i_{1_h}^*, i_{2_h}^*, r_h^*, s_v^*, i_v^*)$ with

$$\begin{aligned}
s_h^* &= \frac{r_h^*\theta_h + \wedge_h}{2bi_v^* n\beta_h + \mu_h}, & r_h^* &= \frac{i_{1_h}^*\gamma_h + i_{2_h}^*\delta_h}{\theta_h + \mu_h},\\
i_{1_h}^* &= \frac{bi_v^* n s_h^* \beta_h}{\gamma_h + \mu_h + \sigma_h}, & s_v^* &= \frac{\wedge_v}{bi_{1_h}^*\beta_v + bi_{2_h}^*\beta_v + \mu_v},\\
i_{2_h}^* &= \frac{bi_v^* n s_h^* \beta_h}{\gamma_h + \mu_h}, & i_v^* &= \frac{b(i_{1_h}^* + i_{2_h}^*) s_v^* \beta_v}{\mu_v}.
\end{aligned}$$

3.2. Basic Reproduction Number

The basic reproduction number uses the next generation matrix method. The basic reproduction number is the dominant eigenvalue of the FV^{-1} matrix where F is the transmission matrix of new infection in the population and V is the transition matrix of individual movements between subpopulations (i_{1_h}), (i_{2_h}), and (I_v), so the basic reproduction number is obtained as follows:

$$F = \begin{pmatrix} \beta_h b n i_v s_h \\ \beta_h b n i_v s_h \\ \beta_v b (i_{1_h} + i_{2_h}) s_v \end{pmatrix},$$

$$V = \begin{pmatrix} (\mu_h + \gamma_h + \sigma_h) i_{1_h} \\ (\mu_h + \gamma_h) i_{2_h} \\ \mu_v i_v \end{pmatrix}.$$

The dominant eigenvalue of the matrix FV^{-1}, i.e the basic reproduction number, is:

$$\mathcal{R}_0 = \psi_1 \sqrt{\psi_2}, \tag{3}$$

where

$$\begin{aligned}
\psi_1 &= \frac{b}{\mu_v(\gamma_h + \mu_h + \sigma_h)},\\
\psi_2 &= \frac{n\beta_h\beta_v \wedge_h \wedge_v (\gamma_h + \mu_h + \sigma_h)(\gamma_h + \delta_h + 2\mu_h + \sigma_h)}{\mu_h(\delta_h + \mu_h)}.
\end{aligned}$$

Based on the basic reproduction number according to Theorem 1 it can be stated that if $R_0 < 1$ then the disease will disappear from the population. If $R_0 > 1$ according to Theorem 2, then the condition will escalate to an epidemic. Then, in the next section, the stability of two equilibrium points is analyzed, namely the disease-free equilibrium point T^0 and the disease-endemic equilibrium point T^*.

3.3. Equilibrium Point Stability Analysis

In this section, the stability of the disease-free equilibrium point is proven using the following theorem:

> **Theorem 1.** *The disease-free equilibrium point T^0 in the system of eq. (2) is locally asymptotic if $R_0 < 1$.*

Proof. The stability properties of the disease-free equilibrium point T^0 can be known by linearizing the system of eq. (2) around T^0. The Jacobian matrix for the disease-free equilibrium point is obtained T^0 is as follows:

$$J_{T^0} = \begin{pmatrix} -J_{11} & 0 & 0 & J_{14} & 0 & -J_{16} \\ 0 & -J_{22} & 0 & 0 & 0 & J_{26} \\ 0 & 0 & -P_{33} & 0 & 0 & J_{36} \\ 0 & J_{42} & J_{43} & -J_{44} & 0 & 0 \\ 0 & -J_{52} & -J_{53} & 0 & -J_{55} & 0 \\ 0 & J_{62} & J_{63} & 0 & 0 & -J_{66} \end{pmatrix}$$

with

$$\begin{aligned}
J_{11} &= \mu_h & J_{43} &= \delta_h\\
J_{14} &= \theta_h & J_{44} &= \delta_h + \mu_h\\
J_{16} &= \frac{2bn\beta_h \wedge_h}{\mu_h} & J_{52} &= \frac{b\beta_v \wedge_v}{\mu_v}\\
J_{22} &= \gamma_h + \mu_h + \sigma_h & J_{53} &= \frac{b\beta_v \wedge_v}{\mu_v}\\
J_{26} &= \frac{2bn\beta_h \wedge_h}{\mu_h} & J_{55} &= \mu_v\\
J_{33} &= \delta_h + \mu_h & J_{62} &= \frac{b\beta_v \wedge_v}{\mu_v}\\
J_{36} &= \frac{bn\beta_h \wedge_h}{\mu_h} & J_{63} &= \frac{b\beta_v \wedge_v}{\mu_v}\\
J_{42} &= \gamma_h & J_{66} &= \mu_v
\end{aligned}$$

