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Mathematical modeling on the dynamics of dengue fever with vaccination and transovarial transmission with real statistical data

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Abstract
In this work, we developed a deterministic mathematical model to investigate the transmission dynamics of dengue fever while also incorporating both vaccination and transovarial transmission within the mosquito population. We conducted a mathematical analysis of the model, estimated the basic reproduction number, and examined the stability of the equilibria. By using the Center Manifold Theory, our analysis indicates the potential occurrence of backward bifurcation at the endemic equilibrium. To validate the model, we employed the actual dengue case data from Brazil during the first 30 weeks of 2024. The validation results showed strong agreement between the model projections and the observed data, which was then used for forecasting purposes. A sensitivity analysis was also carried out to identify the parameters with the most significant influence on transmission. Furthermore, the model was extended to assess two time-dependent control strategies: the use of mosquito bed nets to minimize human exposure and environmental sanitation to eliminate mosquito breeding habitats. Finally, numerical simulations demonstrated that implementing both control strategies concurrently offers a significantly greater reduction in dengue virus transmission than using either intervention individually.

Keywords Basic reproduction number · Dengue virus · Optimal control theory · Sensitivity analysis · Stability · Bifurcation · Transovarial transmission

Mathematics Subject Classification 34D20 · 92B05 · 92D30

1 Introduction

Dengue fever is a rapidly spreading viral infection transmitted by mosquitoes, predominantly affecting tropical and humid regions in Africa, Central and South America, the Caribbean, the Eastern Mediterranean, South and Southeast Asia, and Oceania Martheswaran

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(2020). The disease is caused by the Dengue virus (DENV), a member of the Flavivirus genus within the Flaviviridae family, and it has four distinct antigenic serotypes, namely DENV-1, 2, 3, and 4 Zerfu et al. (2023). The virus often spreads to humans through the bites of infected female Aedes mosquitoes Aguiar et al. (2022), with Aedes aegypti and Aedes albopictus being the main species, while Aedes aegypti serves as the primary vector of the Dengue virus. According to Naaly et al. (2024), the species Aedes aegypti is more prevalent in tropical and subtropical regions, while the species Aedes albopictus has broader adaptability to both low and high temperate regions.

In terms of transmission, the virus is passed to a person when an infected mosquito's saliva, containing the virus, enters the bloodstream. Once in the human body, DENV infects immune cells and spreads through the lymphatic system, producing a wide range of symptoms from mild fever and body aches to severe hemorrhagic manifestations Ferreira-de-Lima and Lima-Camara (2018). In addition to direct transmission through mosquito bites, female mosquitoes can also pass the virus to their offspring through transovarial (vertical) transmission, a mechanism that enables the virus to persist within mosquito populations even in the absence of human hosts Ferreira-de-Lima and Lima-Camara (2018); Namirimu and Kim (2024). This vertical transmission process ensures the continuous circulation of the virus, making vector control efforts more challenging.

Although direct human-to-human transmission does not occur, the DENV virus can be transmitted vertically to a child from an infected mother during pregnancy or childbirth. Congenital Dengue infections can result in complications such as low birth weight, premature birth, or, in severe cases, neonatal Dengue fever. In rare instances, transmission may also occur through blood transfusions, organ transplants, or accidental exposure through needlestick injuries, especially in healthcare settings where proper screening and safety measures are not strictly followed Namirimu and Kim (2024); Kulkarni et al. (2019).

The typical incubation period for Dengue fever ranges from 4 to 10 days after exposure to an infected mosquito Namirimu and Kim (2024), during which symptoms begin to manifest. Recovery from one of the Dengue virus serotypes often provides lifelong immunity to that infecting serotype but only temporary or, in some cases, partial immunity to others. As a result, subsequent infections with different serotypes increase the risk of developing severe Dengue Ogunlade et al. (2023).

It is worth noting that the majority of Dengue cases occur in the Asia-Pacific region, which accounts for approximately 70% of global cases, followed by Africa (16%), North and South America (14%), and the Oceania region (0.2%) Bhatt et al. (2013); Kline et al. (2013); Damtew et al. (2023). It has been established that climatic factors—especially temperature—are highly significant in virus transmission dynamics, as elevated temperatures beyond regional thresholds tend to favor the survival and proliferation of Dengue vectors, particularly Aedes aegypti and Aedes albopictus. According to Damtew et al. (2023); Butterworth et al. (2017), warmer conditions can accelerate viral replication, shorten the extrinsic incubation period, and increase mosquito biting rates, thereby intensifying disease transmission.

The clinical manifestation of Dengue fever varies, ranging from asymptomatic cases to severe forms. The common symptoms include self-limiting fever, muscle pain, headache, leukopenia, and rash, usually lasting 5–7 days. In more severe cases, patients may experience nausea, vomiting, petechiae, and life-threatening complications such as Dengue hem-

orrhagic fever (DHF) or Dengue shock syndrome (DSS) Argüello et al. (2015); Chen et al. (2023).

Due to the severity of this disease, Dengue mitigation requires supportive care, symptom relief, and close monitoring to prevent complications. Some pharmaceutical interventions include the use of acetaminophen (paracetamol) and other analgesics to manage fever and alleviate pain Namirimu and Kim (2024). In most cases, patients with Dengue fever recover within 2–7 days without hospitalization. However, severe cases may require hospitalization for supportive care, including blood transfusions, intravenous fluids, electrolyte replacement, and close monitoring Namirimu and Kim (2024); Tayal et al. (2023).

Vaccination has also been considered as a measure for mitigating the spread, and two vaccines—Dengvaxia (CYD-TDV) and QDENGA (TAK-003)—have been approved for public use, while several others are currently in various stages of research and development Namirimu and Kim (2024); Lenharo (2023). The Dengvaxia vaccine is recommended for individuals between the ages of 9 and 45 who have had a previous Dengue infection. The vaccine has demonstrated an overall efficacy of approximately 60%, though its use is limited due to an increased risk of severe Dengue in individuals who have not been previously infected. In contrast, the TAK-003 vaccine, endorsed by the WHO and approved in several countries, has demonstrated promising efficacy rates—reducing symptomatic Dengue by 61.2%, hospitalizations by 84%, and DHF by around 70% Namirimu and Kim (2024); Lenharo (2023).

Over the last two decades, mathematical modeling has become an essential tool for studying infectious disease transmission. Several models have been developed to analyze Dengue dynamics, including its interactions with co-circulating pathogens (e.g., Carvalho et al. (2019); Nuraini et al. (2007); Edussuriya et al. (2021); Jan et al. (2020); Alemneh (2020)). Sa'adah and Sari (2023) developed a model incorporating vaccination and Wolbachia-based control strategies, examining seasonal influences by introducing a periodic mosquito birth rate. Their analysis revealed that seasonal fluctuations drive recurring mosquito population patterns annually. Aldila et al. (2023) constructed a model to evaluate the role of public awareness, case detection, and healthcare capacity in Dengue control in Jakarta. Their findings suggested that media campaigns were more effective in reducing the basic reproduction number than case detection alone. Furthermore, they observed that increasing case detection did not always lead to better outcomes due to variations in healthcare quality. Kumar et al. (2024) proposed a transmission model emphasizing the impact of reducing mosquito biting rates and enhancing human recovery rates on Dengue control. Yoda et al. (2024) developed an optimal control model incorporating treatment strategies and prevention measures to minimize human–mosquito contact. Pongsumpun et al. (2023) expanded on this approach by integrating vaccination into their control strategies, demonstrating through simulations that optimal interventions significantly accelerated infection eradication. Olarte and Munoz (2021) introduced a Dengue model that accounts for periodic environmental fluctuations, highlighting the role of environmental variability in disease transmission. Naaly et al. (2024) examined the combined effects of vector control, treatment, and public awareness, finding that a multi-pronged strategy was more effective than any single or dual intervention. Jose et al. (2023) formulated a model to investigate the co-dynamics of Zika and Dengue infections, while Omame et al. (2023) developed a multi-disease model assessing the interactions between COVID-19, Zika, chikungunya, and Dengue. Their study indicated that preventive

measures targeting Zika, Dengue, and chikungunya could substantially reduce the co-infection burden with COVID-19.

In this work, we constructed a deterministic mathematical model to investigate the transmission dynamics of the Dengue virus. The model takes into account key factors such as vaccination for individuals with previous DENV infections and transovarial (vertical) transmission in mosquitoes, while recognizing that the Dengue virus can sustain itself within mosquito populations even in the absence of human hosts. Given these factors, we conducted a comprehensive analysis of the model and introduced two time-dependent control measures: the use of mosquito bed nets to prevent susceptible individuals from contacting DENV and environmental sanitation efforts, such as clearing waterlogged areas to eliminate mosquito breeding sites. The structure of the remainder of this paper is as follows: Section 2 covers the construction of the mathematical model and its basic properties; Section 3 presents the model analysis; Section 4 discusses the extension of the Dengue model with optimal control; and Section 5 provides the concluding remarks.

2 Construction of the dengue transmission mathematical model

The construction of a mathematical model has been crucial for understanding the complex dynamics of biological systems and this will also be applied to the dynamics of the Dengue transmission dynamics. So, to comprehensively understand the transmission dynamics of Dengue fever, we develop a Dengue epidemiological model that incorporates both the human and mosquito (vector) populations, primarily consisting of Aedes aegypti mosquitoes. At any given time *t*, the total human population, denoted as $N_H(t)$, is categorized into seven groups namely: susceptible individuals $(S_H(t))$, individuals exposed to the Dengue virus $(E_H(t))$, individuals infected with Dengue but asymptomatic $(I_A(t))$, symptomatic infected individuals $(I_S(t))$, treated individuals $(T_H(t))$, individuals who have recovered from Dengue $(R_H(t))$, and individuals vaccinated against Dengue $(V_H(t))$. The total human population can thus be represented mathematically as:

$$N_H(t) = S_H(t) + E_H(t) + I_A(t) + I_S(t) + T_H(t) + R_H(t) + V_H(t).$$

In addition, at any given time *t*, the total mosquito (vector) population, denoted as $N_V(t)$, is divided into three categories: susceptible mosquitoes $(S_V(t))$, mosquitoes exposed to the Dengue virus $(E_V(t))$, and mosquitoes infected with the Dengue virus $(I_V(t))$. Therefore, the total mosquito population is expressed mathematically as:

$$N_V(t) = S_V(t) + E_V(t) + I_V(t).$$

The susceptible individuals class is replenished by the influx of individuals into the population, either through birth or immigration, at a rate α_H. Susceptible individuals become infected with the Dengue virus (DENV) upon exposure to infected mosquitoes carrying the virus, at a rate λ_H. This rate is given by the expression:

$$\lambda_H = \frac{\zeta \beta_H I_V}{N_H},$$

where ζ represents the mosquito-biting rate, while β_H is the transmission probability that an infected mosquito with DENV transmits the virus to susceptible humans. Individuals in the exposed class advance to the infected class at a rate η, with a fraction (q) of them developing symptoms of Dengue fever, and the remaining fraction $(1-q)$ remaining asymptomatic. The rate at which asymptomatic infected individuals transition to symptomatic cases is denoted by ψ. Additionally, based on existing literature, it is known that a significant portion of individuals infected with DENV remain asymptomatic. As such, we assume in the model formulation that DENV-induced mortality occurs only among individuals with severe symptomatic cases, at a rate δ. The rates τ_1 and τ_2 represent the treatment rates for asymptomatic and symptomatic infected individuals, respectively. DENV-induced mortality occurs in the treated class at a rate $\theta\delta$, where θ is a modification parameter that accounts for the reduction in Dengue-related death due to treatment. Treated individuals recover from Dengue fever at a rate σ. Recovered individuals can be re-infected with Dengue fever at a rate $\omega\lambda_H$, where ω represents the re-nfection factor. Among the re-infected individuals, a proportion (k) remain asymptomatic, while the remaining proportion $(1-k)$ develop symptomatic cases. It is important to note that the Dengue fever vaccine (Dengvaxia) is administered exclusively to individuals who have previously been infected and recovered from Dengue fever. This is represented in the model by the rate ξ. Additionally, there is a vaccine-waning rate φ, which accounts for the gradual loss of vaccine-induced immunity over time. The natural death rate for humans, denoted by γ_H, is a constant and applies uniformly across all human classes.

The introduction of new mosquitoes into the susceptible mosquito population occurs at a rate of $(1-\phi I_V)\alpha_V$, where α_V is the rate at which new mosquitoes are introduced into the population, and ϕ represents the fraction of new mosquitoes that are already infected with DENV due to transovarial transmission (the transmission of the virus from the mother mosquito to her offspring). Susceptible mosquitoes become exposed to DENV after taking a blood meal from an infected individual. The exposure rate for mosquitoes, denoted by λ_V, is given by:

$$\lambda_V = \frac{\zeta\beta_V(I_A + I_S)}{N_H},$$

where β_V is the probability of DENV transmission from infected humans to susceptible mosquitoes. Exposed mosquitoes transition to the infected class at a rate μ. The death rate of mosquitoes is represented by γ_V, which is constant and applies uniformly across all mosquito compartments.

Based on the above formulations and assumptions, the dynamics of the Dengue model can be described by a system of non-linear differential equations. These equations govern the changes in the various compartments of the human and mosquito populations over time. The system of equations is as follows:

$$
\begin{aligned}
\frac{dS_H}{dt} &= \alpha_H - \lambda_H S_H - \gamma_H S_H + \varphi V_H,\\
\frac{dE_H}{dt} &= \lambda_H S_H - (\eta + \gamma_H) E_H,\\
\frac{dI_A}{dt} &= (1-q)\eta E_H - (\psi + \tau_1 + \gamma_H) I_A + k\omega\lambda_H R_H,\\
\frac{dI_S}{dt} &= q\eta E_H + \psi I_A - (\tau_2 + \delta + \gamma_H) I_S + (1-k)\omega\lambda_H R_H,\\
\frac{dT_H}{dt} &= \tau_1 I_A + \tau_2 I_S - (\sigma + \theta\delta + \gamma_H) T_H,\\
\frac{dR_H}{dt} &= \sigma T_H - (\xi + \omega\lambda_H + \gamma_H) R_H,\\
\frac{dV_H}{dt} &= \xi R_H - (\varphi + \gamma_H) V_H,\\
\frac{dS_V}{dt} &= (1 - \phi I_V)\alpha_V - \lambda_V S_V - \gamma_V S_V,\\
\frac{dE_V}{dt} &= \lambda_V S_V - (\mu + \gamma_V) E_V,\\
\frac{dI_V}{dt} &= \mu E_V + \phi\alpha_V I_V - \gamma_V I_V.
\end{aligned}
\tag{1}
$$

where

$$
\lambda_H = \frac{\zeta\beta_H I_V}{N_H}, and \quad \lambda_V = \frac{\zeta\beta_V (I_A + I_S)}{N_H}.
$$

This system of differential equations describes the interactions between the human and mosquito populations, capturing the dynamics of susceptible, exposed, infected, treated, recovered, and vaccinated individuals, as well as the mosquito life cycle and infection process (Table 1).

2.1 Basic properties of the dengue model

2.1.1 Positivity of solution

To ensure that the Dengue model (1) is biologically meaningful, it is necessary to show that all the state variables of the Dengue model is positive at all time $t \geq 0$ in the feasible region χ, given by

$$
\chi = \chi_H \cup \chi_V \subset \mathbb{R}_+^7 \times \mathbb{R}_+^3, \tag{2}
$$

where

$$
\chi_H = \left\{ (S_H, E_H, I_A, I_S, T_H, R_H, V_H) \in \mathbb{R}_+^7 : N_H \leq \frac{\alpha_H}{\gamma_H} \right\},
$$

and

Table 1 Description of the model variables and parameters

Variable	Description
S_H	Susceptible humans
E_H	Exposed humans to DENV
I_A	Asymptomatic infected humans with DENV
I_S	Symptomatic infected humans with DENV
T_H	Treated humans with DENV
R_H	Recovered humans from DENV
S_V	Susceptible mosquitoes (vectors)
E_V	Exposed mosquitoes (vectors) to DENV
I_V	Infected mosquitoes (vectors) to DENV
Parameter	Description
α_H	Introduction rate of humans into the population
α_V	Introduction rate of new mosquitoes into the population
β_H	The transmission probability from infected mosquitoes with DENV to susceptible individuals
β_V	The transmission probability from DENV infected humans to susceptible mosquitoes
ζ	Mosquito biting rate
γ_H	Human natural death rate
η	Human advancement rate from being exposed to being infected
q	Proportion of exposed individuals that advanced to being infected that are symptomatic
ψ	Human advancement rate from infected asymptomatic to infected symptomatic
τ_1	Treatment rate of asymptomatic infected individuals with Dengue
τ_2	Treatment rate of symptomatic infected individuals with Dengue
δ	Dengue induced death rate
θ	Modification parameter that accounts for reduction in Dengue induced death in the treated class
σ	Recovery rate of treated individuals
ξ	Vaccination rate
φ	Waning rate of Dengue vaccine
ω	Modification parameter that accounts for Dengue re-infection
k	Proportion of Dengue re-infected individuals that are asymptomatic
ϕ	Transovarial transmission rate
μ	Mosquito advancement rate from being exposed to being infected
γ_V	Mosquito death rate

$$\chi_V = \left\{ (S_V, E_V, I_V) \in \mathbb{R}^3_+ : N_V \leq \frac{\alpha_V}{\gamma_V} \right\}.$$

Theorem 1 *Let the initial data for the Dengue model (1) be* $S_H(0) \geq 0$, $E_H(0) \geq 0$, $I_A(0) \geq 0$, $I_S(0) \geq 0$, $T_H(0) \geq 0$, $R_H(0) \geq 0$, $V_H(0) \geq 0$, $S_V(0) \geq 0$, $E_V(0) \geq 0$, $I_V(0) \geq 0$. *Then the solution* (S_H, E_H, I_A, I_S, T_H, R_H, V_H, S_V, E_V, I_V) *of the Dengue model (1) are positive for all time* $t \geq 0$.

