Mathematical Modelling and Control

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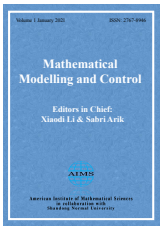

MMC, 5(2): 131–146.
DOI: 10.3934/mmc.2025010
Received: 18 July 2024
Revised: 31 August 2024
Accepted: 18 October 2024
Published: 20 May 2025

Research article

Prevention of dengue virus transmission: insights from host-vector mathematical model

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Abstract: Dengue fever is caused by the dengue virus transmitted by female *Aedes Aegypti*. The spread of dengue fever remains a critical public health issue. Dengue fever is a cycle between humans and mosquitoes. A high number of infected humans causes a high number of infected mosquitoes, and vice versa. Therefore, in this study, we develop a new mathematical model that considers the host for the human population and the vector for the mosquito population. This study focuses on capturing the fundamental dynamics of dengue virus transmission using a widely understandable host-vector model. The analysis is divided into two models: one without control and one with control. In the mathematical model without control, we obtain the basic reproduction number, which is determined using the next-generation matrix. We use the basic reproduction number to analyze the local stability of the disease-free equilibrium and the Routh-Hurwitz criterion for the endemic equilibrium. Additionally, we employ LaSalle's invariance principle and Lyapunov functions to analyze the global stability of both equilibriums. Moreover, we predict the virus spread through a sensitivity analysis of the key parameters that influence the basic reproduction number. We extend the model with control, which promotes for a thorough understanding of the effects of a single-control strategy in depth. Following this analysis, a preventive control strategy using Pontryagin's Maximum Principle is developed to minimize contact between the host and vector, ultimately reducing the spread of the dengue virus. Then, these strategies are numerically solved to investigate the control efforts required to reduce the number of infected classes.

Keywords: dengue; host-vector model; La-Salle Lyapunov; control; Pontryagin

1. Introduction

Dengue fever, which is caused by the dengue virus, is known as one of the most important tropical diseases. There is a circle of dengue virus transmission that concerns humans and mosquitoes; therefore, dengue fever is also called a mosquito-borne disease. The spread of the virus occurs through the bite of an infected female *Aedes Aegypti* mosquito. So far, four serotypes have been detected for the dengue virus: DEN1, DEN2, DEN3, and DEN4 [1]. Humans can be immune to one serotype but can be reinfected by another serotype. Thus, humans living in areas endemic to dengue disease can be reinfected 3 or 4 times by viruses with different serotypes. Since the period of mosquito infection ends through death, the mosquitoes never recover from the infection [1]. The virus has an incubation period of 4 to 10 days for the mosquito, during which, it becomes an infected vector. Suppose the infected mosquito bites a human who is susceptible to the virus. In that case, the virus will enter the human body until it can be transmitted back to another mosquito. Infected humans can transmit the virus through the Aedes Aegypti mosquito for 4 to 5 days after their first symptoms appear [2].

Every year, approximately 100–400 million people are

infected by the dengue virus [3]. The COVID-19 pandemic more or less changed the dynamics of dengue fever cases. Medical reports which focused on this outbreak showed that the dengue disease had lower reporting rates. Dengue fever cases experienced a decline between 2020–2022 (during the COVID-19 pandemic). As a result, in 2023, the dengue fever cases significantly increased globally. In particular, Indonesia has become one of the Southeast Asian countries, ranked among the world's 30 most susceptible to an endemic [4]. This condition becomes a challenge for researchers to reduce the endemicity of dengue fever that occurs year to year.

Understanding the transmission of disease from a mathematical perspective involves the use of mathematical models. Large-scale data-based simulations can be conducted using mathematical models to estimate the spread of epidemics [5, 6]. However, it is also crucial to theoretically study epidemic models to understand their qualitative behavior. This understanding is valuable to inform the control policies. Disease epidemics have another point of view in memory and can have wide-ranging effects in the fractional models using integer order, as studied by [7, 8]. In particular, mathematical modeling concerning the cycle of transmission of the dengue virus has been explored, such as in the host-vector model by the author in [9–12]. The author in [10] modified the model in [9] regarding the incubation period, that is, a percentage of infected humans who are incapable of transmitting the disease. Based on [10], the authors in [11] considered mathematical modeling to predict and control dengue transmission. Furthermore, the optimal control by [12] was performed to take precautions. Utilizing the optimal control theory to model the spread of disease is valuable for researchers and policymakers. This approach helps to explore evidence-based disease prevention and control strategies. The findings from this analysis can contribute to more impactful public health interventions and improved outcomes for populations affected by infectious diseases [13, 14]. Optimal control within the host-vector dengue model is often used to assess the effectiveness of various intervention strategies [15, 16].

Optimal control in dynamical systems is one of the mathematical theories that elaborate on determining the control policy to achieve certain optimality criteria [17]. The optimality procedures involve assigning manipulation system inputs or controls over time to achieve the intended behavior while minimizing or maximizing the objective function. Some notes that must be provided in the theory of optimal control in dynamic systems are control variables, objective functions, constraints, and optimality conditions. For continuous time problems, Pontryagin's Maximum Principle provides the necessary conditions for optimal solutions [18]. It involves Hamilton functions, adjoint variables, and control laws.

Different from the previous studies, in this study, we analyze the mathematical model of dengue virus transmission to control the infected class. Due to the high death rate of dengue fever, we modify the mathematical model in [11] by adding a death rate parameter. There is a possibility that someone will be reinfected with another serotype of the dengue virus; therefore, we enhance a parameter to represent this condition. Additionally, we develop the analysis of the model in [11] by discussing the global stability for both disease-free and endemic equilibrium points. The most commonly used methods for global stability analyses are the Lyapunov stability theorem and LaSalle's invariance principle (see, for example, [19, 20]). However, it is often challenging to construct Lyapunov functions, since there is no general method available. Remarkable mathematical models can contribute to reducing both the infected host and the infected vector population. It is very promising to use host-vector models from a mathematical perspective to answer this.

There are five main findings from this work. First, we develop a new mathematical model that includes deaths due to the dengue virus and considers reinfection of the dengue virus to a person. Second, a global stability analysis is performed using the tricky Lyapunov function. Third, a sensitivity analysis is presented to understand the parameters that influence the spread of the virus. Fourth, the model is developed to provide control over the virus transmission. Fifth, through theoretical and numerical analyses, we can conclude that the presence of control makes the solution behavior move to a disease-free state more quickly.

2. Formulation of the model

In this study, we divide the population into the host and the vector. The total host (human) population is denoted by N_H. The host population itself has three classes: susceptible, infected, and recovered at time t. Meanwhile, the vector population is divided into two classes: susceptible and infected at time t. We denote N_V as the total vector (mosquito) population. The host population on the susceptible class at t is denoted by $S_H(t)$, the host population that is infected at time t is denoted by $I_V(t)$, and the host population that is recovered at time t is denoted by $R_H(t)$. Furthermore, we denote $S_V(t)$ to represent the susceptible vector population at time t, and $I_V(t)$ to denote the infected vector population at time t.

We use the notation μ_H to represent the birth rate of the host population. In this model, we assume a closed host population; thus, the birth rate is equal to the natural death rate. Due to the high death rate due to dengue fever, we modify the mathematical model in [11] by adding the death rate of the host population caused by the virus, which is denoted by μ. If the number of mosquito bites per day is denoted by b, then each day there will be bN_V mosquito bites. Therefore, the host will receive $\frac{bN_V}{N_H}\frac{I_V}{N_V}$ infected mosquito bites per day. If β_H denotes the transmission rate from the mosquito to the host, then $\beta_H\frac{bN_V}{N_H}\frac{I_V}{N_V}$ denotes the infection rate of the susceptible hosts. We use α to represent the susceptible host, who can be susceptible again after recovering from dengue fever. Furthermore, we denote γ_H as the recovery rate.

The population of the vector has a growth rate as much as A, meanwhile μ_V denotes the mortality rate for the vector. Since a mosquito will bite $\frac{b}{N_H}$ per day per person, then one mosquito will get the virus by $\frac{b}{N_H}I_H$ per day. If the transmission rate from the host to the mosquito is denoted by β_V, then the infection rate per susceptible vector is $\beta_V\frac{b}{N_H}I_H$. Figure 1 portrays the mathematical modeling.