The eigenvalues of the Jacobian matrix J_{T^0} can be obtained by solving $|\lambda I - J_{T^0}| = 0$ or $(-J_{11} + \lambda)(-J_{44} + \lambda)(-J_{55} + \lambda)(-J_{36}(-J_{22}J63 + J_{63}\lambda) + ((-J_{33} + \lambda)(-J_{26}J_{62} + J_{22}J_{66} - J_{22}\lambda - J_{66}\lambda + \lambda^2) = 0$

Based on the characteristic equation above, the eigenvalues λ_1,λ_2, and λ_3 are obtained as follows: $\lambda_1 = J_{11} = -\mu_h$,$\lambda_2 = J_{44} = -\theta_h$,$\lambda_3 = J_{55} = -\mu_v$. Because, $\mu_h, \mu_v, \theta_h > 0$ we get $\lambda_1, \lambda_2, \lambda_3 < 0$. The other three eigenvalues $\lambda_4, \lambda_5, \lambda_6$ are analyzed by solving the following characteristic equation using the Routh-Hurwitz criterion:

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \tag{4}$$

with

$$\begin{aligned}
a_1 &= \gamma_h + \delta_h + 2\mu_h + \mu_v + \sigma_h,\\
a_2 &= \mu_h^2 + \mu_h\sigma_h + 2\frac{\mu_h\mu_v(\gamma_h+\mu_h+\sigma_h)(\mu_h+\delta_h)}{(\gamma_h+\mu_h+\sigma_h)(\gamma_h+\delta_h+2\mu_h+\sigma_h)}\\
&\quad + \frac{(\delta_h(\mu_h+\sigma_h))(\gamma_h+\mu_h+\sigma_h)}{(\gamma_h+\delta_h+2\mu_h+\sigma_h)} + \frac{\delta_h\mu_v(\delta_h)}{(\gamma_h+\delta_h+2\mu_h+\sigma_h)}\\
&\quad + \frac{\gamma_h\mu_v(\gamma_h+\mu_h+\mu_h+\sigma_h)}{(\gamma_h+\delta_h+2\mu_h+\sigma_h)} + \frac{\mu_v\sigma_h(\gamma_h+\mu_h+\mu_h+\sigma_h)}{(\gamma_h+\delta_h+2\mu_h+\sigma_h)}\\
&\quad + \frac{\gamma_h(\delta_h+\mu_h)(\gamma_h+\mu_h+\mu_h+\sigma_h)}{(\gamma_h+\delta_h+2\mu_h+\sigma_h)}\\
&\quad + \frac{(\delta_h(\mu_h+\sigma_h))(\delta_h+\mu_h)}{(\gamma_h+\delta_h+2\mu_h+\sigma_h)} + \frac{(\gamma_h(\delta_h+\mu_h))\delta_h}{(\gamma_h+\delta_h+2\mu_h+\sigma_h)}\\
&\quad + 2\left(\frac{\mu_v(\gamma_h+\mu_h+\sigma_h)^2(\delta_h+\mu_h)}{(\gamma_h+\mu_h+\sigma_h)(\gamma_h+\delta_h+2\mu_h+\sigma_h)}\right)(1-R_0^2),\\
a_3 &= (\mu_v(\gamma_h+\mu_h+\sigma_h)(\delta_h+\mu_h))(1-R_0^2).
\end{aligned}$$

Based on the Routh-Hurwitz criterion, an equilibrium point T^0 is stable if it satisfies $a_1 > 0, a_3 > 0$, and $a_1a_2 - a_3 > 0$. Because all parameters are positive $a_1 > 0$. Because $R_0 < 1$ then $a_3 > 0$ and $a_1a_2 - a_3 > 0$. Thus, all the Jacobian matrix $J_(T^0)$ eigenvalues are negative. So, the system of eq. (2) is locally asymptotically stable around point T^0. □

Next, the stability of the disease endemic equilibrium point is proven using the following theorem:

Theorem 2. *The endemic equilibrium point T^* in the system of eq. (2) is locally asymptotic if $\mathcal{R}_0 > 1$.*