Proof The first equation of the Dengue model system (1) can be explicitly written as

$$\frac{dS_H}{dt} = \alpha_H - \lambda_H S_H - \gamma_H S_H + \varphi V_H$$

From the above equation, we have that

$$\frac{dS_H}{dt} \geq -\lambda_H S_H - \gamma_H S_H. \tag{3}$$

Which can be re-written as

$$\frac{dS_H}{dt} \geq -\left(\lambda_H + \gamma_H\right) S_H.$$

Using the method of separation of variables, we have

$$\int \frac{dS_H}{S_H} \geq -\int \left(\lambda_H + \gamma_H\right) dt.$$

Integrating the above equation yields

$$\ln S_H \geq -\left(\lambda_H + \gamma_H\right) t + C.$$

Which gives

$$S_H(t) \geq Ce^{-(\lambda_H + \gamma_H)t}. \tag{4}$$

Applying the initial condition, when $t = 0$, we have

$$S_H(0) \geq Ce^{-(\lambda_H + \gamma_H)(0)},$$
$$S_H(0) \geq Ce^0$$

we have that

$$S_H(0) \geq C$$

Therefore, form Eq. (4) we obtained

$$S_H(t) \geq S_H(0)e^{-(\lambda_H+\gamma_H)t} > 0. \tag{5}$$

Employing similar approach, we have

$E_H(t) \geq E_H(0)e^{-(\eta+\gamma_H)t} > 0, I_A(t) \geq I_A(0)e^{-(\psi+\tau_1+\gamma_H)t} > 0, I_S(t) \geq I_S(0)e^{-(\tau_2+\delta+\gamma_H)t} > 0,$
$T_H(t) \geq T_H(0)e^{-(\sigma+\theta\delta+\gamma_H)t} > 0, R_H(t) \geq R_H(0)e^{-(\xi+\omega\lambda_H+\gamma_H)t} > 0, V_H(t) \geq V_H(0)e^{-(\varphi+\gamma_H)t} > 0,$
$S_V(t) \geq S_V(0)e^{-(\lambda_V+\gamma_V)t} > 0, E_V(t) \geq E_V(0)e^{-(\mu+\gamma_V)t} > 0, I_V(t) \geq I_V(0)e^{-\gamma_V t} > 0.$

Hence, all the solution sets are positive for $t \geq 0$. □

2.1.2 Invariant region

Lemma 1 *The closed region χ in (2) is a positively invariant set for the Dengue model (1) with positive initial conditions in $\mathbb{R}_+^{10}$.*

Proof By adding the human compartments of the Dengue model (1), the rate of change of the total human population is obtained in the form of

$$\frac{dN_H}{dt} = \alpha_H - \gamma_H N_H - \delta I_S - \theta\delta T_H. \tag{6}$$

Similarly, by adding the mosquito compartment of the Dengue model (1), the rate of change of the total mosquito population is obtained in the form of

$$\frac{dN_V}{dt} = \alpha_V - \gamma_V N_V. \tag{7}$$

Thus, whenever $N_H > \frac{\alpha_H}{\gamma_H}$ and $N_V > \frac{\alpha_V}{\gamma_V}$, then $\frac{dN_H}{dt} < 0$ and $\frac{dN_V}{dt} < 0$. Hence, the right hand side of Eqs. (6) and (7) are bounded by $\alpha_H - \gamma_H N_H$ and $\alpha_V - \gamma_V N_V$ respectively. Using the Comparison theorem in Lakshmikantham et al. (1989), it can be shown that

$$N_H(t) \leq N_H(0)e^{-\gamma_H t} - \frac{\alpha_H}{\gamma_H}(1 - e^{-\gamma_H t}), \tag{8}$$

and

$$N_V(t) \leq N_V(0)e^{-\gamma_V t} - \frac{\alpha_V}{\gamma_V}(1 - e^{-\gamma_V t}). \tag{9}$$

It follows that $N_H(t) \leq \frac{\alpha_H}{\gamma_H}$ and $N_V(t) \leq \frac{\alpha_V}{\gamma_V}$, if $N_H(0) \leq \frac{\alpha_H}{\gamma_H}$ and $N_V(0) \leq \frac{\alpha_V}{\gamma_V}$. Therefore, the closed region χ is positively invariant and attracts all the solution in $\mathbb{R}_+^{10}$. Hence, the Dengue model (1) is biologically and mathematically well posed in the invariant set χ Hethcote (2000). Thus, it is satisfactory to consider the dynamics of the Dengue model in the invariant set χ. □

3 Model analysis

This section presents the qualitative analysis of the Dengue model (1). The analysis will entail the computation of the basic reproduction number as well as the examination of the stability of the Dengue equilibrium points.

3.1 Dengue-free equilibrium point

To determine the Dengue-free equilibrium (DFE) of the system, we need to set all the infected variables to zero, as the DFE represents the steady state where there are no infected individuals in the population. This means that the classes of infected humans (I_A, I_S), exposed humans (E_H), infected mosquitoes (I_V), and exposed mosquitoes (E_H) will all be zero at the equilibrium. Thus, setting the right-hand side of the differential equations to zero and solving for the non-infected variables yields the Dengue-free equilibrium, given by

$$\ell_0 = (S_H^*, E_H^*, I_A^*, I_S^*, T_H^*, R_H^*, V_H^*, S_V^*, E_V^*, I_V^*) = \left(\frac{\alpha_H}{\gamma_H}, 0, 0, 0, 0, 0, \frac{\alpha_V}{\gamma_V}, 0, 0\right) \quad (10)$$

3.2 The basic reproduction number

Here, we shall discuss the basic reproduction number, often denoted by $\mathcal{R}_0$ and it represents the expected number of secondary infections generated by a single infectious individual introduced into a fully susceptible population Van den Driessche and Watmough (2002). It is a key parameter that helps assess whether an outbreak will occur or die out. To compute $\mathcal{R}_0$, we apply the Next-Generation Matrix Approach as described in Van den Driessche and Watmough (2002) and applied in literature such as Babasola et al. (2023); Omondi et al. (2024). We define non-negative matrix $\mathcal{F}$ and the non-singular matrix $\mathcal{V}$, which represent the new infection terms and the remaining transition terms at the Dengue-free equilibrium point. These are obtained as

$$\mathcal{F} = \begin{bmatrix} \frac{\zeta\beta_H I_V S_H}{N_H} \\ 0 \\ 0 \\ 0 \\ \frac{\zeta\beta_V (I_A + I_S) S_V}{N_H} \\ \phi\alpha_V I_V \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} (\eta + \gamma_H) E_H \\ -(1-q)\eta E_H + (\psi + \tau_1 + \gamma_H) I_A \\ -q\eta E_H - \psi I_A + (\tau_2 + \delta + \gamma_H) I_S \\ -\tau_1 I_A - \tau_2 I_S + (\sigma + \theta\delta + \gamma_H) T_H \\ (\mu + \gamma_V) E_V \\ -\mu E_V + \gamma_V I_V \end{bmatrix}$$

Evaluating $\mathcal{F}$ and $\mathcal{V}$ at the Dengue-free equilibrium, we have

$$\mathcal{F} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & \zeta\beta_H \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\zeta\beta_V \alpha_V \gamma_H}{\alpha_H \gamma_V} & \frac{\zeta\beta_V \alpha_V \gamma_H}{\alpha_H \gamma_V} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi\alpha_V \end{bmatrix},$$

$$\mathcal{V} = \begin{bmatrix} (\eta+\gamma_H) & 0 & 0 & 0 & 0 & 0 \\ -(1-q)\eta & (\psi+\tau_1+\gamma_H) & 0 & 0 & 0 & 0 \\ -q\eta & -\psi & (\tau_2+\delta+\gamma_H) & 0 & 0 & 0 \\ 0 & -\tau_1 & -\tau_2 & (\sigma+\theta\delta+\gamma_H) & 0 & 0 \\ 0 & 0 & 0 & 0 & (\mu+\gamma_V) & 0 \\ 0 & 0 & 0 & 0 & -\mu & \gamma_V \end{bmatrix}$$

$$\mathcal{FV}^{-1} = \begin{bmatrix} 0 & 0 & 0 & 0 & \Upsilon_4 & \Upsilon_6 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \Upsilon_1 & \Upsilon_2 & \Upsilon_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \Upsilon_5 & \Upsilon_7 \end{bmatrix},$$

where $\Upsilon_1 = \frac{\zeta\beta_V\alpha_V\gamma_H D_1}{\alpha_H\gamma_V D_1 D_3} + \frac{\zeta\beta_V\alpha_V\gamma_H(q\eta D_3+\psi D_2)}{\alpha_H\gamma_V D_1 D_3 D_4}$, $\Upsilon_2 = \frac{\zeta\beta_V\alpha_V\gamma_H}{\alpha_H\gamma_V D_3} + \frac{\zeta\beta_V\alpha_V\gamma_H\psi}{\alpha_H\gamma_V D_3 D_4}$, $\Upsilon_3 = \frac{\zeta\beta_V\alpha_V\gamma_H}{\alpha_H\gamma_V D_4}$, $\Upsilon_4 = \frac{\zeta\beta_H\mu}{\gamma_V D_9}$, $\Upsilon_5 = \frac{\phi\alpha_V\mu}{\gamma_V D_9}$, $\Upsilon_6 = \frac{\zeta\beta_H}{\gamma_V}$, $\Upsilon_7 = \frac{\phi\alpha_V}{\gamma_V}$.

With $D_1 = \eta+\gamma_H$, $D_2 = (1-q)\eta$, $D_3 = \psi+\tau_1+\gamma_H$, $D_4 = \tau_2+\delta+\gamma_H$, $D_5 = (1-k)\omega$, $D_6 = \sigma+\theta\delta+\gamma_H$, $D_7 = \xi+\gamma_H$, $D_8 = \varphi+\gamma_H$, and $D_9 = \mu+\gamma_V$.

Hence, it follows from Van den Driessche and Watmough (2002) that $\mathcal{R}_0 = \rho(\mathcal{FV}^{-1})$, where ρ is the spectral radius or dominant eigenvalues of the matrix $\mathcal{FV}^{-1}$.

The characteristic polynomial of the matrix $\mathcal{FV}^{-1}$, is given by

$$\lambda^4(\lambda^2 - \Theta_1\lambda - \Theta_2) \tag{11}$$

where

$$\Theta_1 = \frac{\phi\alpha_V}{\gamma_V}, and \quad \Theta_2 = \frac{\zeta^2\beta_H\beta_V\alpha_V\gamma_H\mu(q\eta D_3 + \psi D_2 + D_2D_4)}{\alpha_H\gamma_V^2 D_1 D_3 D_4 D_9}. \tag{12}$$

The quadratic equation for the characteristic polynomial given in (11) can be therefore written as $\mathscr{S}(\lambda) = \left(\lambda^2 - \Theta_1\lambda - \Theta_2\right)$, with $\Theta_1 > 0, \Theta_2 > 0$. Hence, the associated solution of $\mathscr{S}(\lambda) = 0$ gives a unique and positive root which is the dominant eigenvalue of the basic reproduction number of the Dengue model, given by

$$\mathcal{R}_0 = \frac{1}{2}\left(\Theta_1 + \left(\Theta_1^2 + 4\Theta_2\right)^{\frac{1}{2}}\right) \tag{13}$$

Furthermore, $\mathscr{S}(0) = -\Theta_2 < 0$, and $\mathscr{S}(1) = 1 - (\Theta_1 + \Theta_2)$. We define $\mathcal{R}_0^D = \Theta_1 + \Theta_2$, given by

$$\mathcal{R}_0^D = \frac{\phi\alpha_V}{\gamma_V} + \frac{\zeta^2\beta_H\beta_V\alpha_V\gamma_H\mu(q\eta D_3 + \psi D_2 + D_2D_4)}{\alpha_H\gamma_V^2 D_1 D_3 D_4 D_9} \tag{14}$$

That is

$$\mathcal{R}_0^D = \mathcal{R}_{0T}^D + \mathcal{R}_{0HV}^D,$$

where $\mathcal{R}_{0T}^D = \frac{\phi\alpha_V}{\gamma_V}$ is the contribution from transovarial transmission, and

$\mathcal{R}_{0HV}^{D} = \frac{\zeta^2\beta_H\beta_V\alpha_V\gamma_H\mu(q\eta D_3+\psi D_2+D_2D_4)}{\alpha_H\gamma_V^2 D_1D_3D_4D_9}$ is the contribution from human and vector interaction. Again, from the relation $\mathscr{S}(1) = 1 - \mathcal{R}_0^D$, we obtained the following

(i) When $\mathcal{R}_0^D = 1$ then $\mathscr{S}(1) = 0$, thus the nonnegative root of the equation $\mathscr{S}(\lambda) = 0$ is one. That is, $\mathcal{R}_0 = 1$.
(ii) When $\mathcal{R}_0^D < 1$ then $\mathscr{S}(1) > 0$, so the nonnegative root of the equation lies within the interval 0 and 1 as $\mathscr{S}(0) < 0$, thus $\mathcal{R}_0 < 1$.
(iii) When $\mathcal{R}_0^D > 1$ then $\mathscr{S}(1) < 0$ again $\mathscr{S}(0)\,0$, so the nonnegative root $\mathscr{S}(\lambda) = 0$ is greater than one (that is $\mathcal{R}_0 > 1$).

Based on the above observations, one can conclude that $\mathcal{R}_0^D = 1$ if and only if $\mathcal{R}_0 = 1$. Similarly, $\mathcal{R}_0^D < 1$ and $\mathcal{R}_0^D > 1$ if and only if $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$ respectively. Thus, since the two threshold parameter $\mathcal{R}_0$ and $\mathcal{R}_0^D$ are equivalent as also noted in Jose et al. (2023); Oguntolu et al. (2024). Hence, we shall be using $\mathcal{R}_0^D$ as the Dengue basic reproduction number in the subsequent sections.

3.3 The local stability of the dengue fever-free equilibrium

Theorem 2 *The Dengue fever-free equilibrium (ℓ_0) of the Dengue model (1) is locally (asymptotically) stable if $\mathcal{R}_0^D < 1$, and unstable whenever $\mathcal{R}_0^D > 1$.*

Proof The local stability of the disease-free equilibrium is examined by deriving the Jacobian matrix of the Dengue model (1) and evaluating it at the disease-free equilibrium point (ℓ_0), given by

$$\mathcal{J}(\ell_0) = \begin{bmatrix} -\gamma_H & 0 & 0 & 0 & 0 & 0 & \varphi & 0 & 0 & -\zeta\beta_H \\ 0 & -D_1 & 0 & 0 & 0 & 0 & \varphi & 0 & 0 & \zeta\beta_H \\ 0 & D_2 & -D_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & q\eta & \psi & -D_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_1 & \tau_2 & -D_6 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma & -D_7 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \xi & -D_8 & 0 & 0 & 0 \\ 0 & 0 & -\hbar & -\hbar & 0 & 0 & 0 & -\gamma_V & 0 & -\phi\alpha_V \\ 0 & 0 & \hbar & \hbar & 0 & 0 & 0 & 0 & -D_9 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mu & \phi\alpha_V - \gamma_V \end{bmatrix}$$

where

$D_1 = \eta + \gamma_H, D_2 = (1-q)\eta, D_3 = \psi + \tau_1 + \gamma_H, D_4 = \tau_2 + \delta + \gamma_H, D_5 = (1-k)\omega,$

$D_6 = \sigma + \theta\delta + \gamma_H$, $D_7 = \xi + \gamma_H$, $D_8 = \varphi + \gamma_H$, $D_9 = \mu + \gamma_V$, and $\hbar = \frac{\zeta\beta_V\alpha_V\gamma_H}{\alpha_H\gamma_V}$.

