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Research article

Global stability and sensitivity analysis of vector-host dengue mathematical model

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Abstract: Dengue impacts 129 nations, threatens over 50% of the global population, and results in around 400 million illnesses annually. The purpose of this paper was to build the global stability and sensitivity analysis of a vector-host dengue mathematical model with compartments of symptomatic and hospitalized infected humans. Additionally, it aimed to assess the impact of the immunological response of vulnerable individuals, through the ingestion of natural foods, on the transmission of the disease. The solution's positivity and boundedness proved the model's mathematical well-posedness. To examine endemicity, the reproduction number was calculated using the next-generation technique. The Lyapunov function approach was employed to illustrate the model's global stability. Our mathematical discoveries were illustrated through numerical simulations of the dengue epidemic. The dynamical system sensitivity analysis suggests that the best way to control illness is to increase the immune system rate of susceptible hosts by consuming natural foods.

Keywords: basic reproduction number (R_0); dengue; lyapunov; sensitivity; global stability
Mathematics Subject Classification: 34D08, 34D20, 34D23, 92D30, 93D05, 97M10, 97M60

1. Introduction

Dengue fever, a highly transmissible illness, threatens more than two to three million individuals annually on a global scale [1,2]. Dengue, the second most lethal mosquito-borne illness after malaria,

results in hundreds of fatalities and over 4 million infections each year [3]. More than 129 nations are home to it, and it can cause from mild to severe symptoms, especially in the tropics and subtropics [4,5]. About three quarters of all dengue cases are dengue fever (DF), the most prevalent type of disease. Five distinct serotypes of dengue virus define the viral infection known as dengue fever, which is transmitted by mosquitoes: DEN-1, DEN-2, DEN-3, DEN-4, and DEN-5. When mosquitoes bite an infected person, they take the virus and let it incubate for eight to ten days. Following a mosquito bite that carries an infection, patients often start symptoms for five to seven days after. DF symptoms include a high temperature, intense headaches, joints and muscles discomfort, and rashes [6,7].

People, insects, and the virus disseminate dengue in several environments. Due to space complexity, dengue transmission studies are complex [8]. The epidemic has many causes. Worldwide host and vector mobility boosted viral circulation, urban congestion encouraged multiple transmissions from a single infected vector, and vector control mechanisms were lost. Temperature, precipitation, and humidity affect vector development from egg viability to adult longevity and dispersal, among other dengue transmission factors [9]. Unplanned construction, high population density, and unstable trash collection promote mosquito breeding sites and dengue.

Dengue has no cure; thus, severe cases require hospitalization and supportive care [10]. A cheap, efficient vaccine is essential for universal control. Governments mostly use vector control and insect repellents to lower infection rates. Community education about dengue hazards is essential for prevention [11]. Media campaigns and community involvement may spread preventative knowledge. Community-government miscommunications can raise dengue risk, making ongoing community awareness crucial for successful interventions.

Mathematical models of infectious illness are valuable tools for comprehending and forecasting the patterns of outbreaks, comprehending the advancement and development of diseases, and estimating the consequences of health interventions on people or populations [12–14]. The models generated by mathematics can be used to improve tactics for reducing disease spread and enhance public health decisions with epidemiological evidence. Numerous authors have widely employed mathematical modeling to inform public health initiatives aimed at controlling the transmission of dengue [15,16]. The mathematical modeling approach is quite demanding due to the intricate nature of the dengue transmission mechanism. Utilizing a more intricate model can enhance the accuracy of the modeling process, but it also presents challenges when attempting to derive analytical data and draw conclusions. Therefore, the researcher must construct a practical yet uncomplicated model based on realistic assumptions. Utilizing actual incidence data is essential for accurately adjusting the model's performance. Various methodologies can be employed to build the dengue transmission model, including differential equations, fractional differential equations, stochastic differential equations, and alternative techniques. Rao et al. [17] introduced a variational model for delayed impulsive epidemic models, tackling mathematical challenges in the reaction-diffusion model with a delayed impulse. Yang et al. [18] investigated the presence of positive periodic solutions to neutral-type integral differential equations inside an epidemic model, employing Mawhin's continuation theorem and the characteristics of neutral-type operators. Zhao et al. [19] explored the stability of impulsive stochastic competition models with time-varying delays, focusing on persistence, extinction, and practical exponential stability. Xiao et al. [20] provided an innovative methodology for analyzing the dynamics of rumors inside social networks, differentiating between enthusiasts and ordinary individuals. Hattaf [21] presented a novel definition of fractional derivatives that integrates singular and non-singular kernels, along with an associated fractional integral, augmenting their utility in computational biology. Hattaf [22]

introduced a novel class of generalized differential and integral operators that generalize the definitions of fractal-fractional derivatives and integral operators employed to model complex dynamics in various disciplines.