Proof. The Castillo-Chaves and Song Theorem will be used to prove the endemic equilibrium points stability analysis. Suppose $\varphi = \beta_h$ are the bifurcation parameters and $s_h = x_1, i_1h = x_2, i_2h = x_3, r_h = x_4, s_v = x_5$, and $i_v = x_6$. So eq. (2) is taken as

$$\begin{aligned}
f_1(x_1,x_2,x_3,x_4,x_5,x_6) &= \wedge_h - 2\beta_h bnx_6x_1 - \mu_h x_1 + \theta_h x_4\\
f_2(x_1,x_2,x_3,x_4,x_5,x_6) &= \beta_h bnx_6x_1 - (\mu_h+\gamma_h+\sigma_h)x_2\\
f_3(x_1,x_2,x_3,x_4,x_5,x_6) &= \beta_h bnx_6x_1 - (\mu_h+\gamma_h)x_2\\
f_4(x_1,x_2,x_3,x_4,x_5,x_6) &= \gamma_h x_2 + \delta_h x_3 - (\mu_h+\theta_h)x_4\\
f_5(x_1,x_2,x_3,x_4,x_5,x_6) &= \wedge_v - \beta_v b(x_2+x_3)x_5 - \mu_v x_5\\
f_6(x_1,x_2,x_3,x_4,x_5,x_6) &= \beta_v b(x_2+x_3)x_5 - \mu_v x_6
\end{aligned} \tag{5}$$

If $\mathcal{R}_0 = 1$ and $\varphi = \beta_h$ the disease-free equilibrium point T^0 has five negative eigenvalues, and one zero eigenvalue is $a_4 = 0$. The zero eigenvalue has a right eigenvector $(v_1, v_2, v_3, v_4, v_5, v_6)$ and left eigenvector $(w_1, w_2, w_3, w_4, w_5, w_6)$ as follows:

Suppose $v_3 > 0$ and $w_3 > 0$ then

$$\begin{aligned}
v_1 &= \frac{J_{14}J_{42}J_{26}J_{33} + J_{14}J_{36}J_{22}J_{43} - J_{16}J_{22}J_{33}J_{44}}{J_{11}J_{36}J_{22}J_{44}}v_3,\\
v_1 &= -\frac{1}{\mu_h}bn\beta_h \wedge_h (\gamma_h\mu_h(2\delta_h+\theta_h+2\mu_h)\\
&\quad + (2\mu_h(\theta_h+\mu_h) + \delta_h(\theta_h+2\mu_h))(\mu_h+\sigma_h),\\
v_2 &= \frac{J_{26}J_{33}}{J_{36}J_{22}}v_3,\\
v_4 &= \frac{J_{42}J_{26}J_{33} + J_{36}J_{22}J_{43}}{J_{36}J_{22}J_{44}}v_3,\\
v_5 &= -(\frac{J_{52}J_{26}J_{33} + J_{36}J_{22}J_{53}}{J_{36}J_{22}J_{55}}v_3,\\
v_6 &= \frac{J_{33}}{J_{36}}v_3,\\
w_1 &= 0,\\
w_4 &= 0,\\
w_5 &= 0,\\
w_2 &= J_{22}(\frac{J_{62}J_{33}}{J_{63}}w_3),\\
w_6 &= \frac{J_{33}}{J_{63}}w_3.
\end{aligned}$$

Based on the above solution, we obtain $v_1 < 0, v_2 > 0, v_3 > 0, v_4 > 0, v_5 < 0$, and $v_6 > 0$ and $w_1 = w_5 = w_4 = 0, w_2 > 0, w_3 > 0, w_6 > 0$. Then, we obtain partial derivatives to satisfy a and b using the Castillo-Chavez and Song Theorem as follows:

$$\sum_{k,i,j=1}^{6} w_k v_i v_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(T^0,\varphi);\ \sum_{k,i=1}^{6} w_k v_i \frac{\partial^2 f_k}{\partial x_i \partial \varphi}(T^0,\varphi). \tag{6}$$

Based on the system of eq. (5), the following is obtained:

$$\begin{aligned}
\frac{\partial^2 f_1}{\partial x_1\partial x_6}(T^0,\varphi) &= \frac{\partial^2 f_1}{\partial x_6\partial x_1}(T^0,\varphi^*) = -2bn\varphi\\
\frac{\partial^2 f_2}{\partial x_1\partial x_6}(T^0,\varphi) &= \frac{\partial^2 f_2}{\partial x_6\partial x_1}(T^0,\varphi^*) = bn\varphi\\
\frac{\partial^2 f_3}{\partial x_1\partial x_6}(T^0,\varphi) &= \frac{\partial^2 f_3}{\partial x_6\partial x_1}(T^0,\varphi^*) = bn\varphi\\
\frac{\partial^2 f_5}{\partial x_2\partial x_5}(T^0,\varphi) &= \frac{\partial^2 f_5}{\partial x_5\partial x_2}(T^0,\varphi) = -\beta_v b\\
\frac{\partial^2 f_5}{\partial x_3\partial x_5}(T^0,\varphi) &= \frac{\partial^2 f_5}{\partial x_5\partial x_3}(T^0,\varphi) = -\beta_v b\\
\frac{\partial^2 f_6}{\partial x_2\partial x_5}(T^0,\varphi) &= \frac{\partial^2 f_6}{\partial x_5\partial x_2}(T^0,\varphi) = \beta_v b\\
\frac{\partial^2 f_6}{\partial x_3\partial x_5}(T^0,\varphi) &= \frac{\partial^2 f_6}{\partial x_5\partial x_3}(T^0,\varphi) = \beta_v b\\
\frac{\partial^2 f_2}{\partial x_6\partial \varphi}(T^0,\varphi) &= \frac{b\wedge_h}{\mu_h}.
\end{aligned}$$