The eigenvalues of the Jacobian matrix $\mathcal{J}(\ell_0)$ above are $\lambda_1 = -\gamma_H$, $\lambda_2 = -\gamma_V$, $\lambda_3 = -D_8$, $\lambda_4 = -D_7$, $\lambda_5 = -D_6$, and the roots of the characteristic polynomial given below

$$\mathscr{A}(\lambda) = \lambda^5 + \mathcal{P}_1\lambda^4 + \mathcal{P}_2\lambda^3 + \mathcal{P}_3\lambda^2 + \mathcal{P}_4\lambda + \mathcal{P}_5, \tag{15}$$

with

$$\mathcal{P}_1 = D_1 + D_3 + D_4 + D_9 + \gamma_V - \phi\alpha_V,$$
$$\mathcal{P}_2 = D_1(D_3 + D_4 + D_9 + \gamma_V) + D_3(D_4 + D_9 + \gamma_V) + D_4(D_9 + \gamma_V) + D_9\gamma_V - \phi\alpha_V(D_1 + D_3 + D_4 + D_9),$$

$$\mathcal{P}_3 = D_1D_3(D_4 + D_9 + \gamma_V) + D_1D_4(D_9 + \gamma_V) + \gamma_V(D_1D_9 + D_4D_9 + D_3D_4) + D_3D_9(D_4 + \gamma_V) - \phi\alpha_V(D_1(D_3 + D_4 + D_9) + D_3(D_4 + D_9) + D_4D_9),$$
$$\mathcal{P}_4 = D_1D_3D_4(D_9 + \gamma_V) + \gamma_V D_9(D_1D_3 + D_1D_4 + D_3D_4) - \phi\alpha_V(D_1D_3(D_4 + D_9) + D_4D_9(D_1 + D_3)) - \frac{\zeta^2\beta_H\beta_V\alpha_V\gamma_H\mu(q\eta + D_2)}{\alpha_H\gamma_V},$$
$$\mathcal{P}_5 = \gamma_V D_1D_3D_4D_9\left(1 - \left(\frac{\phi\alpha_V}{\gamma_V} + \frac{\zeta^2\beta_H\beta_V\alpha_V\gamma_H\mu(q\eta D_3 + \psi D_2 + D_2D_4)}{\alpha_H\gamma_V^2 D_1D_3D_4D_9}\right)\right) = \gamma_V D_1D_3D_4D_9\left(1 - \mathcal{R}_0^D\right).$$

Applying the Routh-Hurwitz criterion Hassan et al. (2022); Omede et al. (2023) to the polynomial (15), which states that all roots of the polynomial (15) have negative real parts if and only if the coefficient $\mathcal{P}_i > 0$, for $i = 1, 2, 3, 4, 5$, $\mathcal{P}_1\mathcal{P}_2\mathcal{P}_3 > \mathcal{P}_3^2 + \mathcal{P}_1^2\mathcal{P}_4$, and $(\mathcal{P}_1\mathcal{P}_4 - \mathcal{P}_5)(\mathcal{P}_1\mathcal{P}_2\mathcal{P}_3 - \mathcal{P}_3^2 - \mathcal{P}_1^2\mathcal{P}_4) > \mathcal{P}_5(\mathcal{P}_1\mathcal{P}_2 - \mathcal{P}_3)^2 + \mathcal{P}_1\mathcal{P}_5^2$. Clearly, for $\mathcal{P}_5 > 0$; then $\mathcal{R}_0^D < 1$. Therefore, using the Routh-Hurwitz stability criterion, the Dengue fever-free equilibrium of the Dengue model (1) is locally asymptotically stable when $\mathcal{R}_0^D < 1$.

Epidemiologically, Theorem 2 suggests that if the reproduction number $\mathcal{R}_0^D$ for Dengue fever is less than 1 ($\mathcal{R}_0^D < 1$), then a small influx of Dengue-infected individuals into the population will not lead to an outbreak. However, it is crucial to recognize that is conclusion relies on the assumption that the number of infected individuals is sufficiently low at the outset. If the initial population of infected individuals is large enough, then the disease could still spread despite $\mathcal{R}_0^D < 1$. In this case, the epidemic might not die out immediately, and as a result, a larger number of infected individuals could sustain transmission temporarily, potentially leading to a localized outbreak or larger-scale transmission, even if the long-term dynamics suggest that the disease cannot sustain itself in the population. Thus, while $\mathcal{R}_0^D < 1$ indicates that the disease will eventually fade out if left unchecked, the actual trajectory of the epidemic in the short term will depend on the initial population of the infected individuals and the dynamics of disease spread within the population. □

3.4 Existence and stability of the dengue endemic equilibrium

In this section, we aim to explore the conditions for the existence of the Dengue endemic equilibrium point, which occurs when the infected variables are non-zero. To determine these conditions, we analyze the Dengue model (1) by expressing it in terms of the forces of infection, as defined by the following equations:

$$\lambda_H^{**} = \frac{\zeta\beta_H I_V^{**}}{N_H^{**}}, \quad \text{and} \quad \lambda_V^{**} = \frac{\zeta\beta_V(I_A^{**} + I_S^{**})}{N_H^{**}}. \tag{16}$$

where λ_H^{**} represents the force of infection for the human population while λ_V^{**} represents the force of infection for the mosquito population at the Dengue endemic equilibrium point. Thus, we have

$$N_H^{**} = S_H^{**} + E_H^{**} + I_A^{**} + I_S^{**} + T_H^{**} + R_H^{**} + V_H^{**}. \tag{17}$$

With

$$\begin{aligned}
S_H^{**} &= \frac{\alpha_H D_1 D_3 D_4 D_6 D_8(\omega\lambda_H^{**} + D_7) - \lambda_H^{**}\alpha_H\sigma D_1 D_8 \mathscr{C}_1}{\mathcal{O}_1},\\
E_H^{**} &= \frac{\lambda_H^{**}\alpha_H D_3 D_4 D_6 D_8(\omega\lambda_H^{**} + D_7) - \lambda_H^{**2}\alpha_H\sigma D_8 \mathscr{C}_1}{\mathcal{O}_1},\\
I_A^{**} &= \frac{\lambda_H^{**}\alpha_H D_2 D_4 D_6 D_8(\omega\lambda_H^{**} + D_7) + \lambda_H^{**2}\alpha_H\sigma\tau_2 D_8(q\eta k\omega - D_2 D_5)}{\mathcal{O}_1},\\
I_S^{**} &= \frac{\lambda_H^{**}\alpha_H D_6 D_8(\omega\lambda_H^{**} + D_7)(q\eta D_3 + \psi D_2) + \lambda_H^{**2}\alpha_H\sigma\tau_1 D_8(D_2 D_5 - q\eta k\omega)}{\mathcal{O}_1},\\
T_H^{**} &= \frac{\lambda_H^{**}\alpha_H D_8(\omega\lambda_H^{**} + D_7)\,(\tau_1 D_2 D_4 + \tau_2(q\eta D_3 + \psi D_2))}{\mathcal{O}_1},\\
R_H^{**} &= \frac{\lambda_H^{**}\alpha_H\sigma D_8(\omega\lambda_H^{**} + D_7)\,(\tau_1 D_2 D_4 + \tau_2(q\eta D_3 + \psi D_2))}{\mathcal{O}_1},\\
V_H^{**} &= \frac{\lambda_H^{**}\alpha_H\sigma\xi(\omega\lambda_H^{**} + D_7)\,(\tau_1 D_2 D_4 + \tau_2(q\eta D_3 + \psi D_2))}{\mathcal{O}_1},\\
S_V^{**} &= \frac{\alpha_V D_9(\gamma_V - \phi\alpha_V)}{\mathcal{O}_2}, E_V^{**} = \frac{\lambda_V^{**}\alpha_V(\gamma_V - \phi\alpha_V)}{\mathcal{O}_2}, I_V^{**} = \frac{\lambda_V^{**}\alpha_V\mu}{\mathcal{O}_2}.
\end{aligned} \tag{18}$$

And

$D_1 = \eta + \gamma_H$, $D_2 = (1-q)\eta$, $D_3 = \psi + \tau_1 + \gamma_H$, $D_4 = \tau_2 + \delta + \gamma_H$, $D_5 = (1-k)\omega$, $D_6 = \sigma + \theta\delta + \gamma_H$, $D_7 = \xi + \gamma_H$, $D_8 = \varphi + \gamma_H$, $D_9 = \mu + \gamma_V$, $\mathcal{O}_1 = D_1 D_3 D_4 D_6 D_8(\omega\lambda_H^{**} + D_7)(\lambda_H^{**} + \gamma_H) - \lambda_H^{**}\sigma D_1 D_8(\lambda_H^{**} + \gamma_H)\mathscr{C}_1 - \lambda_H^{**}\xi\sigma\varphi\mathscr{C}_2$, and $\mathcal{O}_2 = D_9(\lambda_V^{**} + \gamma_V)(\gamma_V - \phi\alpha_V) + \lambda_V^{**}\phi\alpha_V\mu$.

With $\mathscr{C}_1 = \tau_1 k\omega D_4 + \tau_2(\psi k\omega + D_3 D_5)$, and $\mathscr{C}_2 = \tau_1 D_2 D_4 + \tau_2(q\eta D_3 + \psi D_2)$.

Substituting (18) and (17) into (16), we obtained

$$\mathscr{L}(\lambda_H^{**}) = \mathscr{B}_1\lambda_H^{**4} + \mathscr{B}_2\lambda_H^{**3} + \mathscr{B}_3\lambda_H^{**2} + \mathscr{B}_4\lambda_H^{**} + \mathscr{B}_5 = 0 \tag{19}$$

where

$$\begin{aligned}
\mathscr{B}_1 &= \zeta\beta_V\chi_1\chi_3\chi_9 + \chi_3^2\chi_{10},\\
\mathscr{B}_2 &= \chi_4(\zeta\beta_V\chi_1\chi_9 + \chi_3\chi_{10}) + \chi_3(\zeta\beta_V\chi_2\chi_9 + \chi_4\chi_{10}) - \zeta^2\beta_H\beta_V\alpha_V\mu\chi_1\chi_6,\\
\mathscr{B}_3 &= \chi_5(\zeta\beta_V\chi_1\chi_9 + \chi_3\chi_{10}) + \chi_4(\zeta\beta_V\chi_2\chi_9 + \chi_4\chi_{10}) + \chi_3\chi_5\chi_{10} - \zeta^2\beta_H\beta_V\alpha_V\mu(\chi_1\chi_7 + \chi_2\chi_6),\\
\mathscr{B}_4 &= \chi_5(\zeta\beta_V\chi_2\chi_9 + \chi_4\chi_{10}) + \chi_4\chi_5\chi_{10} - \zeta^2\beta_H\beta_V\alpha_V\mu(\chi_1\chi_8 + \chi_2\chi_7),\\
\mathscr{B}_5 &= \chi_5^2\chi_{10} - \zeta^2\beta_H\beta_V\alpha_V\mu\chi_2\chi_8.
\end{aligned} \tag{20}$$

With

$\chi_1 = \alpha_H D_8 \left(\omega D_6(D_2D_4 + q\eta D_3 + \psi D_2) + \sigma(D_2D_5 - q\eta k\omega)(\tau_1 - \tau_2)\right),$
$\chi_2 = \alpha_H D_6 D_7 D_8 (D_2D_4 + q\eta D_3 + \psi D_2),$
$\chi_3 = \alpha_H D_8 \left(\omega D_6(D_3D_4 + D_2D_4 + q\eta D_3 + \psi D_2) + \omega\mathscr{C}_2 + \sigma(D_2D_5 - q\eta k\omega)(\tau_1 - \tau_2) - \sigma\mathscr{C}_1\right),$
$\chi_4 = \alpha_H \left(D_8 \left(D_4D_6(\omega D_1D_3 + D_3D_7 + D_2D_7) + \sigma(\mathscr{C}_2 - D_1\mathscr{C}_1) + D_7(D_6(q\eta D_3 + \psi D_2) + \mathscr{C}_2)\right) + \xi\sigma\mathscr{C}_2\right),$
$\chi_5 = \alpha_H D_1D_3D_4D_6D_7D_8, \chi_6 = D_1D_8(\omega D_3D_4D_6 - \sigma\mathscr{C}_1),$
$\chi_7 = D_1D_3D_4D_6D_9(\omega\gamma_H + D_7) - \gamma_H\sigma D_1D_8\mathscr{C}_1 - \xi\sigma\varphi\mathscr{C}_2, \chi_8 = \gamma_H D_1D_3D_4D_6D_7D_8,$
$\chi_9 = D_9(\gamma_V - \phi\alpha_V) + \phi\alpha_V\mu, \chi_{10} = \gamma_V D_9(\gamma_V - \phi\alpha_V).$

Expressing $\mathscr{B}_5$ in (20) in terms of the basic reproduction number, we have

$$\begin{aligned}\mathscr{B}_5 =& \alpha^2\gamma_V D_1^2D_3^2D_4^2D_6^2D_7^2D_8^2D_9(\gamma_V \\ &- \phi\alpha_V) - \zeta^2\beta_H\beta_V\alpha_V\alpha_H\gamma_H\mu D_1D_3D_4D_6^2D_7^2D_8^2(D_2D_4 + q\eta D_3 + \psi D_2)\end{aligned} \tag{21}$$

So that

$$\mathscr{B}_5 = \alpha^2\gamma_V^2 D_1^2D_3^2D_4^2D_6^2D_7^2D_8^2D_9\left(1 - \left(\frac{\phi\alpha_V}{\gamma_V} + \frac{\zeta^2\beta_H\beta_V\alpha_V\gamma_H\mu(q\eta D_3 + \psi D_2 + D_2D_4)}{\alpha_H\gamma_V^2 D_1D_3D_4D_9}\right)\right) \tag{22}$$

Thus, we have that

$$\mathscr{B}_5 = \alpha^2\gamma_V^2 D_1^2D_3^2D_4^2D_6^2D_7^2D_8^2D_9\left(1 - \mathcal{R}_0^D\right) \tag{23}$$

From Eq. (19), it is clear that $\mathscr{B}_1 > 0$ because all the model parameters are non-negative. Furthermore, we have $\mathscr{B} > 0$ for $\mathcal{R}_0^D < 1$. Hence the number of all possible nonnegative real roots of the polynomial in Eq. (19) depends on the signs of $\mathscr{B}_2$, $\mathscr{B}_3$, and $\mathscr{B}_4$. This can be assessed by applying Descartes' Rule of Signs to the quartic equation:

$$f(x) = \mathscr{B}_1x^4 + \mathscr{B}_2x^3 + \mathscr{B}_3x^2 + \mathscr{B}_4x + \mathscr{B}_5 = 0,$$

where $x = \lambda_H^{**}$. By analyzing the number of sign changes in the polynomial, we can determine the possible number of positive real roots. Based on this analysis, the following results are derived.