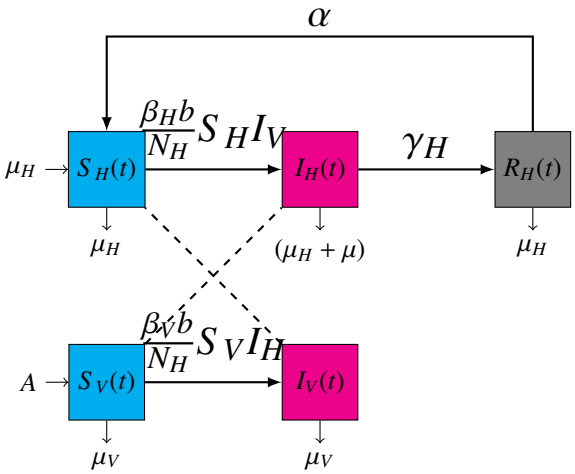


Figure 1. Transfer diagram of the host-vector model.

The mathematical model for dengue virus transmission is a complex system that takes various factors that contribute to the spread of the virus into account, which can be described as follows:

$$\begin{aligned}
\frac{dS_H(t)}{dt} &= \mu_H N_H - \frac{\beta_H b}{N_H} S_H I_V - \mu_H S_H + \alpha R_H, \quad S_H(0) > 0,\\
\frac{dI_H(t)}{dt} &= \frac{\beta_H b}{N_H} S_H I_V - (\mu_H + \mu + \gamma_H) I_H, \quad I_H(0) \geq 0,\\
\frac{dR_H(t)}{dt} &= \gamma_H I_H - \mu_H R_H - \alpha R_H, \quad R_H(0) \geq 0,\\
\frac{dS_V(t)}{dt} &= A - \frac{\beta_V b}{N_H} S_V I_H - \mu_V S_V, \quad S_V(0) > 0,\\
\frac{dI_V(t)}{dt} &= \frac{\beta_V b}{N_H} S_V I_H - \mu_V I_V, \quad I_V(0) \geq 0,
\end{aligned} \tag{2.1}$$

where $S_H + I_H + R_H = N_H$ and $S_V + I_V = N_V$.

To ensure that the model solution reflects the real-world population, it is important to determine the non-negative solutions of System (2.1). Because the negative populations are not feasible, we prove the non-negativity of System (2.1) solutions by Theorem 2.1.

Theorem 2.1. *Solutions of System (2.1) in the set of a vector of a non-negative real number $\mathbb{R}^5_+$ are always non-negative for all time.*

Proof. Let the solutions of System (2.1) rely on $[0, \tau)$, where $0 < \tau < \infty$. Based on the first equation of System (2.1), let $S_H(t) > 0$ for every $t \in [0, \tau)$. If it is not satisfied, then there exist $t_1 \in (0, \tau)$ such that $S_H(t_1) \leq 0$; therefore, $\frac{dS_H(t)}{dt}\Big|_{t=t_1} \leq 0$, and we have $S_H(t) > 0$ for every $t \in [0, t_1)$. We do the

same for the second equation of System (2.1). Let $I_H(t) \geq 0$ for every $t \in [0, t_1)$. If it is not held, then there exist $t_2 \in (0, t_1)$ such that $I_H(t_2) < 0$; therefore, $\frac{dI_H(t)}{dt}\Big|_{t=t_2} < 0$, and we have $I_H(t) \geq 0$ for every $t \in [0, t_1)$.

Furthermore, for the third equation of System (2.1), we claim that $R_H(t) \geq 0$ for every $t \in [0, t_2)$ if $\frac{dR_H(t)}{dt}\Big|_{t=t_3} < 0$. To prove this statement, we use *reductio ad absurdum*. Suppose that there exist $t_3 \in [0, t_2)$ such that $R_H(t_3) < 0$. Then, we obtain the following:

$$\left.\frac{dR_H(t)}{dt}\right|_{t=t_3} = \gamma_H I_H(t_3) - \mu_H R_H(t_3) - \alpha R_H(t_3) \geq 0,$$

since $R_H(t_3) < 0$, $\mu_H > 0$, and $\alpha > 0$. It is a contradiction with the fact that $\frac{dR_H(t)}{dt}\Big|_{t=t_3} < 0$. It implies that if $\frac{dR_H(t)}{dt}\Big|_{t=t_3} < 0$, then $R_H(t) \geq 0$ for every $t \in [0, t_2)$.

In a similar way, we can observe that $S_V > 0$ and $I_V \geq 0$. Now, we come to the first equation of System (2.1); if $\frac{dS_H(t)}{dt}\Big|_{t=t_1} < 0$, then $S_H(t) > 0$ for every $t \in [0, \tau)$. Suppose that there exist $t_1 \in (0, \tau)$ such that $S_H(t_1) \leq 0$,

$$\begin{aligned}\left.\frac{dS_H(t)}{dt}\right|_{t=t_1} =& \mu_H N_H(t_1) - \frac{\beta_H b}{N_H} S_H(t_1) I_V(t_1) \\ & - \mu_H S_H(t_1) + \alpha R_H(t_1) \geq 0.\end{aligned}$$

It is a contradiction to our sufficient condition that $\frac{dS_H(t)}{dt}\Big|_{t=t_1} < 0$; therefore, our supposition should be $S_H(t) > 0$ for every $t \in [0, \tau)$.

Using the same manner, we obtain that $I_H \geq 0$. Hence, all of the solutions of System (2.1) are non-negative for all the time t on $[0, \tau)$, where $0 < \tau < \infty$. □

The intuition of the boundedness of the solution can be explained as follows. We note that N_H remains constant. Furthermore, since μ_V denotes the per capita death rate of the vector population, then $\mu_V N_V$ is the total deaths of the vector. The rate of change of the vector population is represented by the following:

$$\frac{dN_V}{dt} = A - \mu_V N_V.$$

As t approaches infinity, N_V approaches $\frac{A}{\mu_V}$. Thus, $S_V + I_V = \frac{A}{\mu_V}$. Therefore, for N_H and N_V, which are constant, and for convenience, we introduce the following transformation:

$$s_H = \frac{S_H}{N_H}, i_H = \frac{I_H}{N_H}, r_H = \frac{R_H}{N_H}, s_V = \frac{S_V}{A/\mu_V}, i_V = \frac{I_V}{A/\mu_V}. \tag{2.2}$$

Additionally, we use the relation $r_H = 1 - (s_H + i_H)$ and $s_V = 1 - i_V$. Using transformation (2.2), System (2.1) can be written as follows:

$$\frac{ds_H(t)}{dt} = \mu_H - \frac{\beta_H b (A/\mu_V)}{N_H} s_H i_V - \mu_H s_H + \alpha(1 - (s_H + i_H)), \tag{2.3a}$$

$$\frac{di_H(t)}{dt} = \frac{\beta_H b (A/\mu_V)}{N_H} s_H i_V - (\mu_H + \mu + \gamma_H) i_H, \tag{2.3b}$$

$$\frac{di_V}{dt} = \beta_V b s_V i_H - \mu_V i_V = \beta_V b (1 - i_V) i_H - \mu_V i_V. \tag{2.3c}$$

In the next section, we examine the equilibrium points of System (2.3) and their stability. Generally, we obtain two types of equilibrium: disease-free and endemic. The local stability of the equilibrium can be studied through the value of the basic reproduction number. Meanwhile, La-Salle Lyapunov can be applied to analyze the global stability of the equilibrium point.

3. Equilibrium points and their stability

The equilibrium points of System (2.3) will be explained in the following discussion. For convenience, we use the relation (3.1) for our analysis. Let

$$\begin{aligned} M_1 &= \frac{\beta_V b}{\mu_V}(\mu_H + \alpha), \\ M_2 &= \frac{\beta_H b (A/\mu_V)}{N_H} \frac{\beta_V b}{\mu_V}, \\ \hat{R}_0 &= \frac{\beta_H b (A/\mu_V)}{N_H (\mu_H + \mu + \gamma_H)} \frac{\beta_V b}{\mu_V}. \end{aligned} \tag{3.1}$$

3.1. Disease-free equilibrium point

The equilibrium solutions are determined by setting each equation in System (2.3) equal to zero. Based on (2.3a), (2.3c), and (3.1), we obtain the following:

$$s_H = \frac{(\mu_H + \alpha - \alpha i_H)((\beta_V b/\mu_V) i_H + 1)}{M_2 i_H + (\alpha + \mu_H)((\beta_V b/\mu_V) i_H + 1)}, \tag{3.2a}$$

$$i_V = \frac{\beta_V b i_H}{\beta_V b i_H + \mu_V}. \tag{3.2b}$$

By substituting Eq (3.2) into (2.3b), then we have that i_H must satisfy the following quadratic equation:

$$-\left(\alpha\hat{R}_0 + M_1 + M_2\right) i_H^2 + (\mu_H + \alpha)\left(\hat{R}_0 - 1\right) i_H = 0. \tag{3.3}$$

Therefore, the disease-free equilibrium point can be summarized by the following lemma.