Based on the system of eq. (6), the values for a and b are obtained as follows:

$$\begin{aligned}
a &= 2v_1v_6bn\varphi^*(w_2+w_3) + 2w_6v_5\beta_v b(v_2+v_3),\\
b &= w_2v_6\frac{bn\wedge_h}{\mu_h}.
\end{aligned} \tag{7}$$

Based on the Castillo-Chavez and Song Theorem, $a < 0$ and $b > 0$ are obtained so that case 4 is satisfied. As a result, when φ changes from $\varphi < \varphi(\mathcal{R}_0 < 1)$ to $\varphi > \varphi(\mathcal{R}_0 > 1)$ then the stability of the point remains endemic to T^* changing from negative to positive is locally asymptotically stable. So, it is proven that if $\mathcal{R}_0 > 1$, then the stability of the endemic equilibrium point T^* is locally asymptotically stable. □

3.4. Numerical Simulation

Numerical simulations on the modified model were carried out using parameters obtained in several previous studies regarding the spread of dengue fever. The simulation is

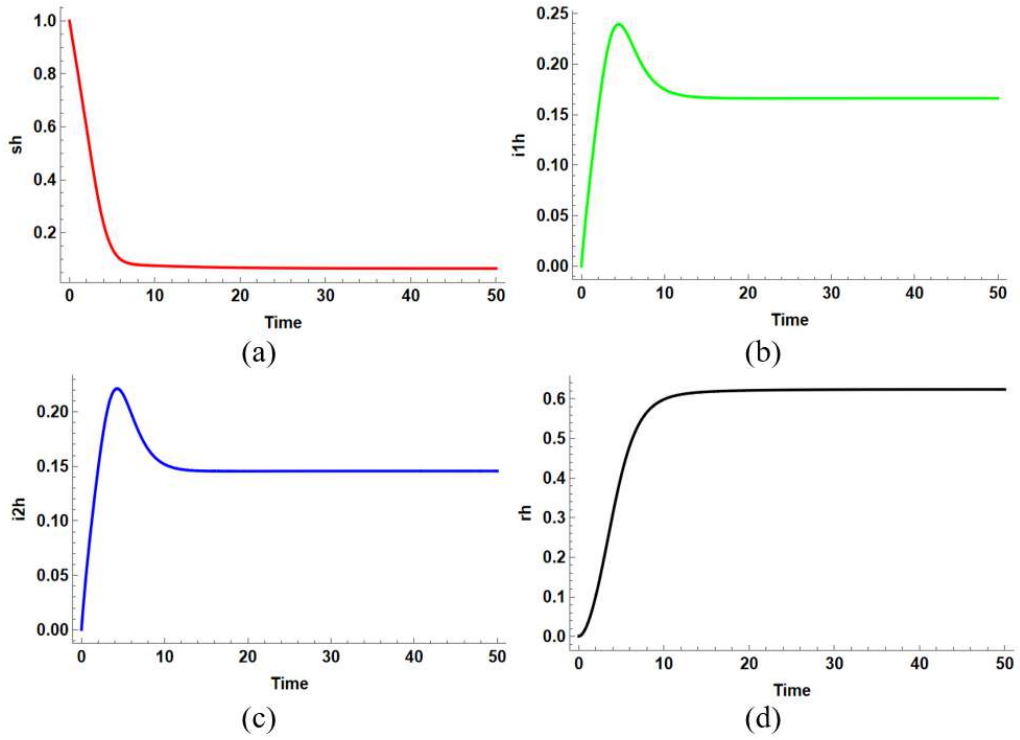

Figure 2. Population dynamics under conditions $\mathcal{R}_0 > 1$

carried out by determining the initial values for each population, namely $s_h(0) = 0.99958$,$i_{1_h}(0) = 0.00004339$,$i_{2_h}(0) = 0.00038243$,and $r_h(0) = 0.00045474$. by using the parameter values in Table 1.

Table 1. Parameter Values Used in the Model

Parameter	Value	Source
$\wedge_h$	0.000045	[12]
μ_h	0.000045	[12]
σ_h	0.00001125	Assumed
γ_h	0.328833	[12]
δ_h	0.375000	Assumed
θ_h	0.175000	Assumed
$\wedge_v$	0.07142	[15]
μ_v	0.07142	[15]
b	0.5	[16]
β_v	0.375000	[12]
β_h	0.750000	[17]
n	5	Assumed

Based on Table 1, the numerical simulation curve given in Figure 2. Figure 2 shows the dynamics of human subpopulations where each population will move towards the endemic equilibrium point T^* or its condition is stable around the equilibrium point T^*. At first, the population of i_{1_h}, i_{2_h} rose sharply, then fell and stabilized towards their respective equilibrium points. The population r_h increases sharply, then lowers and stabilizes towards an equilibrium point. The population s_h will decrease sharply and then stabilize towards an equilibrium point. The basic reproduction number using the dengue fever number is $R_0 = 7.49769 > 1$, which means that dengue fever is endemic.