Theorem 3 *The Dengue model given in (1)*

1. *Has a unique endemic equilibrium if* $\mathcal{R}_0^D > 1$ *and either of the following holds*
   (a) $\mathscr{B}_2, \mathscr{B}_3, \mathscr{B}_4 > 0$;
   (b) $\mathscr{B}_2, \mathscr{B}_3, \mathscr{B}_4 < 0$;
   (c) $\mathscr{B}_2 > 0$, while $\mathscr{B}_3, \mathscr{B}_4 < 0$;
   (d) $\mathscr{B}_2, \mathscr{B}_3 > 0$, while $\mathscr{B}_4 < 0$;
2. *Could exhibits one or more endemic equilibrium if* $\mathcal{R}_0^D > 1$ *and either of the following*
   (a) $\mathscr{B}_2, \mathscr{B}_4 < 0$, while $\mathscr{B}_3 > 0$;
   (b) $\mathscr{B}_2, \mathscr{B}_3 < 0$, while $\mathscr{B}_4 > 0$;
   (c) $\mathscr{B}_2, \mathscr{B}_4 > 0$, while $\mathscr{B}_3 < 0$;
   (d) $\mathscr{B}_2 < 0$, while $\mathscr{B}_3, \mathscr{B}_4 > 0$;

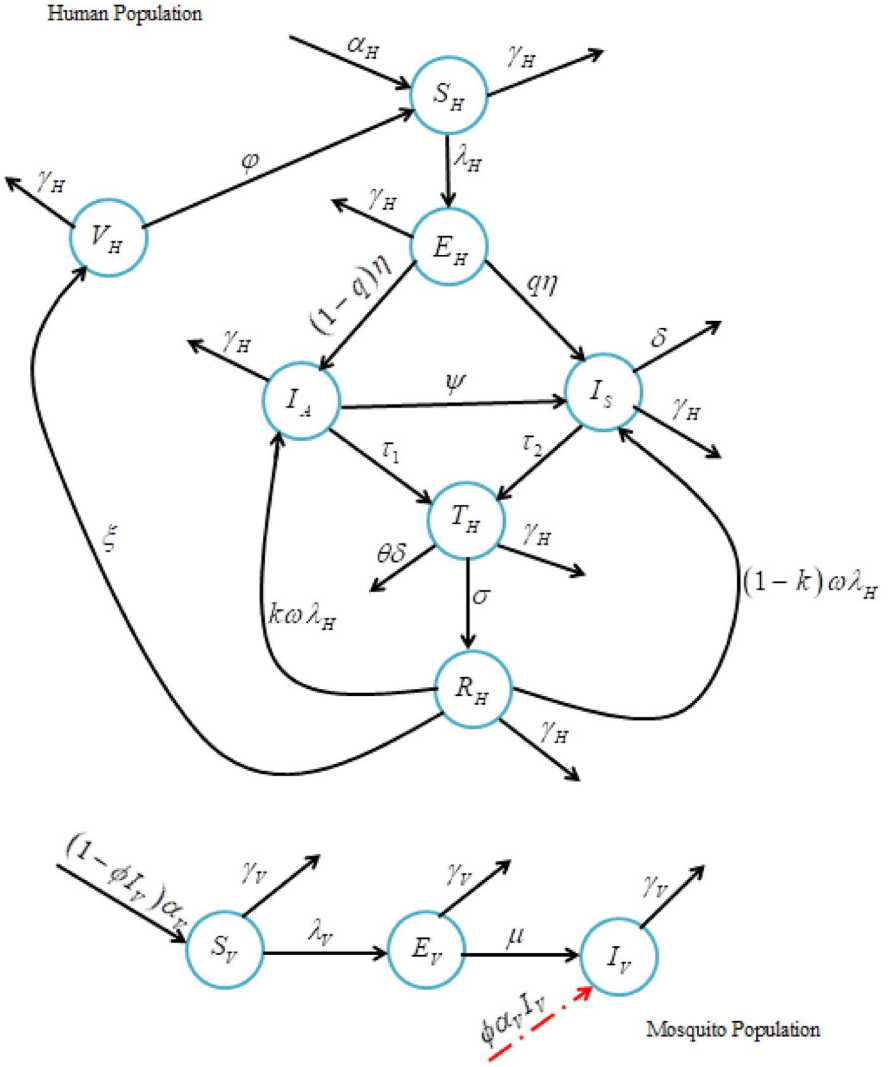

Fig. 1 Schematic representation of the Dengue Fever model

3. *Could have two or more endemic equilibria if* $\mathcal{R}_0^D < 1$ *and any, or all,* $\mathscr{B}_2$, $\mathscr{B}_3$, *and* $\mathscr{B}_4$ *are negative.*

Item 3 of Theorem 3 indicates the possibility of multiple endemic equilibria when $\mathcal{R}_0^D < 1$, which typically suggests the presence of a backward bifurcation Gumel (2012); Omede et al. (2023, 2024). A backward bifurcation is a phenomenon in which both a stable disease-free equilibrium (DFE) and a stable endemic equilibrium can coexist, even when the basic reproduction number $(\mathcal{R}_0^D)$ is less than one. This phenomenon has been observed in various disease transmission models, including those for vector-borne diseases, models incorporating exogenous re-infection, imperfect vaccination, and multi-group interactions. This implies that, even though the disease might not initially be able to spread in the popula-

tion (as indicated by $\mathcal{R}_0^D < 1$), an endemic state can still be sustained under certain conditions. In other words, there may be a threshold for the initial number of infected individuals below which the disease will die out, and above which it can establish an endemic equilibrium, despite the reproduction number being below 1. The backward bifurcation phenomenon has important implications for disease control and intervention strategies. In models of vector-borne diseases like Dengue, the presence of backward bifurcation can affect how interventions (such as vaccination, vector control, or treatment) influence disease dynamics. The occurrence of a backward bifurcation in the Dengue model will be examined in greater detail in the following section.

3.5 Possibility of the existence of backward bifurcation

In this section, we will explore the potential existence of a backward bifurcation by applying the Center Manifold Theorem, a concept extensively studied by Castillo-Chavez and Song in their work Castillo-Chavez and Song (2004). This mathematical tool allows us to analyze the stability of equilibria and the bifurcation structure of nonlinear dynamical systems, particularly in cases where multiple stable equilibria may exist, even when the obtained basic reproduction number is less than one. The application of this theorem in the context of the Dengue model will help us to identify the conditions under which a backward bifurcation may occur and provide deeper understanding of the complex dynamics of disease transmission (Fig. 1).

Theorem 4 *The Dengue model of system (1) exhibits backward bifurcation at* $\mathcal{R}_0^D = 1$.

Proof To apply the Center Manifold Theorem, we will make specific modifications to the state variables of the Dengue model (1). We define the variables as follows: $S_H = x_1, E_H = x_2, I_A = x_3, I_S = x_4, T_H = x_5, R_H = x_6, V_H = x_7$, and $S_V = x_8, E_V = x_9, I_V = x_{10}$. Using vector notation, we express the state as $x = (x_1, x_2, x_3, \ldots, x_{10})^T$ and the system of equations as $\frac{dx}{dt} = F(x)$, where $F = (f_1, f_2, f_3, \ldots, f_{10})^T$. Hence the Dengue model (1) becomes

$$\begin{aligned}
\frac{dx_1}{dt} &\equiv f_1 = \alpha_H - \lambda_H x_1 - \gamma_H x_1 + \varphi x_7,\\
\frac{dx_2}{dt} &\equiv f_2 = \lambda_H x_1 - (\eta + \gamma_H) x_2,\\
\frac{dx_3}{dt} &\equiv f_3 = (1-q)\eta x_2 - (\psi + \tau_1 + \gamma_H) x_3 + k\omega\lambda_H x_6,\\
\frac{dx_4}{dt} &\equiv f_4 = q\eta x_2 + \psi x_3 - (\tau_2 + \delta + \gamma_H) x_4 + (1-k)\omega\lambda_H x_6,\\
\frac{dx_5}{dt} &\equiv f_5 = \tau_1 x_3 + \tau_2 x_4 - (\sigma + \theta\delta + \gamma_H) x_5,
\end{aligned}$$

$$\begin{aligned}
\frac{dx_6}{dt} &\equiv f_6 = \sigma x_5 - (\xi + \omega\lambda_H + \gamma_H)x_6,\\
\frac{dx_7}{dt} &\equiv f_7 = \xi x_6 - (\varphi + \gamma_H)x_7,\\
\frac{dx_8}{dt} &\equiv f_8 = (1 - \phi x_{10})\alpha_V - \lambda_V x_8 - \gamma_V x_8,\\
\frac{dx_9}{dt} &\equiv f_9 = \lambda_V x_8 - (\mu + \gamma_V)x_9,\\
\frac{dx_{10}}{dt} &\equiv f_{10} = \mu x_9 + \phi\alpha_V x_{10} - \gamma_V x_{10}.
\end{aligned} \tag{24}$$

where

$$\lambda_H = \frac{\zeta\beta_H x_{10}}{x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7}, and \quad \lambda_V = \frac{\zeta\beta_V(x_3 + x_4)}{x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7}.$$

We considered the mosquito biting rate along with the transmission probability from an infected mosquito to susceptible individuals (with $\zeta\beta_H = m^*$) as the bifurcation parameter. By solving for m^* at $\mathcal{R}_0^D = 1$, we have

$$m^* = \frac{\alpha_H\gamma_V(\eta + \gamma_H)(\psi + \tau_1 + \gamma_H)(\tau_2 + \delta + \gamma_H)(\mu + \gamma_V)(\gamma_V - \phi\alpha_V)}{\zeta\beta_V\alpha_V\gamma_H\mu(q\eta(\psi + \tau_1 + \gamma_H) + (1 - q)\eta(\psi + \tau_2 + \delta + \gamma_H)} \tag{25}$$

Calculating the Jacobian of the transformed system (24) at the Dengue-free Equilibrium (ℓ_0) with $\zeta\beta_H = m^*$, results in:

$$\mathcal{J}(\ell_0)|_{\zeta\beta_H = m^*} = \begin{bmatrix}
-\gamma_H & 0 & 0 & 0 & 0 & 0 & \varphi & 0 & 0 & -m^*\\
0 & -D_1 & 0 & 0 & 0 & 0 & \varphi & 0 & 0 & m^*\\
0 & D_2 & -D_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & q\eta & \psi & -D_4 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & \tau_1 & \tau_2 & -D_6 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & \sigma & -D_7 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & \xi & -D_8 & 0 & 0 & 0\\
0 & 0 & -\hbar & -\hbar & 0 & 0 & 0 & -\gamma_V & 0 & -\phi\alpha_V\\
0 & 0 & \hbar & \hbar & 0 & 0 & 0 & 0 & -D_9 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mu & \phi\alpha_V - \gamma_V
\end{bmatrix}$$

where

$D_1 = \eta + \gamma_H, D_2 = (1 - q)\eta, D_3 = \psi + \tau_1 + \gamma_H, D_4 = \tau_2 + \delta + \gamma_H, D_5 = (1 - k)\omega,$

$D_6 = \sigma + \theta\delta + \gamma_H$, $D_7 = \xi + \gamma_H$, $D_8 = \varphi + \gamma_H$, $D_9 = \mu + \gamma_V$, and $\hbar = \frac{\zeta\beta_V\alpha_V\gamma_H}{\alpha_H\gamma_V}$.

The right eigenvectors represented as $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9, w_{10})^T$, which is corresponding to the simple zero eigenvalue, can be obtained from $\mathcal{J}(\ell_0)|_{\zeta\beta_H = m^*} w = 0$, expressed as:

$$
\begin{aligned}
&-\gamma_H w_1+\varphi w_7-m^* w_{10}=0,\\
&-D_1 w_2+m^* w_{10},\\
&D_2 w_2-D_3 w_3=0,\\
&q\eta w_2+\psi w_3-D_4 w_4=0,\\
&\tau_1 w_3+\tau_2 w_4-D_6 w_5=0,\\
&\sigma w_5-D_7 w_6=0,\\
&\xi w_6-D_8 w_7=0,\\
&-\frac{\zeta\beta_V\alpha_V\gamma_H w_3}{\alpha_H\gamma_V}-\frac{\zeta\beta_V\alpha_V\gamma_H w_4}{\alpha_H\gamma_V}-\gamma_V w_8-\phi\alpha_V w_{10}=0,\\
&\frac{\zeta\beta_V\alpha_V\gamma_H w_3}{\alpha_H\gamma_V}+\frac{\zeta\beta_V\alpha_V\gamma_H w_4}{\alpha_H\gamma_V}-D_9 w_9=0,\\
&\mu w_9+(\phi\alpha_V-\gamma_V)w_{10}=0.
\end{aligned}
\tag{26}
$$

Solving (26) above, we obtained

$$
\begin{aligned}
&w_1=\frac{(\varphi\xi\sigma(\tau_1 D_2D_4+\tau_2(q\eta D_3+\psi D_2))-D_1D_3D_4D_6D_7D_8)\,w_2}{\gamma_H D_3D_4D_6D_7D_8}, w_2=w_2>0, w_3=\frac{D_2w_2}{D_3},\\
&w_4=\frac{(q\eta D_3+\psi D_2)w_2}{D_3D_4}, w_5=\frac{(\tau_1D_2D_4+\tau_2(q\eta D_3+\psi D_2))w_2}{D_3D_4D_6},\\
&w_6=\frac{\sigma(\tau_1D_2D_4+\tau_2(q\eta D_3+\psi D_2))w_2}{D_3D_4D_6D_7},\\
&w_7=\frac{\xi\sigma(\tau_1D_2D_4+\tau_2(q\eta D_3+\psi D_2))w_2}{D_3D_4D_6D_7D_8}, w_8=-\frac{D_1\left((\gamma_V-\phi\alpha_V)D_9+\phi\alpha_V\mu\right)w_2}{m^*\mu\gamma_V},\\
&w_9=\frac{(\gamma_V-\phi\alpha_V)D_1w_2}{m^*\mu}, and\quad w_{10}=\frac{D_1w_2}{m^*}.
\end{aligned}
\tag{27}
$$

Likewise, the left eigenvector represented by $v=(v_1,v_2,v_3,v_4,v_5,v_6,v_7,v_8,v_9,v_{10})$ satisfying $v.w=1$ and associated with the simple zero eigenvalue, can be determined from the expression $v\mathcal{J}\left(\ell_0\right)|_{\zeta\beta_H=m^*}=0$, given by

$$
\begin{aligned}
&-\gamma_H v_1=0,\\
&-D_1v_2+D_2v_3+q\eta v_4=0,\\
&-D_3v_3+\psi v_4+\tau_1v_5-\frac{\zeta\beta_V\alpha_V\gamma_H v_8}{\alpha_H\gamma_V}+\frac{\zeta\beta_V\alpha_V\gamma_H v_9}{\alpha_H\gamma_V}=0,\\
&-D_4v_4+\tau_2v_5-\frac{\zeta\beta_V\alpha_V\gamma_H v_8}{\alpha_H\gamma_V}+\frac{\zeta\beta_V\alpha_V\gamma_H v_9}{\alpha_H\gamma_V}=0,\\
&-D_6v_5+\sigma v_6=0,\\
&-D_7v_6+\xi v_7=0,\\
&\psi v_1-D_8v_7=0,\\
&-\gamma_V v_8=0,\\
&-D_9v_9+\mu v_{10}=0,\\
&-m^*v_1+m^*v_2-\phi\alpha_V v_8+(\phi\alpha_V-\gamma_V)v_{10}=0.
\end{aligned}
\tag{28}
$$

Solving (28) above, we obtained

$$
\begin{aligned}
&v_1 = v_5 = v_6 = v_7 = v_8 = 0, v_2 = \frac{((\psi + D_4)D_2 + q\eta D_3)\, v_3}{(\psi + D_4)D_1}, v_3 = v_3 > 0, v_4 = \frac{D_3 v_3}{(\psi + D_4)},\\
&v_9 = \frac{m^*\mu\,((\psi + D_4)D_2 + q\eta D_3)\, v_3}{(\gamma_V - \phi\alpha_V)(\psi + D_4)D_1 D_9}, v_{10} = \frac{m^*\,((\psi + D_4)D_2 + q\eta D_3)\, v_3}{(\gamma_V - \phi\alpha_V)(\psi + D_4)D_1}.
\end{aligned} \tag{29}
$$