Lemma 3.1. *If $i_H = 0$, then System (2.3) has a disease free equilibrium point, $E_0 = (1, 0, 0)$.*

Proof. One of the solutions of (3.3) is $i_H = 0$. By taking the right-hand side of (2.3c) to zero and substituting $i_H = 0$, we come to have $i_V = 0$. Moreover, since $i_H = 0$ and $i_V = 0$, then we obtain $s_H = 1$ by taking zero to the right-hand side of (2.3a). □

Now, we will discuss the basic reproduction number that indicates the value of secondary infections resulting from the first infection. This value is determined using the next-generation matrix method [21]. In this study, matrix F_1 is defined as the matrix whose entries consist of the first derivative of all terms that represent the rate of change of all factors contributing to the infectious class at the disease-free equilibrium E_0. Therefore,

$$F_1 = \begin{bmatrix} \frac{\partial}{\partial i_H}\left(\frac{\beta_H b(A/\mu_V)}{N_H} s_H i_V\right) & \frac{\partial}{\partial i_H}\left(\beta_V b i_H\right) \\ \frac{\partial}{\partial i_V}\left(\frac{\beta_H b(A/\mu_V)}{N_H} s_H i_V\right) & \frac{\partial}{\partial i_V}\left(\beta_V b i_H\right) \end{bmatrix}_{\text{at }(1,0,0)} = \begin{bmatrix} 0 & \beta_V b \\ \frac{\beta_H b(A/\mu_H)}{N_H} & 0 \end{bmatrix}. \tag{3.4}$$

We create the matrix F_2 as a matrix of loss terms for each class, which is evaluated at the disease-free equilibrium as follows:

$$F_2 = \begin{bmatrix} \frac{\partial}{\partial i_H}\left(\mu_H + \mu + \gamma_H\right) i_H & \frac{\partial}{\partial i_H}\left(\mu_V + \beta_V b i_H\right) i_V \\ \frac{\partial}{\partial i_V}\left(\mu_H + \mu + \gamma_H\right) i_H & \frac{\partial}{\partial i_V}\left(\mu_V + \beta_V b i_H\right) i_V \end{bmatrix}_{\text{at }(1,0,0)} = \begin{bmatrix} (\mu_H + \mu + \gamma_H) & 0 \\ 0 & (\mu_V) \end{bmatrix}. \tag{3.5}$$

The inverse form of (3.5) is as follows:

$${F_2}^{-1} = \frac{1}{(\mu_H + \mu + \gamma_H)\mu_V} \begin{bmatrix} \mu_V & 0 \\ 0 & \mu_H + \mu + \gamma_H \end{bmatrix} = \begin{bmatrix} \frac{1}{\mu_H+\mu+\gamma_H} & 0 \\ 0 & \frac{1}{\mu_V} \end{bmatrix}. \tag{3.6}$$

Based on Matrices (3.4) and (3.6), we obtain Matrix P, which is a product of F_1 and F_2^{-1}; then,

$$P = F_1 {F_2}^{-1} = \begin{bmatrix} 0 & \frac{\beta_V b}{\mu_V} \\ \frac{\beta_H b(A/\mu_V)}{N_H(\mu_H+\mu+\gamma_H)} & 0 \end{bmatrix}. \tag{3.7}$$

The largest eigenvalue of the matrix P is as follows:

$$\sqrt{\frac{\beta_V b\,(A/\mu_V)}{N_H\,(\mu_H + \mu + \gamma_H)} \frac{\beta_H b}{\mu_V}}.$$

If R_0 is a notation for this value, then, in this study, R_0 is referred to as the basic reproduction number of System (2.3). Furthermore, if

$$\hat{R}_0 = \frac{\beta_V b\,(A/\mu_V)}{N_H\,(\mu_H + \mu + \gamma_H)} \frac{\beta_H b}{\mu_V}, \tag{3.8}$$

then

$$R_0 = \sqrt{\hat{R}_0}\,. \tag{3.9}$$

This can be interpreted as the average number of secondary cases in the susceptible population caused by a single case. In a detailed biological interpretation, this can be seen as follows: an infected host that exists in the susceptible population is bitten during his/her infective period by $\frac{b(A/\mu_V)}{N_H(\mu_H+\mu+\gamma_H)}$ susceptible vectors. This results in a portion $\frac{\beta_V b(A/\mu_V)}{N_H(\mu_H+\mu+\gamma_H)}$ of the vectors becoming infected. On the other hand, an infected vector administers $\frac{b}{\mu_V}$ of bites to the susceptible hosts. These bites lead to a portion $\frac{\beta_H b}{\mu_V}$ of new infections to the host population.

The next section will describe the stability near the disease-free equilibrium point.

3.2. Stability analysis of disease-free equilibrium point

We describe the stability analysis of the disease-free equilibrium in Theorem 3.1.

Theorem 3.1. *Given R_0 in (3.9):*

i. If $\hat{R}_0 > 1$, then E_0 is unstable.

ii. If $\hat{R}_0 < 1$, then E_0 is locally asymptotically stable.

Proof. The Jacobian matrix at E_0 is as follows:

$$J|_{(1,0,0)} = \begin{bmatrix} -\mu_H - \alpha & -\alpha & -\frac{\beta_H b A/\mu_V}{N_H} \\ 0 & -\mu_H - \mu - \gamma_H & \frac{\beta_H A/\mu_V}{N_H} \\ 0 & \beta_V b & -\mu_V \end{bmatrix}. \tag{3.10}$$

If λ denotes the eigenvalue and I denotes the identity matrix, then the eigenvalues of the matrix (3.10) are determined by solving the characteristic equation $|J(E_0) - \lambda I| = 0$. It implies that

$$(-\mu_H - \alpha - \lambda)\left[\lambda^2 + (\mu_H + \mu + \gamma_H + \mu_V)\lambda + (\mu_H + \mu + \gamma_H)\mu_V - \left(\frac{\beta_H b A/\mu_V}{N_H}\right)(\beta_V b)\right] = 0.$$

Therefore, $\lambda_1 = -(\mu_H + \alpha)$ and

$$\lambda_{2,3} = \frac{-(\mu_H + \mu + \gamma_H + \mu_V)}{2} \pm \frac{\sqrt{(\mu_H + \mu + \gamma_H + \mu_V)^2 - 4(\mu_H + \mu + \gamma_H)\mu_V\left(1 - \hat{R}_0\right)}}{2}, \tag{3.11}$$

where $\hat{R}_0$ is written as in (3.8). Thus,

i. If $\hat{R}_0 > 1$, then one of the eigenvalue in (3.11) is positive. Since we have a positive eigenvalue, then E_0 is unstable.

ii. If $\hat{R}_0 < 1$, then the eigenvalues in (3.11) are negative. Since all the eigenvalues are negative, then E_0 is locally asymptotically stable.

□

The stability of the disease-free equilibrium point can be interpreted whenever $\hat{R}_0 < 1$; then, there is no virus transmission in the population, and all humans are free of a dengue virus infection. Next, we analyze the global stability of the disease-free equilibrium points using La Salle Lyapunov [22]. In this study, we consider the Lyapunov function $V : \mathbb{R}^3_{+,0} \to \mathbb{R}^3$, where $\mathbb{R}^3_{+,0} = \left\{(s_H, i_H, i_V) \in \mathbb{R}^3 : s_H > 0, i_H, i_V \geq 0\right\}$, and

$$V = i_H + \frac{\beta_H b\,(A/\mu_V)}{\mu_V N_H} i_V. \tag{3.12}$$

It is easy to show that V and its partial derivatives are continuous in $\mathbb{R}^3$. Additionally, V is positive definite. The orbital derivative of function (3.12) is determined by the following:

$$\begin{aligned}\dot{V} =& \frac{\partial V}{\partial s_H}\frac{ds_H}{dt} + \frac{\partial V}{\partial i_H}\frac{di_H}{dt} + \frac{\partial V}{\partial i_V}\frac{di_V}{dt}\\ =& \frac{\beta_H b\,(A/\mu_V)}{N_H} s_H i_V - (\mu_H + \mu + \gamma_H) i_H\\ &+ \frac{\beta_H b\,(A/\mu_V)}{\mu_V N_H}\left[\beta_V b\,(1 - i_V) i_H - \mu_V i_V\right]\\ =& -\frac{\beta_H b\,(A/\mu_V)}{N_H}\left[1 - s_H\right] i_V\\ &- (\mu_H + \mu + \gamma_H)\left[1 - \hat{R}_0\,(1 - i_V)\right] i_H.\end{aligned}$$

Therefore, we achieve the global stability of the disease-free equilibrium point in Theorem 3.2.