In the next section, this sensitivity analysis uses local methods because of limited parameter space, data limitations because using global methods requires complete and accurate data on all input variables, which may be difficult to obtain or require additional estimates, calculation is more computationally efficient because it requires only derivative calculations. partial work around a particular working point, rather than calculations for all combinations of input variables.

3.5. Dynamics of Human Population with Changes in Average Parameters of Individual Mosquito Bites on Humans (b)

The sensitivity index for the average parameter of individual mosquito bites on humans (b) is positive, meaning that there is a decrease in the value of the basic reproduction number $\mathcal{R}_0$ if this parameter is reduced, and vice versa. Changes in the $\mathcal{R}_0$ value can be seen in Table 2 as follows:

Table 2. The effect of changing the value of parameter b on the $\mathcal{R}_0$ value

Parameter Value b	$\mathcal{R}_0$ Value
0.5	5.30167
0.2	2.12067
0.01	1.06033
0.08	0.848267
0.05	0.530167

The solution field that describes the dynamics of each population due to changes in the b value can be seen in Figure 3. Figure 3 shows that if the b value is increased, the proportion of the human population will also increase. Theorem 1 and Theorem 2 explain how a system is said to experience an outbreak. In Figure $5(a)$, susceptible individuals still exist and are experiencing an episode or have not yet achieved disease freedom when $b = 0.5$ (red line), $b = 0.2$ (blue line), and $b = 0.1$ (green line) on day 150 will stabilize towards its equilibrium point. When b is lowered again to 0.08, the disease decreases, and on the 50th day, it will