Thus, the partial derivatives of $f_1, f_2, f_3, \ldots, f_{10}$ at the Dengue-free equilibrium are given by

$$
\begin{aligned}
&\frac{\partial^2 f_2}{\partial x_2 \partial x_{10}} = \frac{\partial^2 f_2}{\partial x_{10} \partial x_2} = \frac{\partial^2 f_2}{\partial x_3 \partial x_{10}} = \frac{\partial^2 f_2}{\partial x_{10} \partial x_3} = \frac{\partial^2 f_2}{\partial x_4 \partial x_{10}} = \frac{\partial^2 f_2}{\partial x_{10} \partial x_4}\\
&= \frac{\partial^2 f_2}{\partial x_5 \partial x_{10}} = \frac{\partial^2 f_2}{\partial x_{10} \partial x_5} = -\frac{m^*\gamma_H}{\alpha_H},\\
&\frac{\partial^2 f_2}{\partial x_6 \partial x_{10}} = \frac{\partial^2 f_2}{\partial x_{10} \partial x_6} = \frac{\partial^2 f_2}{\partial x_7 \partial x_{10}} = \frac{\partial^2 f_2}{\partial x_{10} \partial x_7} = -\frac{m^*\gamma_H}{\alpha_H},\\
&\frac{\partial^2 f_9}{\partial x_1 \partial x_3} = \frac{\partial^2 f_9}{\partial x_3 \partial x_1} = \frac{\partial^2 f_9}{\partial x_2 \partial x_3} = \frac{\partial^2 f_9}{\partial x_3 \partial x_2} = \frac{\partial^2 f_9}{\partial x_3 \partial x_5} = \frac{\partial^2 f_9}{\partial x_5 \partial x_3} = \frac{\partial^2 f_9}{\partial x_3 \partial x_6} = \frac{\partial^2 f_9}{\partial x_6 \partial x_3} = -\frac{\zeta\beta_V\alpha_V\gamma_H^2}{\alpha_H^2\gamma_V},\\
&\frac{\partial^2 f_9}{\partial x_3 \partial x_7} = \frac{\partial^2 f_9}{\partial x_7 \partial x_3} = -\frac{\zeta\beta_V\alpha_V\gamma_H^2}{\alpha_H^2\gamma_V}, \frac{\partial^2 f_9}{\partial x_3^2} = \frac{\partial^2 f_9}{\partial x_4^2} = \frac{\partial^2 f_9}{\partial x_3 \partial x_4} = \frac{\partial^2 f_9}{\partial x_4 \partial x_3} = -\frac{2\zeta\beta_V\alpha_V\gamma_H^2}{\alpha_H^2\gamma_V},\\
&\frac{\partial^2 f_9}{\partial x_1 \partial x_4} = \frac{\partial^2 f_9}{\partial x_4 \partial x_1} = \frac{\partial^2 f_9}{\partial x_2 \partial x_4} = \frac{\partial^2 f_9}{\partial x_4 \partial x_2} = \frac{\partial^2 f_9}{\partial x_4 \partial x_5} = \frac{\partial^2 f_9}{\partial x_5 \partial x_4} = \frac{\partial^2 f_9}{\partial x_4 \partial x_6} = \frac{\partial^2 f_9}{\partial x_6 \partial x_4} = -\frac{\zeta\beta_V\alpha_V\gamma_H^2}{\alpha_H^2\gamma_V},\\
&\frac{\partial^2 f_9}{\partial x_4 \partial x_7} = \frac{\partial^2 f_9}{\partial x_7 \partial x_4} = -\frac{\zeta\beta_V\alpha_V\gamma_H^2}{\alpha_H^2\gamma_V}, \frac{\partial^2 f_2}{\partial x_{10} \partial m^*} = 1
\end{aligned} \tag{30}
$$

Hence, the direction of the bifurcation at $\mathcal{R}_0^D = 1$ is obtained by the sign of the bifurcation coefficients *a* and *b*. Then, these coefficients can be obtained by using the partial derivatives provided in (30). Specifically, the bifurcation coefficients *a* and *b* are given as

$$
a = \sum_{1,j,k=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\ell_0) \tag{31}
$$

$$
\begin{aligned}
a = &-\frac{2v_3 w_2^2 \gamma_H D_1 \left(D_6 D_7 D_8 (D_2 D_4 + q\eta D_3 + \psi D_2) + (\tau_1 D_2 D_4 + \tau_2 (q\eta D_3 + \psi D_2))(D_8(D_7 + \sigma) + \xi\sigma)\right)}{\alpha_H D_3 D_4 D_6 D_7 D_8}\\
&- \frac{2v_3 w_2^2 m^* \zeta\beta_V\alpha_V\gamma_H\mu(D_2(\psi + D_4) + q\eta D_3)^2\left(\varphi\xi\sigma(\tau_1 D_2 D_4 + \tau_2(q\eta D_3 + \psi D_2)) - D_1 D_3 D_4 D_6 D_7 D_8\right)}{\alpha_H^2\gamma_V D_1 D_3^2 D_4^2 D_6 D_7 D_8 D_9(\gamma_V - \phi\alpha_V)(\psi + D_4)}\\
&- \frac{2v_3 w_2^2 m^* \zeta\beta_V\alpha_V\gamma_H^2\mu(D_2(\psi + D_4) + q\eta D_3)^2\left(\tau_1 D_2 D_4 + \tau_2(q\eta D_3 + \psi D_2)\right)(D_7 D_8 + \sigma D_8 + \xi\sigma)}{\alpha_H^2\gamma_V D_1 D_3^2 D_4^2 D_6 D_7 D_8 D_9(\gamma_V - \phi\alpha_V)(\psi + D_4)}\\
&- \frac{2v_3 w_2^2 m^* \zeta\beta_V\alpha_V\gamma_H^2\mu(D_2(\psi + D_4) + q\eta D_3)(D_2^2 D_4^2 + (q\eta D_3 + \psi D_2)^2)}{\alpha_H^2\gamma_V D_1 D_3^2 D_4^2 D_9(\gamma_V - \phi\alpha_V)(\psi + D_4)}\\
&- \frac{4v_3 w_2^2 m^* \zeta\beta_V\alpha_V\gamma_H^2\mu D_2(D_2(\psi + D_4) + q\eta D_3)(q\eta D_3 + \psi D_2)}{\alpha_H^2\gamma_V D_1 D_3^2 D_4 D_9(\gamma_V - \phi\alpha_V)(\psi + D_4)}\\
&- \frac{2v_3 w_2^2 m^* \zeta\beta_V\alpha_V\gamma_H^2\mu(D_2(\psi + D_4) + q\eta D_3)^2}{\alpha_H^2\gamma_V D_1 D_3 D_4 D_9(\gamma_V - \phi\alpha_V)(\psi + D_4)}.
\end{aligned} \tag{32}
$$

$$
b = \sum_{1,k=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial m^*}(\ell_0) \tag{33}
$$

$$
b = \frac{v_3 w_2 (D_2(\psi + D_4) + q\eta D_3)}{m^*(\psi + D_4)} > 0 \tag{34}
$$

Since the bifurcation coefficient b is positive, it follows from theorem 4.1 in Castillo-Chavez and Song (2004) that the Dengue model (1) undergoes backward bifurcation at $\mathcal{R}_0^D = 1$ whenever $a > 0$.

The Fig. 2 shows the bifurcation representation of the Dengue model with the parameters $\beta_H = 0.2$, $\beta_V = 0.24$, $\zeta = 0.42$, and $\phi = 0.03$, whil other parameters are presented in Table 3. The bifurcation (backward) shows a locally stable Dengue-free equilibrium point and a stable endemic equilibrium point when $\mathcal{R}_0^D < 1$, it illustrates the possible number of positive roots of the polynomial (19) □

3.6 Data-fitting and parameter estimation

Biologically realistic ranges identified from the literature in the context of dengue modeling research Naaly et al. (2024); Damtew et al. (2023); Aldila et al. (2023); Kumar et al. (2024); Pongsumpun et al. (2023); Omame et al. (2023) were considered in the choice of parameter values for mosquito biting rate, mortality, and transmission probabilities. Therefore, the model seeks to capture realistic Aedes aegypti and dengue virus transmission dynamics in tropical environments.

To quantify how well the model captures the observed dengue case data, we computed three statistical goodness-of-fit measures for the model: the coefficient of determination (R^2), root mean square error (RMSE), and mean absolute percentage error (MAPE). These metrics assess the predictive performance of the model, where R^2 measures the proportion of variation explained by the model, while RMSE and MAPE are estimated as the deviation between the observed and predicted values. The calculated values ($R^2 = 0.982$, RMSE = 1.34×10^5, and MAPE = 3.8%) are indicative of an excellent fit of the model to

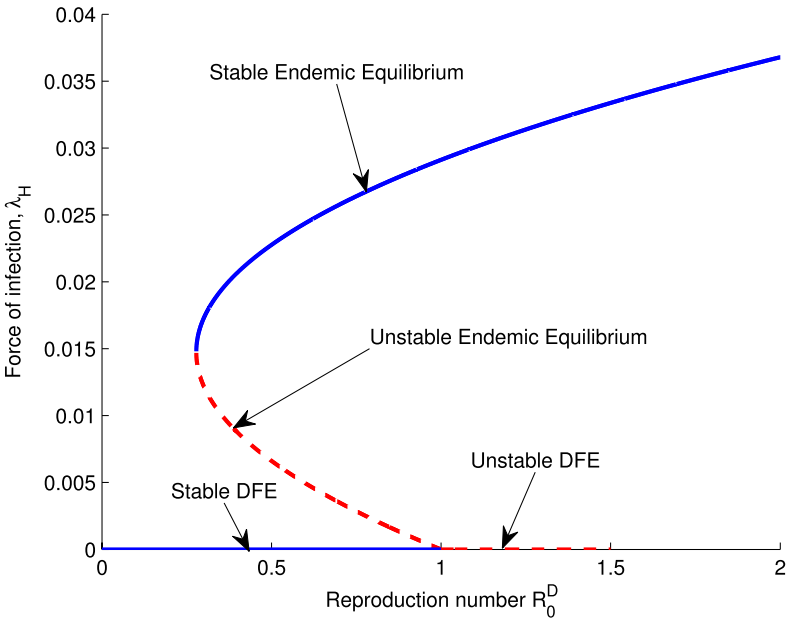

Fig. 2 The Backward bifurcation representation of the Dengue model, with $\beta_H = 0.2$, $\beta_V = 0.24$, $\zeta = 0.42$, and $\phi = 0.03$

the observed data on dengue cases. Data fitting and adequate estimation of parameters are essential approaches in mathematical modeling, as they help to bridge the gap between the theoretical formulation and real-world observation, which will thereby ensure that subsequent predictions from the model are not just accurate, but also meaningful. Hence, in this section, we perform a data fitting into the Dengue model (1) using the total reported cases of Dengue in Brazil from week 1 to week 30 of 2024. The data on total Dengue cases were obtained from the Pan American Health Organization (xxxx), as shown in Table 2. The process of fitting the model to the data was conducted using the MATLAB fmincon function from the Optimization Toolbox McCall (2005). This method relies on the least squares approach, which is a well-established and trusted technique for fitting models. The primary objective of the data fitting is to match the observed data, denoted as Y_i, with the predicted values from the model, represented as X_i. This is achieved by minimizing the sum of squared errors (SSE), which is defined as:

$$SSE = \sum_{i=1}^{k} (Y_i - X_i)^2, \tag{35}$$

Table 2 Total dengue cases in Brazil, 2024

Week	Case
1	83181
2	184921
3	327593
4	522802
5	785364
6	1101851
7	1492843
8	1979949
9	2512962
10	3065290
11	3649621
12	4265172
13	4794616
14	5395130
15	5990179
16	6542048
17	7030920
18	7498641
19	7963140
20	8376002
21	8678872
22	8873741
23	9028647
24	9171538
25	9289698
26	9374637
27	9438348
28	9484715
29	9525266
30	9561238

where Y_i represents the total observed cases and X_i are the estimated values from the model, and k is the total number of data points used in the fitting process. The goal is to find the parameters that best minimize the SSE, thereby providing the best fit of the model to the observed data. We estimated the population of Brazil to be 212.6 million (Instituto Brasileiro de Geografia e Estatística xxxx), and the initial values of the state variables used for the data-fitting are $S_H(0)$ =210000000, $E_H(0)$ =1500000, $I_A(0)$ =100000, $I_S(0)$ = 40000, $T_H(0)$ =83181, $R_H(0) = 0$, V_H =70000, $S_V(0)$ =168000, $E_V(0)$ =100000, and $I_V(0)$ =60000. The fitting results are illustrated in Fig. 3.

3.7 Sensitivity analysis

The sensitivity analysis is a critical approach that helps to determine how small changes in model parameters affect the outcomes of a mathematical model. We note that since the model parameters are often estimated with some level of uncertainty, then this analysis allows us to identify the parameters that have the most influence on model predictions. Therefore, in this section, we conduct a sensitivity analysis for the key parameters that contribute to the calculation of the basic reproduction number, $\mathcal{R}_0^D$, in the Dengue model (1). The goal is to evaluate how each parameter influences the Dengue virus (DENV) transmission dynamics and to identify the parameters that have the most significant effect on the disease spread.

To achieve this, we adopt the methodology outlined in Oguntolu et al. (2024); Omede et al. (2023), which provides a systematic approach for performing sensitivity analysis. Following these principles, we utilize the normalized forward sensitivity index for a variable Y, which measures the differential dependence of Y on a parameter x. This sensitivity index is defined as:

$$^{Y}\mathcal{W}_x = \frac{\partial Y}{\partial x} \times \frac{x}{Y}. \tag{36}$$

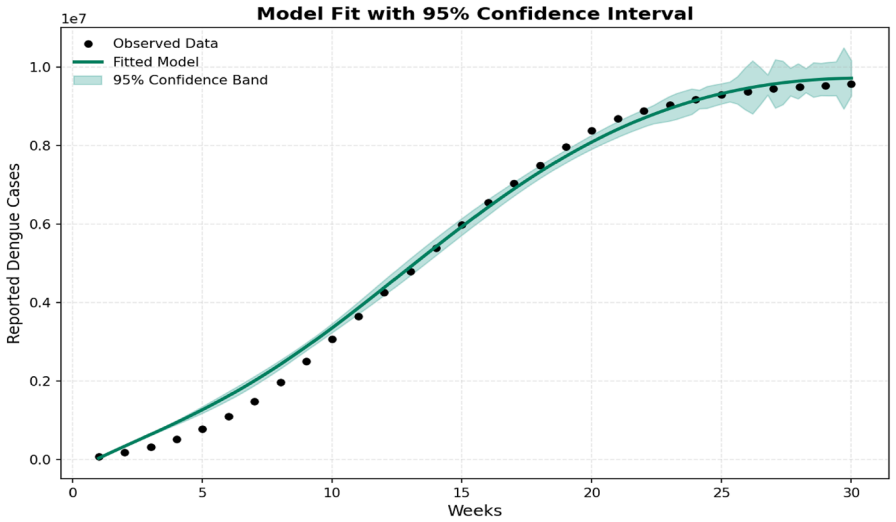

Fig. 3 Total cases of dengue fever in Brazil from week 1 to week 30, 2024

In (36), $\frac{\partial Y}{\partial x}$ represents the partial derivative of Y w.r.t x, indicating how Y changes when x is altered. The term $\frac{x}{Y}$ is the normalization factor which scales the sensitivity index, ensuring that it is dimensionless and enabling comparisons across different parameters. This method provides a quantitative way to assess how sensitive the model's output (in this case, Y) is to changes in individual parameters. By performing this analysis, we can determine the parameters that most significantly influence the transmission dynamics of Dengue. In our specific case for the Dengue model (1), we compute the sensitivity index $\mathcal{R}_0^D$, w.r.t parameter x. This is expressed as:

$$\mathcal{R}_0^D \mathcal{W}_x = \frac{\partial \mathcal{R}_0^D}{x} \times \frac{x}{\mathcal{R}_0^D} \tag{37}$$

Hence, we computed the sensitive indices of the basic reproduction number with respect to the parameters as follows;

$$\mathcal{R}_0^D \mathcal{W}_\phi = \frac{\partial \mathcal{R}_0^D}{\phi} \times \frac{\phi}{\mathcal{R}_0^D} \tag{38}$$

$$\mathcal{R}_0^D \mathcal{W}_\phi = \frac{\alpha_V}{\gamma_V} \times \frac{\phi\alpha_H\gamma_V^2(\eta+\gamma_H)(\psi+\tau_1+\gamma_H)(\tau_2+\delta+\gamma_H)(\mu+\gamma_V)}{\phi\alpha_H\alpha_V\gamma_V(\eta+\gamma_H)(\psi+\tau_1+\gamma_H)(\tau_2+\delta+\gamma_H)(\mu+\gamma_V)+\mathcal{Q}} \tag{39}$$

where $\mathcal{Q} = \zeta^2\beta_H\beta_V\alpha_V\gamma_H\mu\,(q\eta(\psi+\tau_1+\gamma_H)+(1-q)\eta(\psi+\tau_2+\delta+\gamma_H))$.