Many modeling analysis of viral illnesses like dengue, chikungunya, and zika have been conducted in the literature. Naaly et al. [23] studied how vector management, treatment, and mass awareness affect dengue fever transmission dynamics. The authors in [24] discovered awareness and control strategies for dengue disease transmission. Researchers Aldila et al. [25] looked into the interaction between social awareness, the detection of cases, and the capability of hospitals in Jakarta to eliminate dengue. Li et al. [26] analyzed the effects of awareness initiatives on dengue and identified the optimal control approaches. Bonyah et al. [27] performed an analysis of the fractional stochastic modeling of dengue illness, specifically focusing on the perspective of social awareness. The authors [28] conducted a mathematical analysis to investigate how community ignorance affects dengue population dynamics. Sood et al. [29] conducted an investigation on a sophisticated healthcare system designed to forecast and avert dengue virus illness. Macalalag et al. [30] analyzed the dengue model's global stability incorporating awareness and temporal delays. A dengue disease model was used by Saha et al. [31] to study the host-vector dynamics using the optimal control technique. The authors of [32] conducted a study on forecasting dengue epidemic outbreaks by utilizing climate variation and Monte Carlo techniques. Bhuju et al. [33] examined the fuzzy SEIR-SEI dengue disease model's sensitivity and bifurcation analysis. Harshit [34] conducted a sensitivity study to examine dengue transmission. Based on the sensitivity analysis of the dengue epidemic using a modified saturation incidence rate, the authors of [35] invented an optimal control strategy. Guo et al. [36] performed a stability analysis and simulation of a delayed dengue transmission model incorporating logistic growth and a nonlinear incidence rate. Leandro et al. [37] investigated the spatial distribution of dengue transmission in a city with a high incidence of the disease in Brazil, demonstrating significant spatial organization in the local dynamics of dengue transmission. Hasan et al. [38] performed a sensitivity analysis of a dynamic dengue epidemic model incorporating vector-host interactions. Abidemi et al. [39] performed a Lyapunov stability study and implemented optimization strategies for a model that simulates the transmission of dengue disease. The author of [40] conducted a thorough analysis of effective control measures for managing dengue infection in East Java. A study on the mathematical evaluation of the impact of hospitalization in dengue intervention was carried out by Nawawi et al. [41]. Researchers Hamdan et al. [42] used a case study of hospitalized infected individuals in Malaysia to determine the impact of temperature on creating a deterministic dengue epidemic model. The study by Abidemi et al. [43] offered valuable insights into the dynamics of dengue, with a specific focus on the asymptomatic, isolated, and vigilant compartments. Jan et al. [44] conducted a study on the transmission dynamics of dengue by asymptomatic carriers and the effectiveness of control strategies. A study by Jose et al. [45] used mathematical models to look into how the Zika virus and Dengue fever spread in people who show symptoms of both diseases.

All of these studies are predicated on deterministic models; however, none have examined the consequences of the infection rate within the host or the symptomatically infected and hospitalized humans in question. Furthermore, the immune system's enhancement through the consumption of natural foods and awareness of symptomatic human infections were not taken into account. In light of the aforementioned, this work presents a novel mathematical model that accurately captures the interactions between dengue hosts and vectors. The model includes compartments for infected individuals, individuals with symptoms, and individuals requiring hospitalization. This paradigm will

help us to better understand the reality of dengue transmission, and eradication.

The following parts of this work are arranged as follows: Our model is presented in Section 2, together with a biological justification of its parameters. Section 3 is devoted to analyzing the positivity as well as boundedness of the model. In addition, we analyze the equilibrium points and compute the basic reproduction number, represented as R_0. Section 4 discusses the stability of the model. Section 5 focuses on sensitivity analysis to illustrate the influence of the proposed model on R_0. In Section 6, we introduce a computational method for solving the model. Furthermore, this section presents the numerical findings and their accompanying explanations. Ultimately, the concluding portion of the study provides a concise summary of the main contributions of our work and identifies prospective areas for further research.