Theorem 3.2. *If $\hat{R}_0 < 1$, then the disease-free equilibrium point is globally asymptotically stable.*

Proof. If $\hat{R}_0 < 1$, then we have $\dot{V} < 0$. We also note that $\dot{V}(E_0) = 0$. Therefore, based on La Salle Lyapunov, the disease-free equilibrium E_0 is globally asymptotically stable. □

3.3. Existence and local stability analysis of endemic equilibrium point

Recall the solution of Eq (3.3). We obtained $i_H = 0$, as the solution coincides with the disease-free equilibrium point; therefore, the other solution, $i_H \neq 0$, corresponds to the endemic equilibrium point. Based on (3.3), we obtain the following:

$$i_H = \frac{(\mu_H + \alpha)\left(\hat{R}_0 - 1\right)}{M_1 + M_2 + \alpha\hat{R}_0}. \tag{3.13}$$

The solution $i_H > 0$ in (3.13) exists if $\hat{R}_0 > 1$. Consequently, the existence of the endemic equilibrium point can be concluded through the following Lemma 3.2.

Lemma 3.2. *If $\hat{R}_0 > 1$, then the endemic equilibrium point exists.*

Proof. Note that $\hat{R}_0 > 1$. The solution in (3.13) can be substituted into (3.2); then, we acquire the endemic equilibrium point, $E_1 = \left(s^*_H, i^*_H, i^*_V\right)$, where

$$\begin{aligned}s^*_H &= \frac{M_1 + \alpha + \mu_H + \mu + \gamma_H}{M_1 + M_2 + \alpha\hat{R}_0},\\ i^*_H &= \frac{(\mu_H + \alpha)\left(\hat{R}_0 - 1\right)}{M_1 + M_2 + \alpha\hat{R}_0},\\ i^*_V &= \frac{M_1\left(\hat{R}_0 - 1\right)}{(M_1 + \alpha)\hat{R}_0 + M_2}.\end{aligned}$$

□

Furthermore, a stability analysis of the endemic equilibrium point can be explained by Theorem 3.3.

Theorem 3.3. *If $\hat{R}_0 > 1$, then the endemic equilibrium point E_1 is locally asymptotically stable.*

Proof. We consider that $\hat{R}_0 > 1$. Based on this and (3.1), we derive the Jacobian matrix at E_1 as follows:

$$J|_{(s^*_H, i^*_H, i^*_V)} = \begin{bmatrix} a_{11} & a_{12} & a_{13}\\ a_{21} & a_{22} & a_{23}\\ a_{31} & a_{32} & a_{33}\end{bmatrix}, \tag{3.14}$$

where

$$
\begin{aligned}
a_{11} &= -\frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}-(\mu_H+\alpha),\\
a_{12} &= -\alpha,\\
a_{13} &= -\frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1+\alpha+\mu_H+\mu+\gamma_H}{M_1+M_2+\alpha\hat{R}_0},\\
a_{21} &= \frac{\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2},\\
a_{22} &= -(\mu_H+\mu+\gamma_H),\\
a_{23} &= \frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1+\alpha+\mu_H+\mu+\gamma_H}{M_1+M_2+\alpha\hat{R}_0},\\
a_{31} &= 0,\\
a_{32} &= \beta_V b\left(1-\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}\right),\\
a_{33} &= -\beta_V b\frac{(\mu_H+\alpha)\left(\hat{R}_0-1\right)}{M_1+M_2+\alpha\hat{R}_0}-\mu_V.
\end{aligned}
$$

The eigenvalues of matrix (3.14) are determined by solving the characteristic equation $|J(E_1)-\lambda I| = 0$, where λ denotes the eigenvalues; we solve λ in (3.15) as follows:

$$
\begin{aligned}
&\left[-\frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}-(\mu_H+\alpha)-\lambda\right]\\
&\times[-(\mu_H+\mu+\gamma_H)-\lambda]\left[-\beta_V b\frac{(\mu_H+\alpha)\left(\hat{R}_0-1\right)}{M_1+M_2+\alpha\hat{R}_0}-\mu_V-\lambda\right]\\
&+\left[-\frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1+\alpha+\mu_H+\mu+\gamma_H}{M_1+M_2+\alpha\hat{R}_0}\right]\\
&\times\frac{\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}\\
&\times\beta_V b\left(1-\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}\right)\\
&-\left[-\frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}-(\mu_H+\alpha)-\lambda\right]\\
&\times\frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1+\alpha+\mu_H+\mu+\gamma_H}{M_1+M_2+\alpha\hat{R}_0}\\
&\times\beta_V b\left(1-\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}\right)\\
&-\frac{\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}\\
&\times\left(\beta_V b\frac{(\mu_H+\alpha)\left(\hat{R}_0-1\right)}{M_1+M_2+\alpha\hat{R}_0}+\mu_V+\lambda\right)\alpha=0. \qquad (3.15)
\end{aligned}
$$

Solving (3.15) means that we solve

$$\lambda^3+Q_1\lambda^2+Q_2\lambda+Q_3=0,$$

where

$$
\begin{aligned}
Q_1 =&\frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}+\beta_V b\frac{(\mu_H+\alpha)\left(\hat{R}_0-1\right)}{M_1+M_2+\alpha\hat{R}_0}\\
&+\mu_V+(\mu_H+\alpha)+(\mu_H+\mu+\gamma_H),\\
Q_2 =&\left(\frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}+(\mu_H+\alpha)\right)\\
&\times(\mu_H+\mu+\gamma_H)+\left(\frac{\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}\right.\\
&\left.+(\mu_H+\alpha)(\mu_H+\mu+\gamma_H)\right)\\
&\times\left(\beta_V b\frac{(\mu_H+\alpha)\left(\hat{R}_0-1\right)}{M_1+M_2+\alpha\hat{R}_0}+\mu_V\right)\\
&+\frac{\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1+\alpha+\mu_H+\mu+\gamma_H}{M_1+M_2+\alpha\hat{R}_0}\\
&\times\beta_V b\left(\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}-1\right)\\
&+\alpha\frac{\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2},\\
Q_3 =&\left(\frac{\beta_H b\left({}^{A}/_{\mu_V}\right)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}+(\mu_H+\alpha)\right)(\mu_H+\mu+\gamma_H)\\
&\times\left(\beta_V b\frac{(\mu_H+\alpha)\left(\hat{R}_0-1\right)}{M_1+M_2+\alpha\hat{R}_0}+\mu_V\right)\\
&+\left[\frac{\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1+\alpha+\mu_H+\mu+\gamma_H}{M_1+M_2+\alpha\hat{R}_0}\right]\frac{\beta_H b\,(A/\mu_V)}{N_H}\\
&\times\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}\beta_V b\left(\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}-1\right)\\
&+\left(\frac{\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}+(\mu_H+\alpha)\right)\\
&\times\frac{\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1+\alpha+\mu_H+\mu+\gamma_H}{M_1+M_2+\alpha\hat{R}_0}\\
&\times\beta_V b\left(\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}-1\right)\\
&+\frac{\alpha\beta_H b\,(A/\mu_V)}{N_H}\frac{M_1\left(\hat{R}_0-1\right)}{(M_1+\alpha)\hat{R}_0+M_2}
\end{aligned}
$$

$$\times\left(\beta_V b\frac{(\mu_H+\alpha)\left(\hat{R}_0-1\right)}{M_1+M_2+\alpha\hat{R}_0}+\mu_V\right).$$

In this case, if $\hat{R}_0 > 1$, then it implies $Q_1, Q_2, Q_3 > 0$. By manipulating the algebra, we also obtain that $Q_1, Q_2 > Q_3$. Based on the Routh Hurwitz criterion, the endemic equilibrium point E_1 is locally asymptotically stable. □

3.4. Global stability of the endemic equilibrium point

The discussion in this article continues regarding the global stability around the endemic equilibrium point, particularly when $\alpha = 0$ represents a population with permanent immunity. The dynamic model for the spread of dengue fever is as follows:

$$\begin{aligned}\frac{ds_H(t)}{dt}&=\mu_H-\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H i_V-\mu_H s_H,\\ \frac{di_H(t)}{dt}&=\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H i_V-(\mu_H+\mu+\gamma_H)i_H,\\ \frac{di_V}{dt}&=\beta_V b s_V i_H-\mu_V i_V=\beta_V b(1-i_V)i_H-\mu_V i_V.\end{aligned}\tag{3.16}$$