We have that

$$\mathcal{R}_0^D \mathcal{W}_\phi = \frac{\phi\alpha_H\alpha_V\gamma_V^2(\eta+\gamma_H)(\psi+\tau_1+\gamma_H)(\tau_2+\delta+\gamma_H)(\mu+\gamma_V)}{\phi\alpha_H\alpha_V\gamma_V^2(\eta+\gamma_H)(\psi+\tau_1+\gamma_H)(\tau_2+\delta+\gamma_H)(\mu+\gamma_V)+\gamma_V\mathcal{Q}} \tag{40}$$

Thus

$$\mathcal{R}_0^D \mathcal{W}_\phi = 0.004964 \tag{41}$$

Similarly, we compute the sensitive indices for the remaining basic parameter, given by

$$\begin{aligned}
&\mathcal{R}_0^D \mathcal{W}_{\alpha_V} = 1, \mathcal{R}_0^D \mathcal{W}_{\alpha_H} = -0.9950, \mathcal{R}_0^D \mathcal{W}_{\gamma_H} = 0.7427, \mathcal{R}_0^D \mathcal{W}_\zeta = 1.9901,\\
&\mathcal{R}_0^D \mathcal{W}_{\beta_H} = 0.9955, \mathcal{R}_0^D \mathcal{W}_{\beta_V} = 0.9950, \mathcal{R}_0^D \mathcal{W}_\eta = 0.0088, \mathcal{R}_0^D \mathcal{W}_\mu = 0.07421,\\
&\mathcal{R}_0^D \mathcal{W}_q = -0.0232, \mathcal{R}_0^D \mathcal{W}_\psi = -0.0673, \mathcal{R}_0^D \mathcal{W}_{\tau_1} = -0.000351, \mathcal{R}_0^D \mathcal{W}_{\tau_2} = -0.6254,\\
&\mathcal{R}_0^D \mathcal{W}_\delta = -0.0585, \mathcal{R}_0^D \mathcal{W}_{\gamma_V} = -2.0692.
\end{aligned} \tag{42}$$

3.7.1 Interpretation of the sensitivity indices

Here, we shall present the biological interpretation of the sensitivity indices using the computed parameters value from (40) to (42). These values are used to obtain the figure shown in 4. The bar chart in Fig. 4 displays the sensitivity indices of the basic reproduction number ($\mathcal{R}_0^D$) for the Dengue model. We note that the parameters that exhibit positive sensitivity

indices have a significant effect on accelerating the spread of Dengue. In particular, as the values of these parameters increases, while other parameters are held constant, the basic reproduction number also increases significantly. This indicates that an increase in these parameters contributes to an overall increase in Dengue transmission.

On the other hand, parameters with negative sensitivity indices play a crucial role in mitigating the disease burden. As these parameters increase, the basic reproduction number decreases which signifies a reduction in the spread of the Dengue virus (DENV). This suggests that these parameters help to alleviate the transmission dynamics and can be critical targets for control measures for mitigating the impact of Dengue.

3.8 Numerical simulation of the dengue model

The numerical simulation is a fundamental tool widely used in disease transmission dynamics which enables us to analyze the behavior of the system with varying parameter conditions. Moreover, performing numerical computations often helps to identify the system dynamics under various scenarios. Thus, in this section, we conduct a numerical simulation of dengue fever using MATLAB to illustrate a selection of the analytical results, based on the parameter values listed in Table 3. With these specified parameters, the basic reproduction number for the dengue virus in Brazil is determined to be $\mathcal{R}_0^D = 362.8989$ and the subsequent simulation results are shown in Fig. 5.

Figure 5 illustrates the simulation results for the effects of various parameters on the total number of Dengue cases. Specifically, the parameters analyzed include the mosquito death rate (γ_V), the introduction rate of new mosquitoes into the population (α_V), the treatment rate for asymptomatic infected individuals with Dengue (τ_1), and the treatment rate for symptomatic infected individuals with Dengue (τ_2).

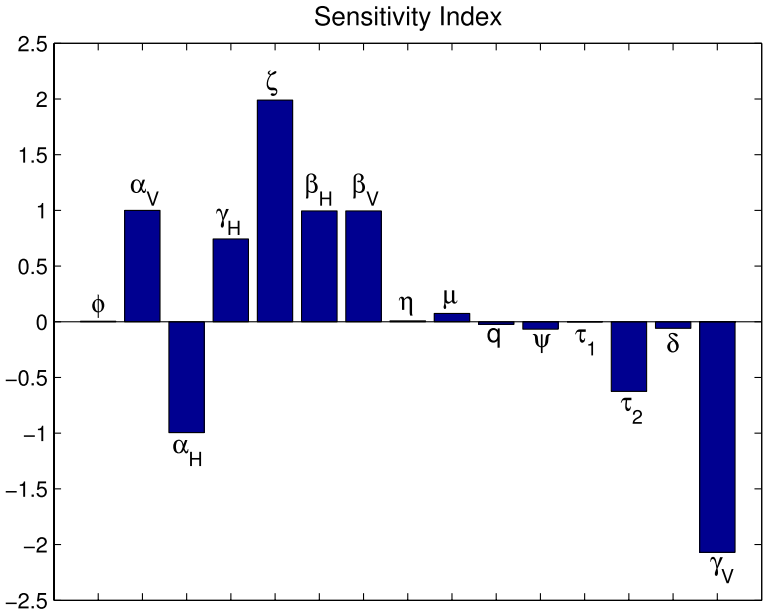

Fig. 4 The Dengue model basic reproduction number sensitivity index

Table 3 Parameter values for model (1)

Parameter	Values	Source
α_H	$\frac{13.2}{1000}$	Birth rate (xxxx)
α_V	0.18	Fitted
β_H	0.9775	Fitted
β_V	0.74	Fitted
ζ	0.9964	Fitted
γ_H	$\frac{7}{1000}$	Death rate (xxxx)
η	0.785	Fitted
q	0.25	Fitted
ψ	0.21	Fitted
τ_1	0.0001	Fitted
τ_2	0.02	Fitted
δ	$\frac{187}{100000}$	Estimated
θ	0.001	Fitted
σ	0.8330	Fitted
ξ	0.7	Fitted
φ	0.6	Fitted
ω	0.7	Fitted
k	0.3	Fitted
ϕ	0.806	Fitted
μ	0.9992	Fitted
γ_V	0.0805300	Fitted

In Fig. 5a, it is observed that an increase in the mosquito death rate (γ_V) significantly reduces the total number of Dengue cases. This suggests that higher mortality rates among mosquitoes can effectively limit the mosquito population, thereby reducing the transmission of Dengue. In Fig. 5b, it is shown that a decrease in the introduction rate of mosquitoes (α_V) into the population leads to a corresponding decline in the total number of Dengue cases. This finding is consistent with the results of the sensitivity analysis, highlighting the importance of controlling mosquito introduction as a strategy to reduce Dengue transmission.

However, in Fig. 5c and d, it is revealed that the treatment rates (τ_1 and τ_2) for asymptomatic and symptomatic infected individuals, respectively, have minimal impact on reducing the total cases of Dengue. This outcome may be due to the fact that Dengue transmission can still be sustained within the mosquito population even in the absence of human hosts, indicating that human treatment alone might not be sufficient to curb the overall transmission dynamics.

Figure 6a and b present contour plots of the basic reproduction number $\mathcal{R}_0^D$ as a function of the mosquito death rate (γ_V) and the treatment rate for asymptomatic infected individuals with Dengue (τ_1) in Fig. 6a, and the treatment rate for symptomatic infected individuals with Dengue (τ_2) in Fig. 6b. From these plots, it is observed that by increasing the mosquito death rate (γ_V) and the treatment rates (τ_1 and τ_2) to 90%, the basic reproduction number can be reduced to below one, indicating a potential reduction in the overall transmission of Dengue.

Figure 6c shows the contour plot of the basic reproduction number as a function of the introduction rate of new mosquitoes (α_V) and the mosquito biting rate (ζ). It is evident from this plot that an increase in either the introduction rate of mosquitoes or the mosquito biting rate significantly raises the basic reproduction number. This highlights the critical

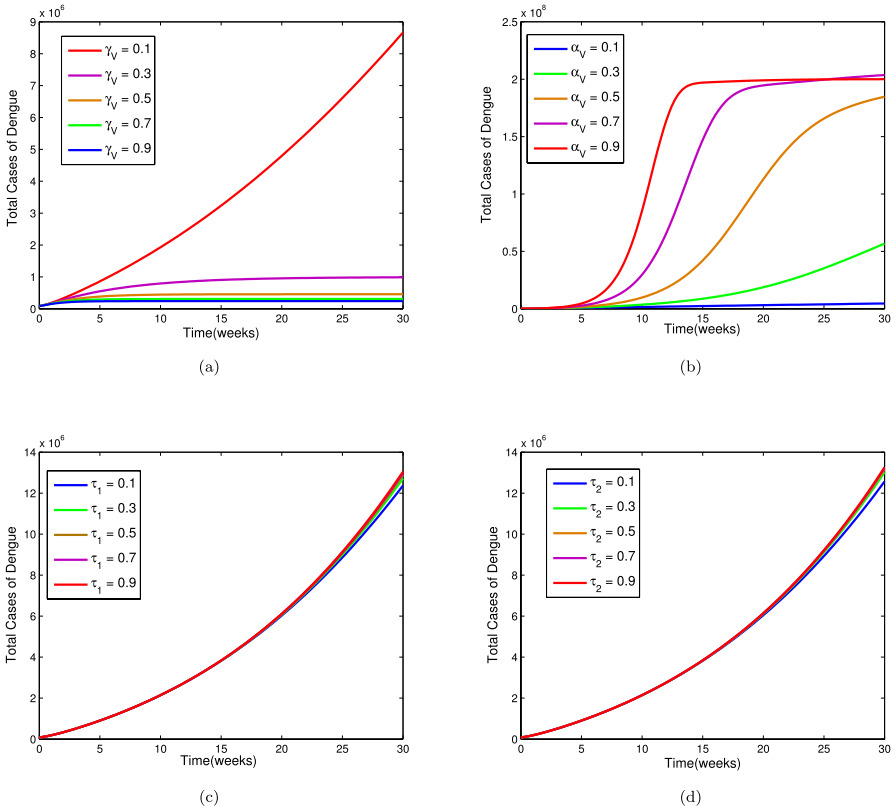

Fig. 5 Simulation of the **a** effect of γ_V on the total cases of Dengue, **b** effect of α_V on the total cases of Dengue, **c** effect of τ_1 on the total cases of Dengue, **d** effect of τ_2 on the total cases of Dengue

role of these factors in enhancing the transmission dynamics of Dengue and emphasizes the importance of controlling mosquito population dynamics and behavior to reduce the spread of the disease.

4 Extension of the dengue fever model with optimal control strategies

In the previous sections, we presented the dengue transmission model, where we discussed the interaction between humans and mosquitoes with a detailed explanation of the model parameters. Here, we extend the model to incorporate optimal control strategies with the sole objective of determining the best combination of interventions to minimize disease transmission. For our Dengue model (1), we incorporate two control measures aimed at mitigating and reducing the spread of dengue virus (DENV). The first control measure is the usage of mosquito bed-nets, which prevent susceptible individuals from acquiring DENV. This is represented by $u_1(t)$, where $u_1(t)$ is the control effort related to the usage of bed-nets over time.

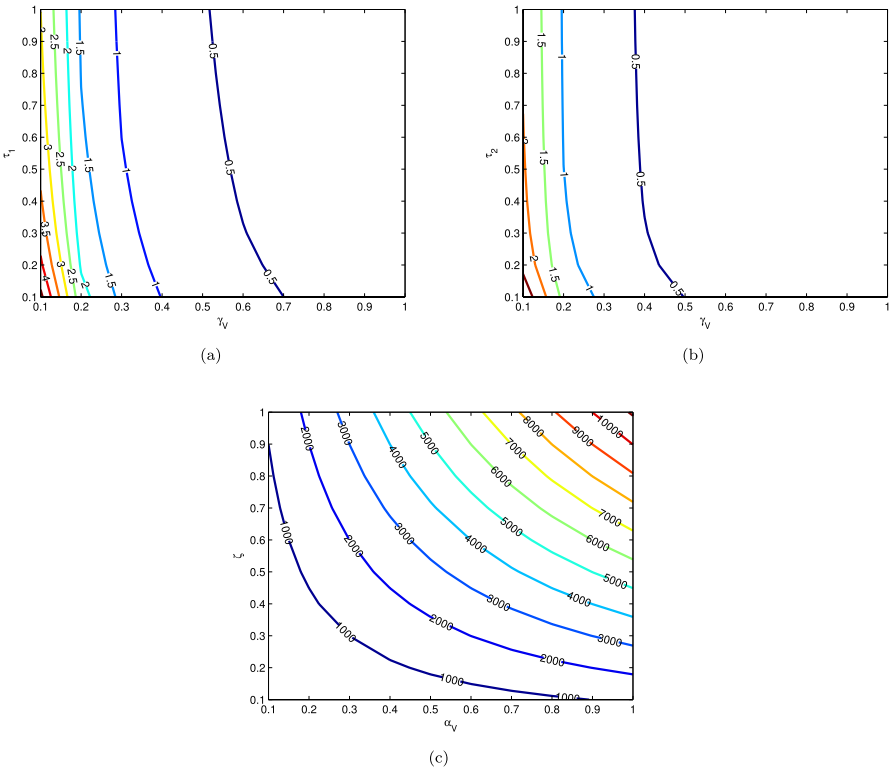

Fig. 6 Contour plot of the reproduction number ($\mathcal{R}_0^D$) as a function of **a**γ_V and τ_1, **b**γ_V and τ_2, **c**α_V and ζ

The second control measure involves environmental sanitation, such as the elimination of mosquito breeding sites by clearing waterlogged areas. This control measure is denoted by $u_2(t)$, where $u_2(t)$ represents the time-dependent effort directed toward environmental sanitation. Thus, by incorporating these control measures, the modified dengue model (1) becomes:

$$\begin{aligned}
\frac{dS_H}{dt} &= \alpha_H - (1-u_1(t))\lambda_H S_H - \gamma_H S_H + \varphi V_H,\\
\frac{dE_H}{dt} &= (1-u_1(t))\lambda_H S_H - (\eta+\gamma_H)E_H,\\
\frac{dI_A}{dt} &= (1-q)\eta E_H - (\psi+\tau_1+\gamma_H)I_A + (1-u_1(t))k\omega\lambda_H R_H,\\
\frac{dI_S}{dt} &= q\eta E_H + \psi I_A - (\tau_2+\delta+\gamma_H)I_S + (1-u_1(t))(1-k)\omega\lambda_H R_H,
\end{aligned}$$

$$\begin{aligned}
\frac{dT_H}{dt} &= \tau_1 I_A + \tau_2 I_S - (\sigma + \theta\delta + \gamma_H)T_H,\\
\frac{dR_H}{dt} &= \sigma T_H - (\xi + (1-u_1(t))\omega\lambda_H + \gamma_H)R_H,\\
\frac{dV_H}{dt} &= \xi R_H - (\varphi + \gamma_H)V_H,\\
\frac{dS_V}{dt} &= (1-u_2(t))(1-\phi I_V)\alpha_V - \lambda_V S_V - \gamma_V S_V,\\
\frac{dE_V}{dt} &= \lambda_V S_V - (\mu + \gamma_V)E_V,\\
\frac{dI_V}{dt} &= \mu E_V + (1-u_2(t))\phi\alpha_V I_V - \gamma_V I_V.
\end{aligned} \tag{43}$$

where

$$\lambda_H = \frac{\zeta\beta_H I_V}{N_H}, and \quad \lambda_V = \frac{\zeta\beta_V(I_A + I_S)}{N_H}.$$

For this, we considered the following objective functional

$$\mathscr{G}[u_1, u_2] = \int_0^{t_f} \left[c_1 I_A + c_2 I_S + \frac{1}{2}\left(c_3 u_1^2(t) + c_4 u_2^2(t)\right)\right] dt \tag{44}$$

In this context, t_f represents the final time, while c_1, c_2, c_3, and c_4 are weight constants designed to balance the terms in the integrand (44), ensuring that no single term dominates. The term $c_1 I_A + c_2 I_S$ accounts for the cost of monitoring infected individuals across all stages. The term $c_3 u_1^2(t)$ reflects the cost associated with acquiring mosquito bed-nets, while $c_4 u_2^2(t)$ represents the implementation cost of environmental sanitation measures.

The main objective here is to minimize both the number of infected individuals and the associated costs. The goal is to determine a pair optimal control $u_1(t)$ and $u_2(t)$ that satisfies the following condition:

$$\mathscr{G}[u_1^*, u_2^*] = \min_{u_1, u_2 \in \Omega} \mathscr{G}[u_1, u_2] \tag{45}$$

where the control set (Ω) is defined as

$$\Omega = \left\{(u_1(t), u_2(t)) \in \mathscr{L}^1(0, t_f) | a_1 \le u_1(t) \ge b_1, a_2 \le u_2(t) \ge b_2\right\} \tag{46}$$

is lesbegue measurable.