2. Dengue model formulation

The compartmental models provide a thorough description of how the epidemic spreads and the various preventive measures that can be taken to stop it. In order to create the epidemic model, we utilize the compartmental modeling approach. The model consists of seven compartments which represent two distinct sub-populations: the human (host) population and the vector (mosquito) population. The total host population, can be divided into five distinct groups: S_h represents susceptible humans who are capable of contracting the disease, I_{h0} signifies infected humans who are not capable of transmitting the disease to others, I_{h1} represents symptomatically infected humans who are able to transmit the disease to others, H_h implies hospitalized infected humans, and R_h reflects the recovered population who have temporary immunity against dengue. Similarly, the vector population N_m of mosquitoes is divided into two groups: mosquitoes capable of being infected (susceptible, S_m) and mosquitoes that are infected by the dengue virus (infected, I_m). Figure 1 shows a transmission diagram illustrating compartment interactions, while Table 1 describes the parameters.

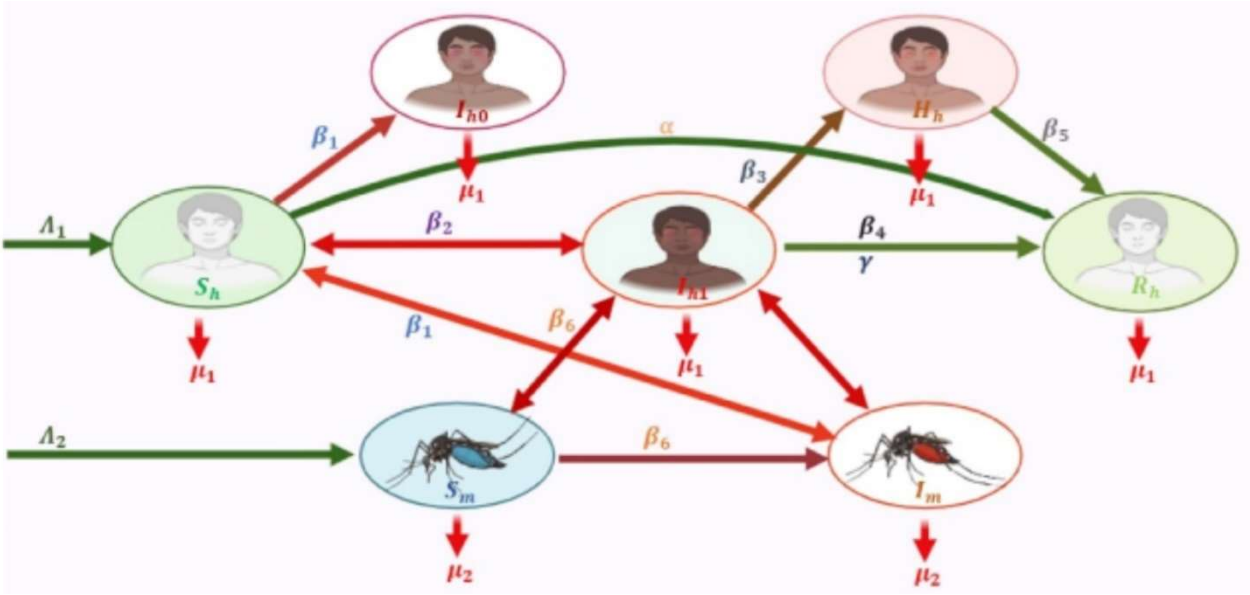

Figure 1. Dengue transmission diagram.

Table 1. Parameter's biological descriptions of dengue model.