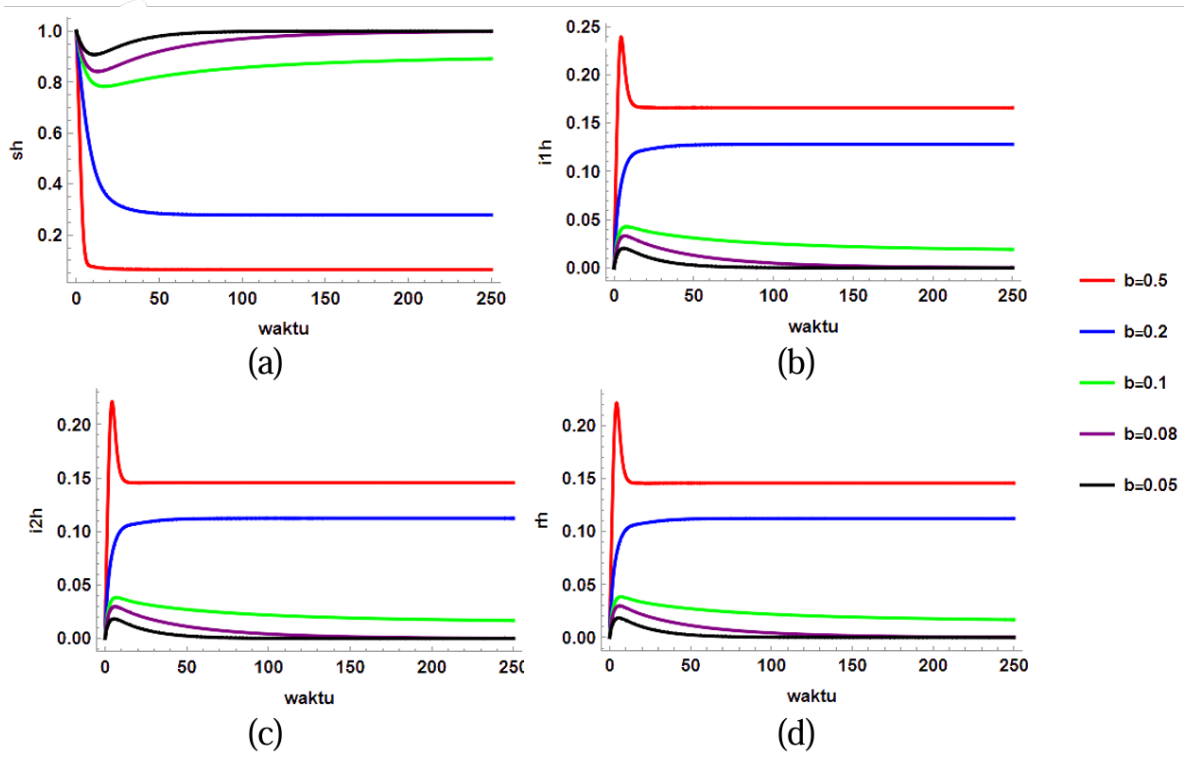

Figure 3. Dynamics of human population with changes in b values

stabilize to its equilibrium point and can be disease-free. When b was lowered again to 0.005, the condition reached a disease-free state more quickly on the 10th day. In Figure $3(b) - 3(c)$, where infected individuals still exist, it can be seen that both the infected population with severe symptoms and the infected population with mild symptoms experienced an outbreak or had not yet achieved disease freedom when $b = 0.5$ (red line) on the 10th day it will stabilize towards its fixed point when $b = 0.2$ (blue line) on the 15th day it will stabilize towards its equilibrium point when $b = 0.1$ (green line) on the 30th day it will stabilize towards its equilibrium point and is still in a state of disease experiencing an outbreak. When b was reduced to 0.08 (purple line) on the 140th day, it had succeeded in reaching a disease-free state. When b was further reduced to 0.05 (black line), the condition came a disease-free state more quickly on the 60th day. Figure $3(d)$, where the recovered individual shows that when b is 0.5 (red line), it will stabilize towards its equilibrium point on day ten but is still experiencing an outbreak or has not yet reached a disease-free state when $b = 0.2$ (blue line) the graph will rise and stabilize on the 10th day and will stabilize at its equilibrium point. When b is reduced to 0.1 (green line) on day 150, it will stabilize towards its equilibrium point and begin approaching a disease-free state. When b is reduced again to a value of 0.08 (purple line), on the 100th day, it has reached a disease-free state and is stable towards its equilibrium point. When $b = 0.05$ (black line), the disease state becomes disease-free more quickly on day 50 and stabilizes towards an equilibrium point. Figure 3 shows that the smaller the β_h value given, the graph will start to decline, and dengue fever will reach a disease-free state. In this case, dengue fever will be disease-free when b is 0.08 and 0.05, with $\mathcal{R}_0$ being 0.848267 and 0.530167.

3.6. Dynamics of Human Population with Changes in Disease Contact Rate Parameters from Infected Mosquitoes to Susceptible Humans (β_h)

The sensitivity index for the average parameter of individual mosquito bites on humans (β_h) is positive, meaning that there is a decrease in the value of the basic reproduction number $\mathcal{R}_0$ if this parameter is reduced, and vice versa. Changes in the $\mathcal{R}_0$ value can be seen in Table 3.

Table 3. The effect of changing the value of parameter b on the $\mathcal{R}_0$ value

Parameter Value β_h	$\mathcal{R}_0$ Value
0.75	5.30167
0.45	4.10665
0.15	2.37098
0.045	1.29864
0.025	0.967948

Based on Table 3, the solution field that describes the dynamics of each population due to changes in β_h values can be seen in Figure 4. Figure 4 shows that if the β_h value is increased, the proportion of the human population will increase. Theorem 1 and Theorem 2 explain how a system is said to experience an outbreak. In Figure $4(a)$, susceptible individuals still exist and experience an episode or have not yet reached disease-free when $\beta_h = 0.75$ (red line), $\beta_h = 0.45$ (blue line), on day 100, it will stabilize towards the equilibrium point. When $beta_h = 0.15$, the graph will decline and stabilize towards its equilibrium point on the 30th day. When β_h is reduced again to 0.045 on day 20, it will stabilize towards its equilibrium point but become an outbreak and approach a disease-free state. When β_h seven was further reduced to 0.025, the condition reached a disease-free state more quickly on the 10th day. In Figure $4(b) - 4(c)$, where infected