The endemic equilibrium $\left(s_H^{**}, i_H^{**}, i_V^{**}\right)$ satisfies the following:

$$\begin{aligned}\mu_H&=\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H^{**}i_V^{**}+\mu_H s_H^{**},\\ (\mu_H+\mu+\gamma_H)i_H^{**}&=\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H^{**}i_V^{**},\\ \mu_V i_V^{**}&=\beta_V b\left(1-i_V^{**}\right)i_H^{**}.\end{aligned}\tag{3.17}$$

Let the function

$$\begin{aligned}W=&s_H-s_H^{**}-s_H^{**}\ln\left(\frac{s_H}{s_H^{**}}\right)+i_H-i_H^{**}-i_H^{**}\ln\left(\frac{i_H}{i_H^{**}}\right)\\&+i_V-i_V^{**}-i_V^{**}\ln\left(\frac{i_V}{i_V^{**}}\right),\end{aligned}\tag{3.18}$$

and note that $g(x)=x-1-\ln x\geq g(1)=0$ for $x>0$. We introduce $y_1=\frac{s_H}{s_H^{**}}$, $y_2=\frac{i_H}{i_H^{**}}$, and $y_3=\frac{i_V}{i_V^{**}}$. The derivative of W along the solution (3.16), and using (3.17), we obtain the following:

$$\begin{aligned}\frac{\partial W}{\partial s_H}\frac{ds_H}{dt}=&\left(1-\frac{s_H^{**}}{s_H}\right)\left(\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H^{**}i_V^{**}+\mu_H s_H^{**}\right.\\&\left.-\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H i_V-\mu_H s_H\right)\\=&-\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H^{**}i_V^{**}(y_1y_3-1)\\&+\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H^{**}i_V^{**}\left(y_3-\frac{1}{y_1}\right)-\mu_H s_H^{**}(y_1-1),\end{aligned}\tag{3.19}$$

$$\begin{aligned}\frac{\partial W}{\partial i_H}\frac{di_H}{dt}=&\left(1-\frac{i_H^{**}}{i_H}\right)\left(\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H i_V\right.\\&\left.-\frac{\beta_H b\left(A/\mu_V\right)}{N_H}\frac{s_H^{**}i_V^{**}}{i_H^{**}}i_H\right)\\=&\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H^{**}i_V^{**}(y_1y_3-\frac{y_1y_3}{y_2}),\end{aligned}\tag{3.20}$$

$$\begin{aligned}\frac{\partial W}{\partial i_V}\frac{di_V}{dt}=&\left(1-\frac{i_V^{**}}{i_V}\right)\left(\beta_V b(1-i_V)i_H-\beta_V b\left(1-i_V^{**}\right)\frac{i_H^{**}}{i_V^{**}}i_V\right)\\=&-\beta_V b i_V^{**}i_H^{**}(y_2y_3+1-y_2-y_3)-\beta_V b y_3\left(1+\frac{1}{y_3^2}\right).\end{aligned}\tag{3.21}$$

If (3.19)–(3.21) are substituted into (3.18), then we have the following:

$$\begin{aligned}\dot{W}=&\frac{\partial W}{\partial s_H}\frac{ds_H}{dt}+\frac{\partial W}{\partial i_H}\frac{di_H}{dt}+\frac{\partial W}{\partial i_V}\frac{di_V}{dt}\\=&-\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H^{**}i_V^{**}\left(\frac{y_1y_3}{y_2}-y_3+\frac{1}{y_1}-1\right)-\mu_H s_H^{**}(y_1-1)\\&-\beta_V b i_V^{**}i_H^{**}(y_2y_3+1-y_2-y_3)-\beta_V b y_3\left(1+\frac{1}{y_3^2}\right)\\\leq&-\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H^{**}i_V^{**}\left(\frac{y_1y_3}{y_2}-y_3+\frac{1}{y_1}-1\right)\\&-\beta_V b i_V^{**}i_H^{**}(y_2y_3+1-y_2-y_3)-\beta_V b y_3\left(1+\frac{1}{y_3^2}\right)\\\leq&-\frac{\beta_H b\left(A/\mu_V\right)}{N_H}s_H^{**}i_V^{**}\left(g\left(\frac{y_1y_3}{y_2}\right)+g\left(\frac{1}{y_1}\right)\right)\\&-\beta_V b i_V^{**}i_H^{**}g(y_2y_3)-\beta_V b y_3\left(1+\frac{1}{y_3^2}\right),\end{aligned}\tag{3.22}$$

where we use the inequality $1-x\leq-\ln x$ and $g(x)\geq 0$. Therefore, we come to the global stability of the endemic equilibrium point in Theorem 3.4.

Theorem 3.4. *If $\hat{R}_0 > 1$, then the endemic equilibrium point is globally asymptotically stable.*

Proof. Based on Lemma 3.2, the endemic equilibrium exists if $\hat{R}_0 > 1$. Moreover, based on (3.22), we have $\dot{W} \leq 0$. Furthermore, $\dot{W} = 0$ is satisfied at $\left(s_H^{**}, i_H^{**}, i_V^{**}\right)$. The claim follows by LaSalle's Invariance Principle [22]. □

4. Sensitivity analysis

A sensitivity analysis explains the impact of changes in the parameter values on the system behavior. We study the sensitivity of the basic reproduction number to determine the robustness of the model to the parameter values measured by the *sensitivity index*. Next, the sensitivity index is defined as follows.

Definition 4.1. *[23, 24] The sensitivity index of variable y is the relative change ratio of y to the relative change of parameter s, and is defined as follows:*

$$\Upsilon_s^y = \frac{\partial y}{\partial s} \times \frac{s}{y}. \tag{4.1}$$

We analyze the sensitivity index of $\hat{R}_0$ using the formula (4.1), especially for the parameters β_V, β_H, b, μ, and α that influence R_0. The sensitivity index of $\hat{R}_0$ can either depend on the values of other parameters or be a constant that does not depend on the parameter values. Based on Definition 4.1 and Eq (3.9), we obtain the following:

$$\begin{aligned}
\Upsilon_{\beta_V}^{\hat{R}_0} &= \frac{\partial \hat{R}_0}{\partial \beta_V} \times \frac{\beta_V}{\hat{R}_0} = 1,\\
\Upsilon_{\beta_H}^{\hat{R}_0} &= \frac{\partial \hat{R}_0}{\partial \beta_H} \times \frac{\beta_H}{\hat{R}_0} = 1,\\
\Upsilon_{b}^{\hat{R}_0} &= \frac{\partial \hat{R}_0}{\partial b} \times \frac{b}{\hat{R}_0} = 2,\\
\Upsilon_{\mu}^{\hat{R}_0} &= \frac{\partial \hat{R}_0}{\partial \mu} \times \frac{\mu}{\hat{R}_0} = -\frac{\mu}{(\mu_H + \mu + \gamma_H)},\\
\Upsilon_{\alpha}^{\hat{R}_0} &= \frac{\partial \hat{R}_0}{\partial \alpha} \times \frac{\alpha}{\hat{R}_0} = 0.
\end{aligned}$$

Based on the signs of the sensitivity index, the value of $\hat{R}_0$ increases by β_V, β_H, and b. On the contrary, it decreases by μ. Moreover, α does not impact the value of $\hat{R}_0$. We note that the most sensitive parameter corresponds to the largest sensitivity index. In this case, b is the most sensitive parameter; an increase of b by 10% increases $\hat{R}_0$ by 10%. Recall that in this case, b represents the number of mosquito bites per day. Based on the sensitivity index, the value of $\hat{R}_0$ decreases if the value of b decreases. Biologically, the value of b can be a target to reduce as the intervention of dengue transmission. We can reduce the value of b by reducing the presence of mosquitoes. In addition, increasing the value of μ decreases $\hat{R}_0$. Changing the value of an individual who can be re-infected cannot alter the value of $\hat{R}_0$.