Parameter	Descriptions
Λ_1	Human population recruitment rates
β_1	Infection transmission rate from vector to host
α	Rate of immune system of susceptible host by the consumption of natural foods
β_2	Infections rate within the host
μ_1	Human population natural mortality
β_3	Rate of symptomatic to hospitalized infected individuals
β_4	Symptomatic infection recovery rate
γ	Awareness rate among symptomatic infected people
β_5	Hospitalized infected individual's recovery rate
Λ_2	Vector population recruitment rates
β_6	Infection transmission rate from human to vector
μ_2	Vector population natural mortality

In order to construct the model, we take into account the rate of immune system of the susceptible host achieved by the consumption of natural foods, denoted by α. β_1 is the infection transmission rate from vector to host, and the infection rate within hosts is β_2. Additionally, the human awareness of individuals who are symptomatically infected (γ) has protected them from dengue by employing mosquito-nets and mosquito -repellent sprays. Also, the hospitalized infected humans H_h are introduced here.

The model is governed by the differential equations that are shown in the following system:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_1 - \beta_1 S_h I_m - \beta_2 S_h I_{h1} - \alpha S_h - \mu_1 S_h,\\
\frac{dI_{h0}}{dt} &= \beta_1 S_h I_m - \mu_1 I_{h0},\\
\frac{dI_{h1}}{dt} &= \beta_2 S_h I_{h1} - \beta_3 I_{h1} - \beta_4 I_{h1} - \gamma I_{h1} - \mu_1 I_{h1},\\
\frac{dH_h}{dt} &= \beta_3 I_{h1} - \beta_5 H_h - \mu_1 H_h,\\
\frac{dR_h}{dt} &= \beta_4 I_{h1} + \beta_5 H_h + \alpha S_h + \gamma I_{h1} - \mu_1 R_h,\\
\frac{dS_m}{dt} &= \Lambda_2 - \beta_6 S_m I_{h1} - \mu_2 S_m,\\
\frac{dI_m}{dt} &= \beta_6 S_m I_{h1} - \mu_2 I_m,
\end{aligned} \tag{2.1}$$

With initial host population $S_h(0) \geq 0, I_{h0}(0) \geq 0, I_{h1}(0) \geq 0, H_h(0) \geq 0, and\ R_h(0) \geq 0$, and the vector population $S_m(0) \geq 0, and\ I_m(0) \geq 0$.

3. Analysis of the dengue model

3.1. Positivity of the solution

A fundamental need for an epidemiological model is that its solutions must exhibit both non-negativity and boundedness. Therefore, it is essential to demonstrate that all variables remain positive for every instance where t is greater than zero.

Theorem 3.1. The system (2.1)'s feasible region stated as

$$\chi = \left\{S_h(t), I_{h0}(t), I_{h1}(t), H_h(t), R_h(t), S_m(t), I_m(t) \epsilon \mathbb{R}_7^+ : N_h(\mathrm{t}) \leq \frac{\Lambda_1}{\mu_1}, N_m(\mathrm{t}) \leq \frac{\Lambda_2}{\mu_2}\right\}$$

is positively invariant along with the initial condition defined by $\mathcal{R}_7^+$.

Proof. One way to express system (1) is :

$$\frac{dY}{dt} = K(Y) + Z, \tag{3.1}$$

$$Y = (S_h, I_{h0}, I_{h1}, H_h, R_h, S_m, I_m)^t,$$

$$K = \begin{pmatrix} -k_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ k_2 & -\mu_1 & 0 & 0 & 0 & 0 & 0 \\ k_3 & 0 & -k_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_5 & -k_6 & 0 & 0 & 0 \\ k_7 & 0 & k_8 & k_9 & -\mu_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -k_{10} & 0 \\ 0 & 0 & 0 & 0 & 0 & k_{11} & -\mu_2 \end{pmatrix},$$

where, $k_1 = \beta_1 I_m + \beta_2 I_{h1} + \alpha + \mu_1$, $k_2 = \beta_1 S_h I_m$, $k_3 = \beta_3 I_{h1}$, $k_4 = \beta_3 + \beta_4 + \gamma + \mu_1$, $k_5 = \beta_3 I_{h1}$, $k_6 = \beta_5 + \mu_1$, $k_7 = \alpha$, $k_8 = \beta_4 + \gamma$, $k_9 = \beta_5$, $k_{10} = \beta_6 I_{h1} + \mu_2$, $k_{11} = \beta_4 \mathrm{I_{h1}}$.