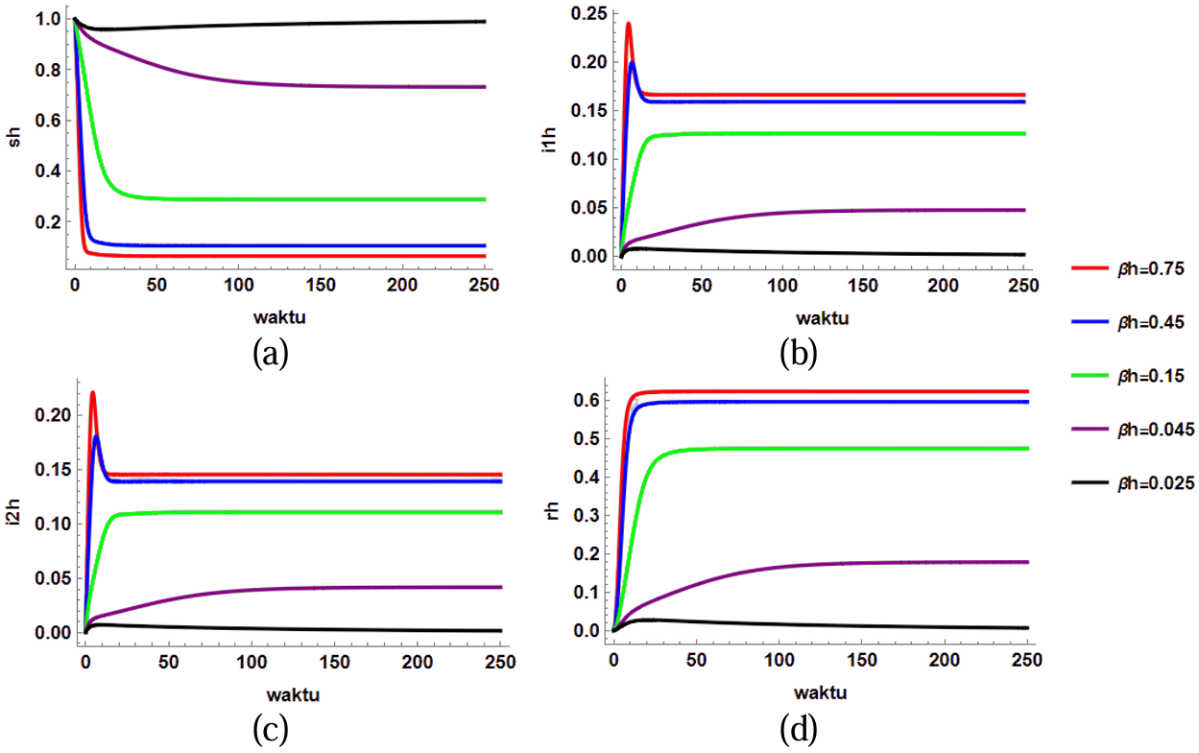

Figure 4. Dynamics of human population with changes in β_h values

individuals still exist, it can be seen that both the infected population with severe symptoms and the infected population with mild symptoms experienced an outbreak or had not yet achieved disease freedom when $\beta_h = 0.75$ (red line) on the 20th day it will stabilize towards its equilibrium point when $\beta_h = 0.45$ (blue line) on the 30th day it will stabilize towards its equilibrium point when $\beta_h = 0.15$ (green line) there is an increase on the graph and The 30th will stabilize towards its equilibrium point and will still be in a state of disease experiencing an outbreak. When β_h is reduced to 0.045 (purple line) on day 100, it will stabilize towards its equilibrium point and approach the disease-free state. When β_h is further reduced to 0.025 (black line), the condition has reached a disease-free state on day 150. Figure $5(d)$, where the recovered individual shows that when β_h is 0.75 (red line) and 0.45 (blue line), the graph increases and then stabilizes to its equilibrium point on day ten but is still experiencing an outbreak or has not yet reached the disease-free, when β_h is reduced to 0.15 (green line) on day 30 it will stabilize towards its equilibrium point. When β_h is reduced again to a value of 0.045 (purple line), on the 100th day, the situation is closer to being disease-free and will stabilize towards equilibrium. When $\beta_h = 0.025$ (black line), the disease state becomes disease-free on the 50th day and stabilizes towards its equilibrium point. Figure 4 shows that the smaller the β_h value given, the graph will start to decline, and dengue fever will reach a disease-free state. In this case, dengue fever will reach a disease-free state with $\mathcal{R}_0$ of 0.967948 when $\beta_h = 0.025$.

4. Conclusion

This study uses the SI_1I_2RS model, which has been modified to show the dynamics of the spread of dengue fever by considering two infected individuals, namely those with mild and severe symptoms.

This model modification produces two equilibrium points: the disease-free equilibrium point, which is locally asymptotically stable when $R_0 < 1$, and the endemic point, which is locally asymptotically stable when $R_0 > 1$. The simulations results can predict the number of cases of dengue fever, and the basic reproduction number is $R_0 = 7.49769 > 1$, where the spread of dengue fever will continue to increase and become endemic.

The numerical simulation results of changes in the parameters of the average individual mosquito bites on humans and the contact rate of disease transmission from infected mosquitoes to susceptible humans significantly influence $\mathcal{R}_0$, where when the average individual mosquito bites on humans (b) is 0.05. The contact rate of disease transmission from infected mosquitoes to susceptible humans (β_h) with a value of 0.025 is enough to suppress the spread of dengue fever outbreaks. Therefore, individuals must maintain cleanliness and take precautions against the spread of dengue fever.