5. Optimal control

This section will delve deeper into the dengue virus transmission and discuss how it can be controlled using the prevention control variable. We denote u_1, where $0 \leq u_1 \leq 1$, as a control variable to reduce the contact between the host and the vector. We have derived the following model:

$$\begin{aligned}
\frac{dS_H(t)}{dt} &= \mu_H N_H - (1-u_1)\frac{\beta_H b S_H I_V}{N_H} - \mu_H S_H + \alpha R_H,\\
\frac{dI_H(t)}{dt} &= (1-u_1)\frac{\beta_H b S_H I_V}{N_H} - \mu_H I_H - \mu I_H - \gamma_H I_H,\\
\frac{dR_H(t)}{dt} &= \gamma_H I_H - \mu_H R_H - \alpha R_H,\\
\frac{dS_V(t)}{dt} &= A - (1-u_1)\frac{\beta_V b S_V I_H}{N_H} - \mu_V S_V,\\
\frac{dI_V(t)}{dt} &= (1-u_1)\frac{\beta_V b S_V I_H}{N_H} - \mu_V I_V.
\end{aligned} \tag{5.1}$$

To investigate the optimality of these efforts, we utilize the objective function G, which minimizes the infected host class and the cost of implementing the u_1 control. In this study, if B is the weight for the infected hosts class, and C is the weight for prevention efforts, then the solution of (5.1) minimizes the number of infected hosts with a minimal control cost $u_1(t)$ such that

$$G(u_1) = \int_0^T \left(BI_H(t) + Cu_1^2(t)\right)dt. \tag{5.2}$$

To further elaborate, the Hamiltonian of this study is as follows:

$$\begin{aligned}
H =& BI_H(t) + Cu_1^2(t)\\
&+ \delta_1\left(\mu_H N_H - (1-u_1)\frac{\beta_H b S_H I_V}{N_H} - \mu_H S_H + \alpha R_H\right)\\
&+ \delta_2\left((1-u_1)\frac{\beta_H b S_H I_V}{N_H} - \mu_H I_H - \mu I_H - \gamma_H I_H\right)\\
&+ \delta_3(\gamma_H I_H - \mu_H R_H - \alpha R_H)
\end{aligned}$$

$$+\delta_4\left(A-(1-u_1)\frac{\beta_V bS_V I_H}{N_H}-\mu_V S_V\right)$$
$$+\delta_5\left((1-u_1)\frac{\beta_V bS_V I_H}{N_H}-\mu_V I_V\right),$$

where $\delta_1, \delta_2, ..., \delta_5$ are costate variables. We use the Maximum Pontryagin principle to determine the existence of an optimal control [17, 18, 24, 25]. The following theorem provides an idea.

Theorem 5.1. *If u_1^* is the optimal control that minimizes $G(u_1)$ with System (5.1) as the constraint, then there exist costate variables $\delta_1, \delta_2, ..., \delta_5$ such that*

$$\begin{aligned}
\frac{d\delta_1}{dt} =&\delta_1\left((1-u_1)\frac{\beta_H bI_V}{N_H}+\mu_H\right)-\delta_2(1-u_1)\frac{\beta_H bI_V}{N_H},\\
\frac{d\delta_2}{dt} =&-B+\delta_2(\mu_H+\mu+\gamma_H)-\delta_3\gamma_H+\delta_4(1-u_1)\frac{\beta_V bS_V}{N_H}\\
&-\delta_5(1-u_1)\frac{\beta_V bS_V}{N_H},\\
\frac{d\delta_3}{dt} =&-\alpha\delta_1+\delta_3(\mu_H+\alpha),\\
\frac{d\delta_4}{dt} =&\delta_4(1-u_1)\frac{\beta_V bI_H}{N_H}-\delta_5(1-u_1)\frac{\beta_V bI_H}{N_H},\\
\frac{d\delta_5}{dt} =&\delta_1(1-u_1)\frac{\beta_H bS_H}{N_H}-\delta_2(1-u_1)\frac{\beta_H bS_H}{N_H}+\delta_5\mu_V,
\end{aligned}$$

with transversality conditions $\delta_i(T)=0, i=1,...,5$, and the u_1^* satisfies the optimality condition

$$u_1^*=\min\left\{\max\left(0,\frac{(\delta_2-\delta_1)\frac{\beta_H bS_H^* I_V^*}{N_H}+(\delta_5-\delta_4)\frac{\beta_V bS_V^* I_H^*}{N_H}}{2C}\right),1\right\}.$$

Proof. The costate equations are obtained by differentiating the Hamiltonian function, evaluated at the optimal control, as follows:

$$\begin{aligned}
\frac{d\delta_1}{dt} =&-\frac{\partial H}{\partial S_H}=\delta_1\left((1-u_1)\frac{\beta_H bI_V}{N_H}+\mu_H\right)-\delta_2(1-u_1)\frac{\beta_H bI_V}{N_H},\\
\frac{d\delta_2}{dt} =&-\frac{\partial H}{\partial I_H}=-B+\delta_2(\mu_H+\mu+\gamma_H)-\delta_3\gamma_H\\
&+\delta_4(1-u_1)\frac{\beta_V bS_V}{N_H}-\delta_5(1-u_1)\frac{\beta_V bS_V}{N_H},\\
\frac{d\delta_3}{dt} =&-\frac{\partial H}{\partial R_H}=-\alpha\delta_1+\delta_3(\mu_H+\alpha),\\
\frac{d\delta_4}{dt} =&-\frac{\partial H}{\partial S_V}=\delta_4(1-u_1)\frac{\beta_V bI_H}{N_H}-\delta_5(1-u_1)\frac{\beta_V bI_H}{N_H},\\
\frac{d\delta_5}{dt} =&-\frac{\partial H}{\partial I_V}=\delta_1(1-u_1)\frac{\beta_H bS_H}{N_H}-\delta_2(1-u_1)\frac{\beta_H bS_H}{N_H}+\delta_5\mu_V.
\end{aligned}$$

The optimal control at the solutions $S_H^*, I_H^*, R_H^*, S_V^*, I_V^*$ at System (5.1) is obtained by the following:

$$\frac{\partial H}{\partial u_1}=0\Leftrightarrow 2Cu_1^*+\delta_1\frac{\beta_H bS_H^* I_V^*}{N_H}-\delta_2\frac{\beta_H bS_H^* I_V^*}{N_H}+\delta_4\frac{\beta_V bS_V^* I_H^*}{N_H}-\delta_5\frac{\beta_V bS_V^* I_H^*}{N_H}=0$$
$$\Leftrightarrow u_1^*=\frac{(\delta_2-\delta_1)\frac{\beta_H bS_H^* I_V^*}{N_H}+(\delta_5-\delta_4)\frac{\beta_V bS_V^* I_H^*}{N_H}}{2C},$$

and

$$u_1^*=\min\left\{\max\left(0,\frac{(\delta_2-\delta_1)\frac{\beta_H bS_H^* I_V^*}{N_H}+(\delta_5-\delta_4)\frac{\beta_V bS_V^* I_H^*}{N_H}}{2C}\right),1\right\}.$$

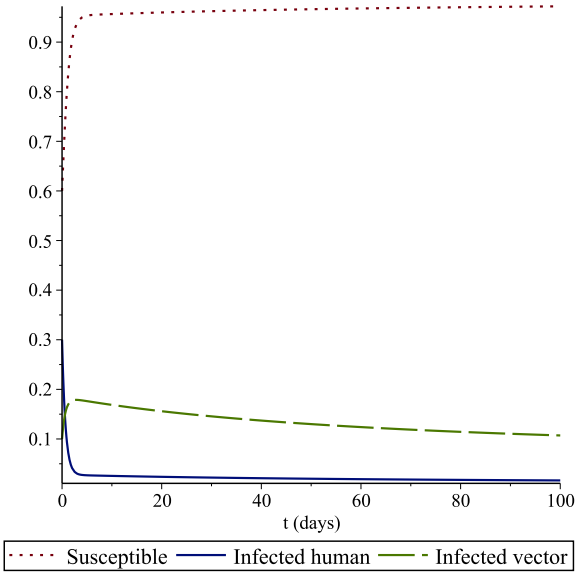
□

6. Numerical simulation

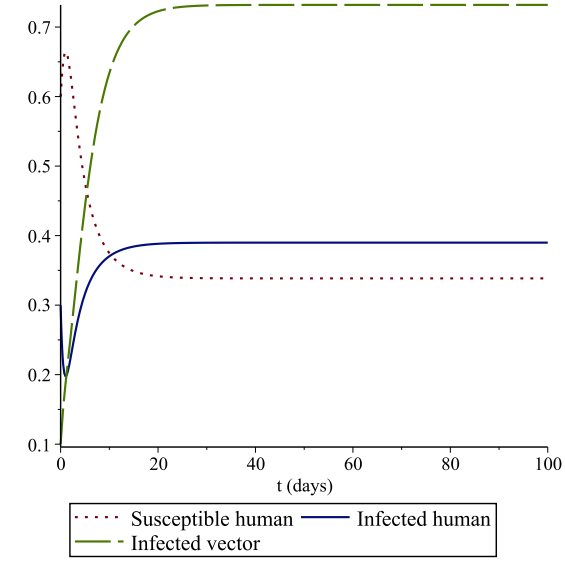
To illustrate the analytical results, in this simulation, we select parameter values based on prior research, see [9, 11] and the references cited therein. We use the following parameters: $\alpha = 0,9619$; $\mu_V = 0,071428$; $b = 0,5$; $\beta_V = 1$; $\beta_H = 0,75$; $\mu_H = 0,391389$; $\mu = 0,8$; and $\gamma_H = 0,1428$. Using these parameter values, using $A = 400$, and $N_H = 1000$, we obtain $\hat{R}_0 \approx 22,036 > 1$. On the other hand, if we use $A = 40$; $N_H = 10000$, then we have $\hat{R}_0 \approx 0,22 < 1$.