4.1 Characterization of the optimal control model

As previously mentioned above, the incorporation of optimal control strategies into the dengue transmission model allows us to determine the most effective measures for minimizing disease and it is important to note that the characterization of the optimal control model provides a fundamental framework for analyzing the systems dynamic where the proposed

intervention strategies can be adjusted over time. So, here, we shall present the necessary conditions for the optimal control using the Pontryagin's Maximum Principle Pontryagin (2018). This principle is used to converts (43) and (44) into an optimization problem of minimizing the Hamiltonian $\mathcal{H}$, with respect to the pre-defined controls ($u_1(t)$ and $u_2(t)$) at each point in time. To do this, we begin by constructing the Hamiltonian $\mathcal{H}$ from the objective functional given in (44) with the system dynamics described by (43). This therefore results in the following optimality conditions:

$$\begin{aligned}
\mathcal{H} = {} & c_1 I_A + c_2 I_S + \frac{1}{2}\left(c_3 u_1^2(t) + c_4 u_2^2(t)\right) + \mathcal{L}_1\left[\alpha_H - \frac{(1-u_1(t))\zeta\beta_H I_V S_H}{N_H} - \gamma_H S_H + \varphi V_H\right] + \\
& \mathcal{L}_2\left[\frac{(1-u_1(t))\zeta\beta_H I_V S_H}{N_H} - (\eta+\gamma_H)E_H\right] + \\
& \mathcal{L}_3\left[(1-q)\eta E_H - (\psi+\tau_1+\gamma_H)I_A + \frac{(1-u_1(t))k\omega\zeta\beta_H I_V R_H}{N_H}\right] + \\
& \mathcal{L}_4\left[q\eta E_H + \psi I_A - (\tau_2+\delta+\gamma_H)I_S + \frac{(1-u_1(t))(1-k)\omega\zeta\beta_H I_V R_H}{N_H}\right] + \\
& \mathcal{L}_5\left[\tau_1 I_A + \tau_2 I_S - (\sigma+\theta\delta+\gamma_H)T_H\right] + \mathcal{L}_6\left[\sigma T_H - \left(\xi + \frac{(1-u_1(t))\omega\zeta\beta_H I_V}{N_H} + \gamma_H\right)R_H\right] + \\
& \mathcal{L}_7\left[\xi R_H - (\varphi+\gamma_H)V_H\right] + \mathcal{L}_8\left[(1-u_2(t))(1-\phi I_V)\alpha_V - \frac{\zeta\beta_V(I_A+I_S)S_V}{N_H} - \gamma_V S_V\right] + \\
& \mathcal{L}_9\left[\frac{\zeta\beta_V(I_A+I_S)S_V}{N_H} - (\mu+\gamma_V)E_V\right] + \mathcal{L}_{10}\left[\mu E_V + (1-u_2(t))\phi\alpha_V I_V - \gamma_V I_V\right].
\end{aligned} \tag{47}$$

Where the adjoint functions associated to the state variables S_H, E_H, I_A, I_S, T_H, R_H, V_H, S_V, E_V, I_V are $\mathcal{L}_1, \mathcal{L}_2, \mathcal{L}_3, \mathcal{L}_4, \mathcal{L}_5, \mathcal{L}_6, \mathcal{L}_7, \mathcal{L}_8, \mathcal{L}_9, \mathcal{L}_{10}$ respectively. Also, we obtained the system of adjoint equations by taking the partial derivatives of the Hamiltonian (47) with respect to the associated state and control variables.

Theorem 5 *Given the optimal control pair u_1^* and u_2^*, and the solutions S_H^*, E_H^*, I_A^*, I_S^*, T_H^*, R_H^*, V_H^*, S_V^*, E_V^*, I_V^* of the corresponding state system (43) that minimizes $\mathcal{G}[u_1, u_2]$ over Ω, then there exists adjoint functions $\mathcal{L}_1, \mathcal{L}_2, \mathcal{L}_3, \mathcal{L}_4, \mathcal{L}_5, \mathcal{L}_6, \mathcal{L}_7, \mathcal{L}_8, \mathcal{L}_9, \mathcal{L}_{10}$, such that*

$$\begin{aligned}
&\frac{d\mathcal{L}_1}{dt} = \Delta_1, \frac{d\mathcal{L}_2}{dt} = \Delta_2, \frac{d\mathcal{L}_3}{dt} = \Delta_3, \frac{d\mathcal{L}_4}{dt} = \Delta_4, \frac{d\mathcal{L}_5}{dt} = \Delta_5, \\
&\frac{d\mathcal{L}_6}{dt} = \Delta_6, \frac{d\mathcal{L}_7}{dt} = \Delta_7, \frac{d\mathcal{L}_8}{dt} = \Delta_8, \frac{d\mathcal{L}_9}{dt} = \Delta_9, \frac{d\mathcal{L}_{10}}{dt} = \Delta_{10}.
\end{aligned} \tag{48}$$

The expression for Δ_1, Δ_2, Δ_3, Δ_4, Δ_5, Δ_6, Δ_7, Δ_8, Δ_9, and Δ_{10} are in Appendix A.

Furthermore, the transerversality conditions is given by

$$\mathcal{L}_i(t_f) = 0, \quad i = 1, 2, 3, ..., 10. \tag{49}$$

The following characterization holds;

$$
\begin{aligned}
u_1^*(t) &= \max\left\{0, \min\left(1, \frac{1}{c_3}\left(\frac{(\mathcal{L}_2-\mathcal{L}_1)\zeta\beta_H I_V S_H}{N_H} + \frac{(\mathcal{L}_3 k + \mathcal{L}_4(1-k) - \mathcal{L}_6))\omega\zeta\beta_H I_V R_H}{N_H}\right)\right)\right\},\\
u_2^*(t) &= \max\left\{0, \min\left(1, \frac{\mathcal{L}_8(1-\phi I_V)\alpha_V + \mathcal{L}_{10}\phi\alpha_V I_V}{c_4}\right)\right\}.
\end{aligned} \tag{50}
$$

Proposition 1 *Corollary 4.2 of (Fleming and Rashel 1975 Fleming and Reshel (1975) gives the existence of an optimal control pair ($u_1(t)$, and $u_2(t)$) due to the convexity of the integrand of $\mathscr{G}$ with respect of ($u_1(t)$, and $u_2(t)$), a prior boundedness of the state solutions, and the local Lipschitz property of the model (43) with respect to the variables.*

Proof Using the Pontryagin's Maximum Principles, we obtained

$$
\begin{aligned}
\frac{d\mathcal{L}_1}{dt} &= -\frac{\partial\mathcal{H}}{\partial S_H}, \quad \mathcal{L}_1(t_f) = 0,\\
\frac{d\mathcal{L}_2}{dt} &= -\frac{\partial\mathcal{H}}{\partial E_H}, \quad \mathcal{L}_2(t_f) = 0,\\
\frac{d\mathcal{L}_3}{dt} &= -\frac{\partial\mathcal{H}}{\partial I_A}, \quad \mathcal{L}_3(t_f) = 0,\\
\frac{d\mathcal{L}_4}{dt} &= -\frac{\partial\mathcal{H}}{\partial I_S}, \quad \mathcal{L}_4(t_f) = 0,\\
\frac{d\mathcal{L}_5}{dt} &= -\frac{\partial\mathcal{H}}{\partial T_H}, \quad \mathcal{L}_5(t_f) = 0,\\
\frac{d\mathcal{L}_6}{dt} &= -\frac{\partial\mathcal{H}}{\partial R_H}, \quad \mathcal{L}_6(t_f) = 0,\\
\frac{d\mathcal{L}_7}{dt} &= -\frac{\partial\mathcal{H}}{\partial V_H}, \quad \mathcal{L}_7(t_f) = 0,\\
\frac{d\mathcal{L}_8}{dt} &= -\frac{\partial\mathcal{H}}{\partial S_V}, \quad \mathcal{L}_8(t_f) = 0,\\
\frac{d\mathcal{L}_9}{dt} &= -\frac{\partial\mathcal{H}}{\partial E_V}, \quad \mathcal{L}_9(t_f) = 0,\\
\frac{d\mathcal{L}_{10}}{dt} &= -\frac{\partial\mathcal{H}}{\partial I_V}, \quad \mathcal{L}_{10}(t_f) = 0,
\end{aligned} \tag{51}
$$

and considering the optimality conditions;

$$
\frac{\partial\mathcal{H}}{\partial u_1} = 0, and \quad \frac{\partial\mathcal{H}}{\partial u_2} = 0. \tag{52}
$$

This optimal control pair ($u_1(t)$, and $u_2(t)$) can be obtained subject to the state variables. By applying a bounded interval on the controls, the characterization can be solved as follows;

For the control $u_1(t)$, we have

$$
\frac{\partial\mathcal{H}}{\partial u_1} = u_1 c_3 - \frac{(\mathcal{L}_2-\mathcal{L}_1)\zeta\beta_H I_V S_H}{N_H} - \frac{(\mathcal{L}_3 k + \mathcal{L}_4(1-k) - \mathcal{L}_6))\omega\zeta\beta_H I_V R_H}{N_H} = 0 \tag{53}
$$

we have that

$$u_1^*(t) = \frac{1}{c_3}\left(\frac{(\mathcal{L}_2 - \mathcal{L}_1)\zeta\beta_H I_V S_H}{N_H} + \frac{(\mathcal{L}_3 k + \mathcal{L}_4(1-k) - \mathcal{L}_6))\omega\zeta\beta_H I_V R_H}{N_H}\right). \quad (54)$$

For the control $u_2(t)$, we have

$$\frac{\partial \mathcal{H}}{\partial u_2} = u_2 c_4 - \mathcal{L}_8(1 - \phi I_V)\alpha_V - \mathcal{L}_{10}\phi\alpha_V I_V = 0, \quad (55)$$

which yields

$$u_2^*(t) = \frac{\mathcal{L}_8(1 - \phi I_V)\alpha_V + \mathcal{L}_{10}\phi\alpha_V I_V}{c_4}. \quad (56)$$

Clearly, the optimality conditions obtained by differentiating the Hamiltonian with respect to the controls are valid for points inside the control set.

This concludes the proof. . □

4.2 Numerical simulations of the dengue model with optimal control

Similar to how the numerical simulations were conducted in subsect. 3.8, here we also perform the numerical simulation of the extended model that combined the control measures as the incorporation of optimal control into the model allows us to evaluate the effectiveness of these measures under different conditions. However, the computational approach adopted here helps to determine the optimal control solution which involves employing the forward-backward sweep method. This approach starts with an initial conjecture for the optimal controls and the algorithm proceeds by advancing the state variables forward in time after which we utilize the fourth-order Runge–Kutta method. Following this forward simulation, the state variables and the initial control hypothesis are used to retroactively solve the adjoint Eq. (48) by moving backward in time. Thus, the backward time integration is accomplished through the implementation of the fourth-order Runge–Kutta method in reverse. The controls, denoted as $u_1(t)$, and $u_2(t)$ are subsequently updated and employed to solve the state and adjoint systems anew.

This iterative process persists until a significant convergence is attained in the current state, adjoint, and control variables, as elucidated in prior studies Lenhart and Workman (2007); Kang et al. (2024); Hassan et al. (2024). The parameter values utilized for the numerical illustration are delineated in Table 3. The value of the weight factors used are $c_1 = 1$, $c_2 = 1$, $c_3 = 0.15$, and $c_4 = 0.08$. The control measures are combination of efforts involving the following

1. Strategy A: Control with $u_1(t)$ only.
2. Strategy B: Control with $u_2(t)$ only.
3. Strategy C: Combination of control $u_1(t)$ and control $u_2(t)$.

4.2.1 Strategy A: control with $u_1(t)$ only

See Fig. 7.

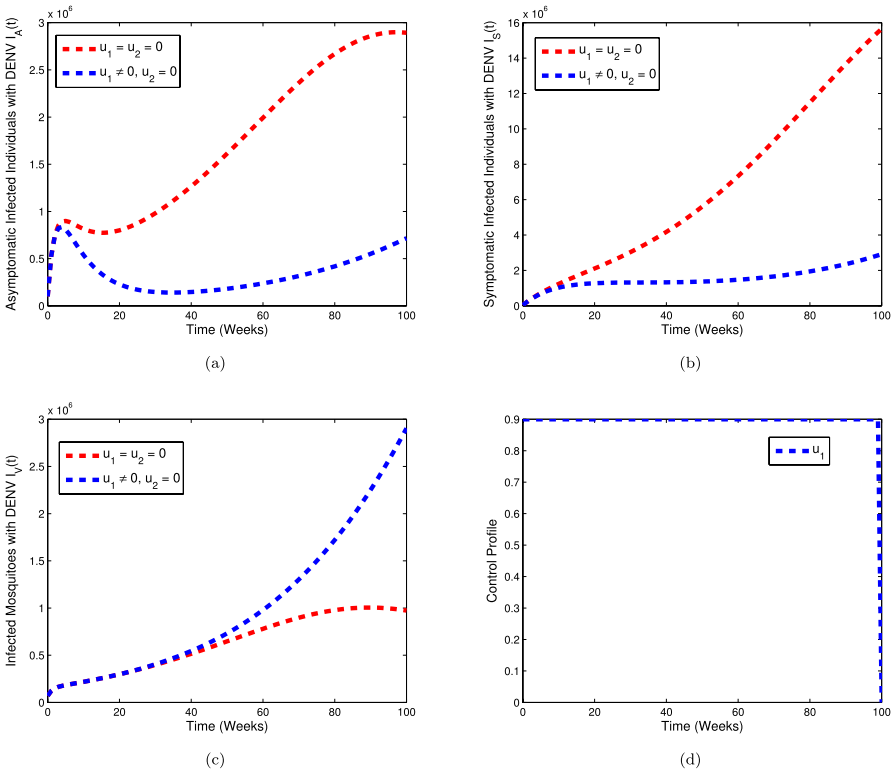

Fig. 7 Simulation of **a** the effect of u_1 on asymptomatic infected individuals with DENV, **b** effect of u_1 on symptomatic infected individuals with DENV, **c** effect of u_1 on infected mosquitoes with DENV, **d** the control profile u_1

4.2.2 Strategy B: control with $u_2(t)$ only

See Fig. 8.

4.2.3 Strategy C: combination of control $u_1(t)$ and control $u_2(t)$

See Fig. 9.

4.3 Discussion of results

The discussion of our results displayed in Figs. 7, 8, 9 are provided here. To start with, Fig. 7 displays the simulation result of the impact of strategy A, which is the usage of mosquito bed-net $(u_1(t))$ as a standalone measure on the asymptomatic infected individuals with DENV, symptomatic infected individuals with DENV, and Infected mosquitoes with DENV. When strategy A is implemented, we could observe from the Fig. 7a that the number of asymptomatic infected individuals with DENV reduces from 1000000 to 150000 in 30 weeks, and further reduces from 2900000 to 750000 in 100 weeks. In Fig. 7b , It could be clearly seen that the number of symptomatic infected individuals with DENV reduces

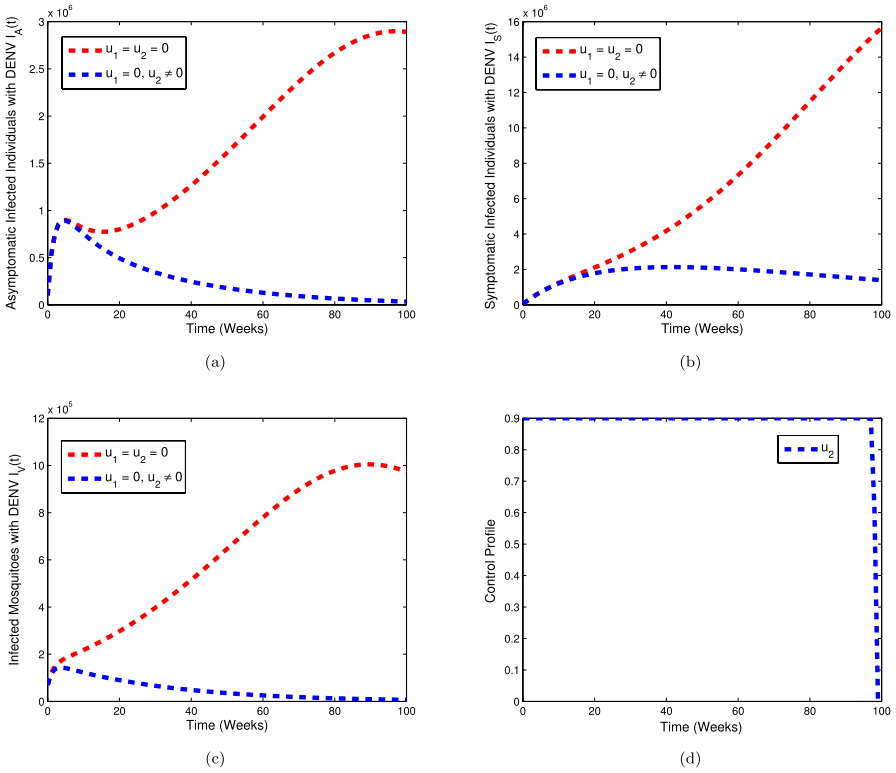

Fig. 8 Simulation of **a** the effect of u_2 on asymptomatic infected individuals with DENV, **b** effect of u_2 on symptomatic infected individuals with DENV, **c** effect of u_2 on infected mosquitoes with DENV, **d** the control profile u_2

from 3200000 to 1050000 in 30 weeks, and reduces from 15800000 to around 2800000 in 100 weeks. In Fig. 7c , it was observed that there was no changes in the number of infected mosquitoes with DENV within 40 weeks, but it later observed that the number of infected mosquitoes increases from 1000000 to 3000000 in 100 weeks when the strategy A was implemented. Figure 7d is the corresponding control profile.