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References

[1] A. Saputra, "Community Behavior Patterns That Influence The Causes of Dengue Hemorrhagic Fever (DHF) in The Pondok Petir Elementary Region," *Muhammadiyah Int. Public Heal. Med. Proceeding*, vol. 1, no. 1, pp. 811–820, 2021. DOI:10.53947/miphmp.v1i1.137

[2] M. A. Kleden, A. Atti, and A. H. Talahatu, "Factors causing Dengue Hemorrhagic Fever (DHF) in Sikka District, East Nusa Tenggara Province," *Jambura Journal of Biomathematics (JJBM)*, vol. 4, no. 1, pp. 80–87, 2023. DOI:10.34312/jjbm.v4i1.19460

[3] Z. E. Fitri *et al.*, "A Combination of Forward Chaining and Certainty Factor Methods for Early Detection of fever: Dengue Hemorrhagic Fever, Malaria and Typhoid," *Sci. J. Informatics*, vol. 9, no. 1, pp. 23–31, 2022. DOI:10.15294/sji.v9i1.33007

[4] M. Nabilah *et al.*, "Forecasting the number of dengue fever based on weather conditions using ensemble forecasting method," *IAES Int. J. Artif. Intell.*, vol. 12, no. 1, pp. 496–504, 2023. DOI:10.11591/ijai.v12.i1.pp496-504

[5] S. Noisakran and C. P. Guey, "Alternate hypothesis on the pathogenesis of dengue hemorrhagic fever (DHF)/dengue shock syndrome (DSS) in dengue virus infection," *Exp. Biol. Med.*, vol. 233, no. 4, pp. 401–408, 2008. DOI:10.3181/0707-MR-198

[6] R. Tumilaar, P. Sianturi, and Jaharuddin, "Mathematical Model of Dengue Disease Transmission Considering the incubation Period Both Intrinsic and Extrinsic," *IOSR J. Math.*, vol. 10, no. 5, pp. 13–18, 2014. DOI:10.9790/5728-10511318

[7] S. B. Halstead, "Dengue," *Lancet*, vol. 370, no. 9599, pp. 1644–1652, 2007. DOI:10.1016/S0140-6736(07)61687-0

[8] D. J. Gubler, "Dengue and dengue hemorrhagic fever." *Clinical microbiology reviews.*, vol. 11, no. 3, pp. 480–96, 1998. DOI:10.1128/CMR.11.3.480

[9] A. Triska, M. H. Dzulfikar, and A. K. Supriatna, "The dynamics of prisoner population model in Indonesia with a rehabilitation regulation for drug users to overcome prison overcapacity issue," *Jambura Journal of Biomathematics (JJBM)*, vol. 4, no. 1, pp. 55–62, 2023. DOI:10.34312/jjbm.v4i1.18898

[10] A. T. R. Sidik *et al.*, "The existence of Neimark-Sacker bifurcation on a discrete-time SIS-Epidemic model incorporating logistic growth and allee effect," *Jambura Journal of Biomathematics (JJBM)*, vol. 3, no. 2, pp. 58–62, 2022. DOI:10.34312/jjbm.v3i2.17515

[11] S. Dundar, B. Gokkurt, and Y. Soylu, "Mathematical Modelling at a Glance: A Theoretical Study," *Procedia - Soc. Behav. Sci.*, vol. 46, pp. 3465–3470, 2012. DOI:10.1016/j.sbspro.2012.06.086

[12] S. Side and S. M. Noorani, "A SIR model for spread of dengue fever disease (simulation for South Sulawesi, Indonesia and Selangor, Malaysia)," *World J. Model. Simul*, vol. 9, no. 2, pp. 96–105, 2013. DOI: 10.13140/RG.2.1.5042.6721

[13] S. Sanusi *et al.*, "Analysis and Simulation of SIRS Model for Dengue Fever Transmission in South Sulawesi, Indonesia," *Journal of Applied Mathematics*, vol. 2021. no. 1, pp. 2918080, 2021. DOI:10.1155/2021/2918080

[14] S. R. NRE and M. U. Riandi, "Infeksi Virus Dengue Tanpa Gejala pada Keluarga Penderita DBD di Provinsi Jawa Barat," *Indonesian Journal of Biotechnology Medicine*, vol. 1, no. 2, pp. 79–84, 2012. DOI: 10.22435/jbmi.v1i2.4182.79-84

[15] P. Pongsumpun, "Transmission Model for Dengue Disease with and Without The Effect of Extrinsic," *KMITL Sci. Tech. J.*, vol. 6, no. 2, pp. 74–82, 2006.

[16] R. Jan, M. A. Khan, and J. F. Gómez-Aguilar, "Asymptomatic carriers in transmission dynamics of dengue with control interventions," Optim. *Control Appl. Methods*, vol. 41, no. 2, pp. 430–447, 2020. DOI:10.1002/oca.2551

[17] A. N. Aini and A. Shodiqin, "Analisis Kestabilan Dan Simulasi Model Penyakit Demam Berdarah Dengue (DBD)," *AKSIOMA: Jurnal Matematika dan Pendidikan Matematika*, vol. 5, no. 2, pp. 1–19, 2014. DOI:10.26877/aks.v5i2/septembe.756