Figure 2 shows the illustration of Theorems 3.1 and 3.3. Using different initial values in Figure 3 (i.e., (0,45;0,22;0,3), (0,5;0,25;0,165), and (0,4;0,24;0,2) shows us that the solutions tend to the disease-free equilibrium point. On the other hand, we aim to reduce the number of infected humans and infected vectors in the population. Therefore, the endemicity should be controllable. Figure 4 showcases a dynamic tornado plot which illustrates the parameter sensitivity index. We calculate the sensitivity index using the given values and obtain the following values: $\beta_V = \beta_H = 1$, $b = 2$, $\mu = -0,59962$, and $\alpha = 0$. In this case, the number of mosquito bites per day is the most crucial parameter to reduce the spread of the dengue virus.

Introducing control enables the solution to reach the disease-free equilibrium point more rapidly. Figures 5–7 illustrate the dynamics of the system solutions, both without control and with control. The proportion of susceptible humans in the controlled system is higher due to lower transmission rates from control measures. Conversely, the proportion of infected humans in the controlled system is lower throughout the simulation period, highlighting the success of the implemented control strategies in restricting disease transmission. Moreover, a significant decrease in infected vectors indicates that the intervention suppresses the spread within the vector population.



(a) The solution of System (2.3) if $\hat{R}_0 < 1$.



(b) The solution of System (2.3) if $\hat{R}_0 > 1$

Figure 2. If the value of $R_0 \approx 0.22 < 1$, as shown in Figure 2a, then the solution tends to an infection-free steady state E_0. If $R_0 \approx 22.036 > 1$, then the solution tends to the endemic steady state E_1, as shown in Figure 2b.

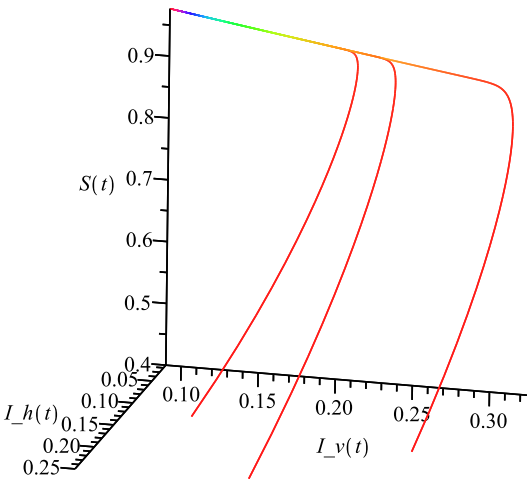


Figure 3. Phase portrait of System (2.3).

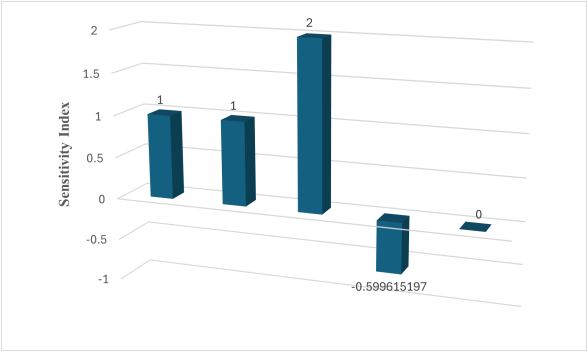
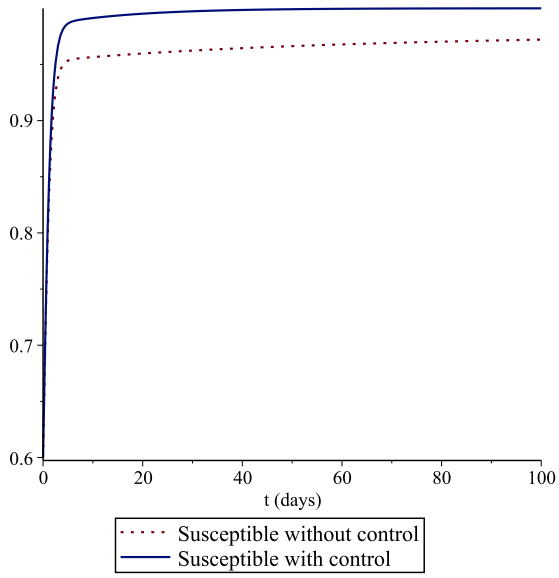
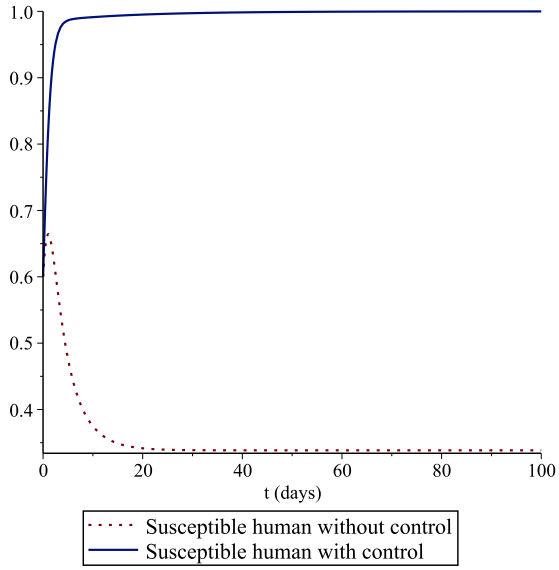


Figure 4. Sensitivity index of the parameter β_V, β_H, b, μ, α for $\hat{R}_0$.

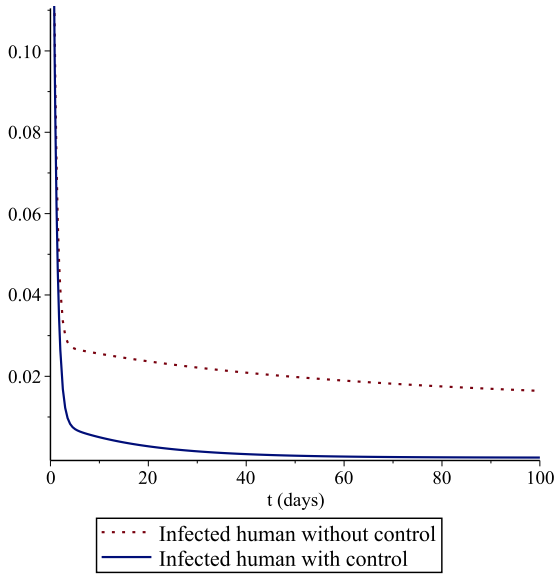


(a) Susceptible host if $\hat{R}_0 < 1$

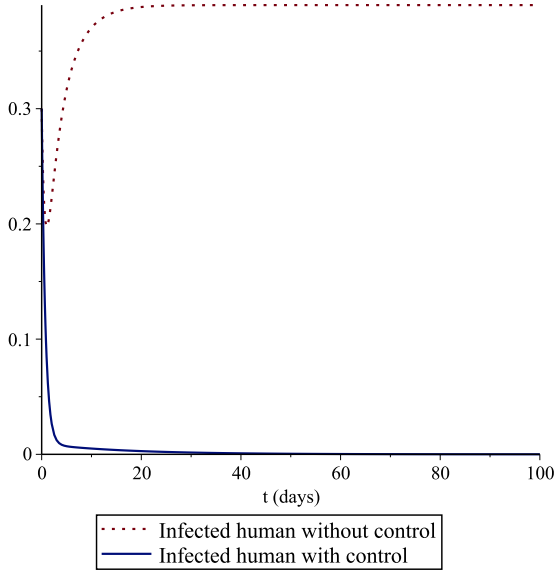


(b) Susceptible host if $\hat{R}_0 > 1$

Figure 5. Susceptible host population profile without control and with control $u_1 = 0.6$.