Figure 8 is the simulation of the impact of strategy B, which is environment sanitation such as clearing of water logs to eliminate mosquito breeding sites ($u_2(t)$) as a standalone measure on the asymptomatic infected individuals with DENV, symptomatic infected individuals with DENV, and Infected mosquitoes with DENV. When strategy B is implemented, Fig. 8a shows that the number of asymptomatic infected individuals with DENV reduces from 1000000 to 300000 in 30 weeks, and further reduces from 2900000 to less than 100000 in 100 weeks. In Fig. 8b , it is observed that the number of symptomatic infected individuals with DENV reduces from 3200000 to 2000000 in 30 weeks, and reduces from 15800000 to around 1700000 in 100 weeks. Moreover, in Fig. 8c , it was observed that the number of infected mosquitoes with DENV reduces from 380000 to 70000 in 30 weeks, and further reduces from 998000 to less than 10000 in 100 weeks when the strategy B was implemented. Figure 8d is the corresponding control profile.

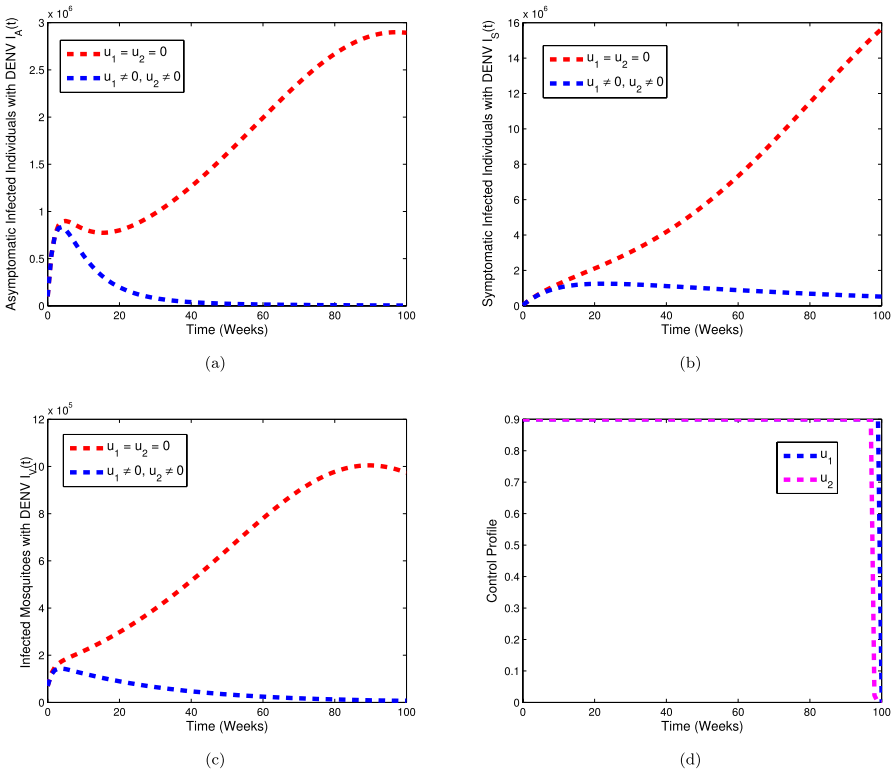

Fig. 9 Simulation of **a** the effect of u_1 and u_2 on asymptomatic infected individuals with DENV, **b** effect of u_1 and u_2 on symptomatic infected individuals with DENV, **c** effect of u_1 and u_2 on infected mosquitoes with DENV, **d** the control profiles u_1 and u_2

Figure 9 is the simulation of the impact of strategy C, which is the combination of the usage of mosquito bed-net ($u_1(t)$)) and the environment sanitation such as clearing of water logs to eliminate mosquito breeding sites ($u_2(t)$) on the asymptomatic infected individuals with DENV, symptomatic infected individuals with DENV, and Infected mosquitoes with DENV. When strategy C is implemented, as also seen in Fig. 9a , the number of asymptomatic infected individuals with DENV reduces from 1000000 to around 120000 in 30 weeks, and further reduces from 2900000 to less than 100000 in 100 weeks. In Fig. 9b , It is observed that the number of symptomatic infected individuals with DENV reduces from 3200000 to 1050000 in 30 weeks, and reduces from 15800000 to around 800000 in 100 weeks. In Fig. 9c , it was observed that the number of infected mosquitoes with DENV reduces from 380000 to 70000 in 30 weeks, and further reduces from 998000 to less than 10000 in 100 weeks when the strategy C was implemented. Figure 9d is the corresponding control profiles.

Based on the availability of efficacy and immunological data for model parameterization, this study examined the two approved dengue vaccines, Dengvaxia and TAK-003. However, there was no specific modeling of regional variations in vaccine coverage, acceptance, and implementation tactics. These variables may have an impact on vaccination programs

Table 4 Goodness of fit statistics for the dengue model compared with observed dengue case data (Weeks one to week thirty, 2024)

Statistic	Value
Coefficient of determination (R^2)	0.982
Root Mean Square Error (RMSE)	1.34×10^5
Mean Absolute Percentage Error (MAPE)	3.8%

in the real world and can be added to future model extensions to more accurately represent behavioral and regional variation.

The study's findings are largely consistent with earlier attempts to model dengue that took into account host-vector dynamics and vaccination strategies. Our study confirms the conclusions of Ferguson et al. (2016) and Coudeville et al. (2017) that the long-term decrease in the incidence of dengue is significantly influenced by vaccine efficacy and coverage. However, by explicitly combining Dengvaxia and TAK-003 into a single transmission structure, our model surpasses previous studies that typically focused on one vaccine at a time. Furthermore, by incorporating transovarial (vertical) transmission, this model deviates from conventional SEIR-type dengue or malaria models (e.g., Chitnis et al. (2008), which only address mosquito infection by horizontal transmission).

Also, the study also highlights Key factors influencing dengue transmission are highlighted in the study, including mosquito biting rate transmission probabilities, and mosquito mortality rate. These findings imply that infection risk can be considerably reduced by lowering mosquito density and human-vector contact through vector control, bed net use, and sanitation. Combining protective and preventive measures has the biggest impact, according to the optimal control analysis. Therefore, integrated vector management provides an affordable method of reducing dengue. The results offer useful recommendations for public health tactics in endemic areas (Table 4).

5 Conclusion

In this work, we presented a mathematical model which was formulated to improve our understanding of the complex transmission dynamics of the Dengue virus. This model investigate the interactions between both human and mosquito populations, while primarily focusing on the vector species Aedes aegypti and Aedes albopictus. We incorporated key factors such as vaccination of individuals previously infected with Dengue and transovarial transmission within the mosquito population. Furthermore, These factors were added to the model to adequately capture the various transmission routes and mechanisms which influence the rapid spread of the virus. To thoroughly analyze the model, we calculated the basic reproduction number ($\mathcal{R}_0^D$), which is a crucial threshold parameter that determines the potential for disease transmission. In addition, we examined the existence and stability of both the dengue-free and dengue-endemic equilibrium points. A sensitivity analysis of the basic reproduction number was also conducted to identify the parameters most influential in the transmission of dengue. The insights obtained from this analysis allowed us to pinpoint critical parameters that could be targeted for intervention strategies. To validate the model, we fitted it to the actual Dengue cases in Brazil from week 1 to week 30 of the year 2024 and this validation enable us to assess the accuracy of the model and refine its parameters for more reliable predictions. Furthermore, in order to determine the effectiveness of potential control measures, we extended the model by incorporating two time-dependent control

strategies namely: the use of mosquito bed nets ($u_1(t)$) and environmental sanitation practices ($u_2(t)$). The key results from this work are then summarized as follows:

1. The Dengue model demonstrates the occurrence of backward bifurcation, which is a well-known characteristic in which both a stable Dengue-free equilibrium and a stable Dengue-endemic equilibrium coexist when the basic reproduction number is less than one. Biologically, the backward bifurcation property implies that even when the basic reproduction number is below one which is typically considered as the necessary condition for the eradication of Dengue fever, this condition is no longer sufficient for the complete elimination of the Dengue virus from the population. As a result, this makes the eradication of the Dengue virus more challenging within the population.
2. The sensitivity analysis showed that the most influential parameters that are responsible for rapid transmission of the Dengue virus are the disease introduction rate of new mosquitoes into the population presented by (α_V), the mosquito-biting rate (ζ), the transmission probability of infected mosquitoes carrying DENV to susceptible individuals (β_H), and the transmission probability of infected individuals with DENV spreading the virus to susceptible mosquitoes (β_V). These parameters play a critical role in the overall spread of the virus.
3. Finally, the results from the optimal control analysis demonstrate that the combined efforts that involve the use of both control measures have a more significant impact on controlling the Dengue virus than the application of any single measure alone.

Based on these findings, we recommend the following actions to mitigate the rapid spread of the disease:

1. *Eliminate Mosquito Breeding Sites* Adopting regular emptying, cleaning, or covering of containers that collect standing water, such as buckets, flowerpots, and discarded tires would be essential in eliminating mosquito breeding. In addition, ensuring that drains and gutters are clear of debris to prevent water stagnation, which serves as breeding grounds for mosquitoes.
2. *Install Physical Barriers* The use of window and door screens, or sleeping under mosquito nets, especially in high-risk areas, will help to reduce the human direct exposure to mosquitoes.
3. *Vaccination* The use of vaccines like Dengvaxia whenever available can further help reduce the risk of infection. It is crucial to consult local health authorities for updated vaccination guidelines and recommendations specific to the region.

Hence, by collectively implementing these measures, communities can significantly reduce the risk of Dengue fever transmission.

Appendix A: Expression adjiont functions

$$\Delta_1 = -\mathcal{L}_1\left(\frac{(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2} - \frac{(1-u_1(t))\zeta\beta_H I_V}{N_H} - \gamma_H\right) +$$
$$\mathcal{L}_2\left(\frac{(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2} - \frac{(1-u_1(t))\zeta\beta_H I_V}{N_H}\right) + \frac{(\mathcal{L}_3 k + \mathcal{L}_4(1-k) - \mathcal{L}_6)(1-u_1(t))\omega\zeta\beta_H I_V R_H}{N_H^2}$$
$$+ \frac{(\mathcal{L}_9 - \mathcal{L}_8)\zeta\beta_V(I_A + I_S)S_V}{N_H^2},$$
$$\Delta_2 = -\frac{\mathcal{L}_1(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2} + \mathcal{L}_2\left(\frac{(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2} + \eta + \gamma_H\right) -$$
$$\mathcal{L}_3\left((1-q)\eta - \frac{(1-u_1(t))k\omega\zeta\beta_H I_V R_H}{N_H^2}\right) - \mathcal{L}_4\left(q\eta - \frac{(1-u_1(t))(1-k)\omega\zeta\beta_H I_V R_H}{N_H^2}\right) -$$
$$\frac{\mathcal{L}_6(1-u_1(t))\omega\zeta\beta_H I_V R_H}{N_H^2} + \frac{(\mathcal{L}_9 - \mathcal{L}_8)\zeta\beta_V(I_A + I_S)S_V}{N_H^2},$$

$$\Delta_3 = -c_1 - \frac{(\mathcal{L}_1 - \mathcal{L}_2)(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2} + \mathcal{L}_3\left(\psi + \tau_1 + \gamma_H + \frac{(1-u_1(t))k\omega\zeta\beta_H I_V R_H}{N_H^2}\right) -$$
$$\mathcal{L}_4\left(\psi - \frac{(1-u_1(t))(1-k)\omega\zeta\beta_H I_V R_H}{N_H^2}\right) - \mathcal{L}_5\tau_1 - \frac{\mathcal{L}_6(1-u_1(t))\omega\zeta\beta_H I_V R_H}{N_H^2} +$$
$$(\mathcal{L}_9 - \mathcal{L}_8)\left(\frac{\zeta\beta_V(I_A + I_S)S_V}{N_H^2} - \frac{\zeta\beta_V S_V}{N_H}\right),$$
$$\Delta_4 = -c_2 - \frac{(\mathcal{L}_1 - \mathcal{L}_2)(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2} + \frac{\mathcal{L}_3(1-u_1(t))k\omega\zeta\beta_H I_V R_H}{N_H^2} +$$
$$\mathcal{L}_4\left(\tau_2 + \delta + \gamma_H + \frac{(1-u_1(t))(1-k)\omega\zeta\beta_H I_V R_H}{N_H^2}\right) - \mathcal{L}_5\tau_2 - \frac{\mathcal{L}_6(1-u_1(t))\omega\zeta\beta_H I_V R_H}{N_H^2} +$$
$$(\mathcal{L}_9 - \mathcal{L}_8)\left(\frac{\zeta\beta_V(I_A + I_S)S_V}{N_H^2} - \frac{\zeta\beta_V S_V}{N_H}\right),$$

$$\Delta_5 = -\frac{(\mathcal{L}_1 - \mathcal{L}_2)(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2} + \frac{(\mathcal{L}_3 k + \mathcal{L}_4(1-k))(1-u_1(t))\omega\zeta\beta_H I_V R_H}{N_H^2} +$$
$$\mathcal{L}_5(\sigma + \theta\delta + \gamma_H) - \mathcal{L}_6\left(\sigma + \frac{((1-u_1(t))\omega\zeta\beta_H I_V R_H}{N_H^2}\right) + \frac{(\mathcal{L}_9 - \mathcal{L}_8)\zeta\beta_V(I_A + I_S)S_V}{N_H^2},$$
$$\Delta_6 = -\frac{(\mathcal{L}_1 - \mathcal{L}_2)(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2} + (1-u_1(t))(\mathcal{L}_3 k + \mathcal{L}_4(1-k))\omega\left(\frac{\zeta\beta_H I_V R_H}{N_H^2} - \frac{\zeta\beta_H I_V}{N_H}\right) -$$
$$\mathcal{L}_6\left(\frac{(1-u_1(t))\omega\zeta\beta_H I_V R_H}{N_H^2} - \frac{(1-u_1(t))\omega\zeta\beta_H I_V}{N_H} - \xi - \gamma_H\right) - \mathcal{L}_7\xi +$$
$$\frac{(\mathcal{L}_9 - \mathcal{L}_8)\zeta\beta_V(I_A + I_S)S_V}{N_H^2},$$

$$
\begin{aligned}
\Delta_7 &= -\mathcal{L}_1\left(\frac{(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2}+\varphi\right)+\frac{\mathcal{L}_2(1-u_1(t))\zeta\beta_H I_V S_H}{N_H^2}+\\
&\quad\frac{(\mathcal{L}_3 k+\mathcal{L}_4(1-k)-\mathcal{L}_6)(1-u_1(t))\omega\zeta\beta_H I_V R_H}{N_H^2}+\mathcal{L}_7(\varphi+\gamma_H)+\frac{(\mathcal{L}_9-\mathcal{L}_8)\zeta\beta_V(I_A+I_S)S_V}{N_H^2},\\
\Delta_8 &= \mathcal{L}_8\left(\frac{\zeta\beta_V(I_A+I_S)}{N_H}+\gamma_V\right)-\frac{\mathcal{L}_9\zeta\beta_V(I_A+I_S)}{N_H},\\
\Delta_9 &= \mathcal{L}_9(\mu+\gamma_V)-\mathcal{L}_{10}\mu,\\
\Delta_{10} &= \frac{(\mathcal{L}_1-\mathcal{L}_2)(1-u_1(t))\zeta\beta_H S_H}{N_H}-\frac{(\mathcal{L}_3 k+\mathcal{L}_4(1-k)-\mathcal{L}_6)(1-u_1(t))\omega\zeta\beta_H R_H}{N_H}+\\
&\quad\mathcal{L}_8(1-u_2(t))\phi\alpha_V-\mathcal{L}_{10}((1-u_2(t))\phi\alpha_V-\gamma_V).
\end{aligned}
\tag{A1}
$$

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Declarations

Conflict of interest The authors declare that they have no conflict of interest.

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