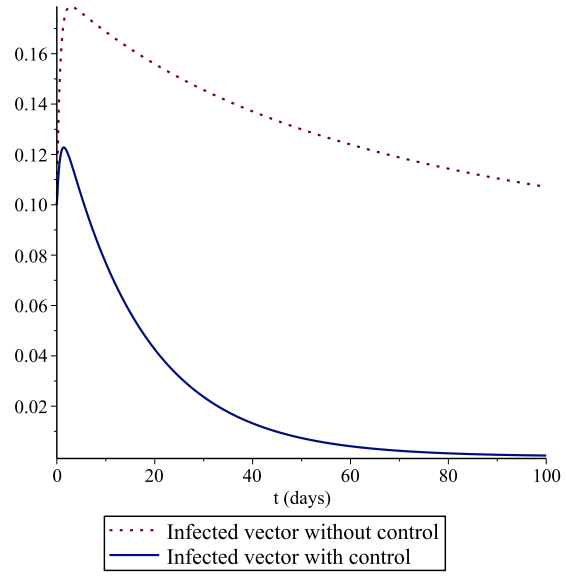


(a) Infected human if $\hat{R}_0 < 1$

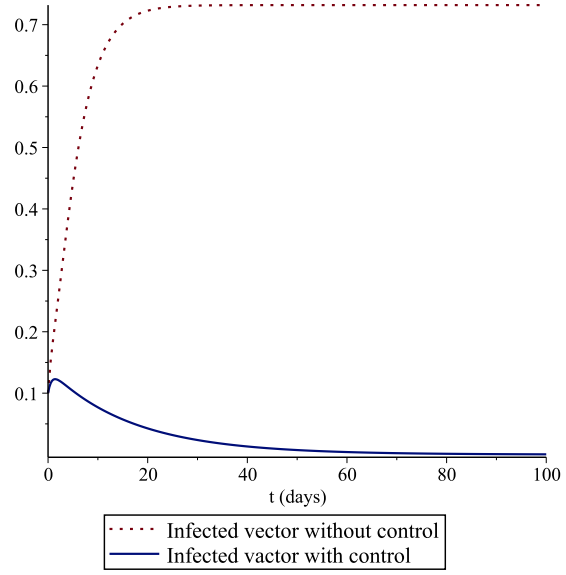


(b) Infected human if $\hat{R}_0 > 1$

Figure 6. Infected human population profile without control and with control $u_1 = 0.6$.

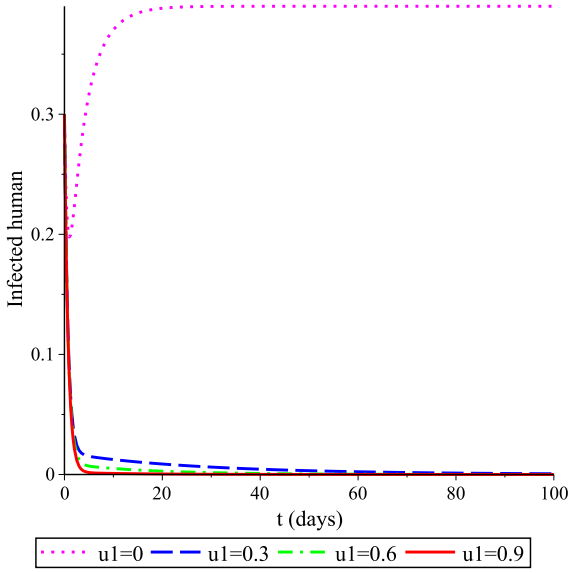


(a) Infected vector if $\hat{R}_0 < 1$

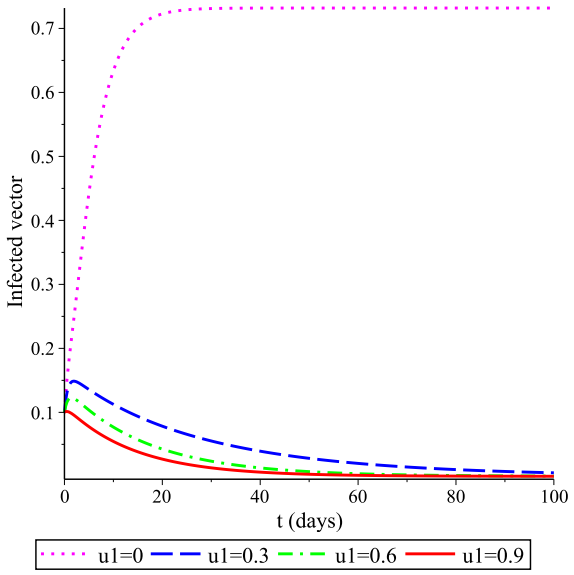


(b) Infected vector if $\hat{R}_0 > 1$

Figure 7. Infected vector population profile without control and with control $u_1 = 0.6$.



(a) Infected human with and without control



(b) Infected vector with and without control

Figure 8. Profile of infected human and infected vector if the sequences of control are applied. The more the control value, the faster the infected classes decrease.

In Figure 8, we present the profiles of infected humans and mosquitoes using several control values. It can be explained that for a system without control, the virus transmission rate is high enough to cause dengue fever to spread rapidly, so that the number of infected humans increases in a short time. This condition is a cycle; the high number of infected humans also causes the high number of infected mosquitoes, and vice versa.

The impact of the control measures aims to reduce the infected host population (i.e., minimize $G(u_1)$). These measures include providing vaccinations, community education regarding the importance of cleanliness to prevent the proliferation of mosquitoes, and promoting awareness of dengue fever symptoms. Therefore, we present the

numerical solution of the optimal control u_1^* in Figure 9 to understand the control behavior. It shows that the control measured significantly reduced the infected hosts at day 30.

Meanwhile, in a system with control, strategic intervention in the prevention efforts is optimally implemented to control the spread of disease and to reduce costs. This treatment makes the behavior of the system solution in infected humans and infected mosquitoes decrease more quickly toward a disease-free condition.

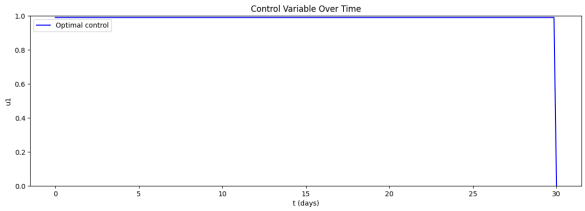


Figure 9. Optimal Control u_1.

7. Conclusions

This paper presents a detailed mathematical model of dengue virus spread in the form of a host-vector that considers the role of humans as hosts and mosquitoes as vectors. The model analysis is generally divided into two, namely the model analysis without control and the model with vector control. Based on the results of the model analysis without control, the population will be free from dengue virus infection if the basic reproduction number is less than one. Mathematically, this is proven by La-Salle Lyapunov in Theorem 3.2. On the contrary, the endemicity holds if the basic reproduction number is more than one, see Theorem 3.4.

The model is further enriched by providing control strategies through the effective prevention of the dengue virus spread. Mathematically, we use the Pontryagin Maximum Principle in Theorem 5.1, which is a powerful optimization tool in the field of control theory. Through a numerical analysis, we showed that the control strategy can significantly reduce the population of infectious classes for both the hosts and the vectors. This is a significant contribution to developing effective strategies to control the spread of dengue fever, which is a major public health problem in many parts of the world.

Our research focused on the potential for a person to be re-infected without considering the different susceptible classes. In fact, a person can be re-infected by various types of dengue viruses. It is important to remember that there are four types of viruses, and being infected with one kind of virus does not confer immunity to the other. Therefore, it can be considered that susceptible host populations to have different dengue virus infections. In terms of memory effects, utilizing a fractional model can significantly advance the research in understanding the behavior of dengue spread [7, 8, 15]. In future research, we can further develop the mathematical model for dengue by including compartments such as Exposed [26], Quarantined [27], and Treated [28]. Control measures, such as vaccination strategies [26], and Wolbachia bacterium [29] are considerable to future studies.

Use of Generative-AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Acknowledgments

The first author would like to thank Universitas Negeri Yogyakarta for the financial support. We also thank the Mathematical Biology team at Universitas Negeri Yogyakarta for the discussion.

Conflict of interest

Authors declare that they have no conflicts of interest.

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