And

$$Z = (\Lambda_1, 0,0,0,0, \Lambda_2, 0)^t.$$

In this case, any entry of the matrix K(Y) off-diagonal is non-negative. Therefore, the matrix is referred to as the Metzler matrix. Furthermore, vector Z possesses a positive character. Consequently, it may be deduced that the system (3.1) is always positively invariant in the region $\mathcal{R}_7^+$.

3.2. Dengue-free equilibrium

Setting each system of model (2.1) to zero, results in the production of the dengue-free equilibrium (DFE). Additionally, the DFE has no infections or recovery procedures in place. Therefore, the DFE of the dengue model is provided by

$$E_0 = (S_h^0, I_{h0}^0, I_{h1}^0, H_h^0, R_h^0, S_m^0, I_m^0) = \left(\frac{\Lambda_1}{\mu_1 + \alpha}, 0,0,0,0, \frac{\Lambda_2}{\mu_2}, 0\right).$$

3.3. Estimation of the basic reproduction number

The basic reproduction number is a critical threshold in mathematical epidemiology research since it helps predict the possibility of disease transmission. Utilizing the next-generation matrix to calculate the system (1)'s R_0 as

$$F = \begin{pmatrix} 0 & 0 & \beta_1 S_h^0 \\ 0 & \beta_2 S_h^0 & 0 \\ 0 & \beta_6 S_m^0 & 0 \end{pmatrix}, V = \begin{pmatrix} \mu_1 & 0 & 0 \\ 0 & \beta_3 + \beta_4 + \mu_1 + \gamma & 0 \\ 0 & 0 & \mu_2 \end{pmatrix}.$$

The next-generation matrix FV^{-1} generates R_0 as

$$R_0 = \frac{\beta_2 \Lambda_1}{(\mu_1+\alpha)(\beta_3+\beta_4+\mu_1+\gamma)}.$$

3.4. Endemic equilibrium

The endemic equilibrium of system (2.1) is

$$E_1 = (S_h^*, I_{h0}^*, I_{h1}^*, H_h^*, R_h^*, S_m^*, I_m^*)$$

where,

$$S_h^* = \frac{\beta_3 + \beta_4 + \mu_1}{\beta_2}, I_{h0}^* = \frac{\beta_1 \beta_6 \Lambda_2 (\beta_3 + \beta_4 + \mu_1 + \gamma) I_{h1}^*}{\mu_1 \mu_2 (\beta_6 + \mu_2)},$$

$$I_{h1}^* = \frac{(\mu_1+\alpha)\mu_2(\beta_5 + \mu_1)(\beta_6 + \mu_2)(R_0 - 1)}{\beta_1 \beta_6 \Lambda_2 + \beta_2 \mu_2 (\beta_6 + \mu_2)}, H_h^* = \frac{\beta_3 I_{h1}^*}{\beta_5 + \mu_1},$$

$$R_h^* = \frac{\beta_2(\beta_4 + \gamma)(\beta_5 + \mu_1) + \beta_2 \beta_3 \beta_5) I_{h1}^* + \alpha(\beta_5 + \mu_1)(\beta_3 + \beta_4 + \mu_1)}{\mu_1 \beta_2 (\beta_5 + \mu_1)},$$

$$S_m^* = \frac{\Lambda_2}{\beta_6 I_{h1}^* + \mu_2}, I_m^* = \frac{\Lambda_2 \beta_6 I_{h1}^*}{\mu_2(\beta_6+\mu_2)}.$$

The endemic equilibria exist if $R_0 > 1$.

4. Stability analysis of the dengue model

4.1. Local stability of E_0 & E_1

Theorem 4.1.1. System (2.1)'s DFE (E_0) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. The Jacobian matrix at the dengue-free equilibrium point (E_0) is

$$J(E_0) = \begin{pmatrix} -(\mu_1+\alpha) & 0 & -\beta_2 S_h^0 & 0 & 0 & 0 & -\beta_1 S_h^0 \\ 0 & -\mu_1 & 0 & 0 & 0 & 0 & \beta_1 S_h^0 \\ 0 & 0 & \beta_2 S_h^0 - (\beta_3+\beta_4+\mu_1+\gamma) & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_3 & -\beta_5-\mu_1 & 0 & 0 & 0 \\ \alpha & 0 & \beta_4+\gamma & \beta_5 & -\mu_1 & 0 & 0 \\ 0 & 0 & -\beta_6 S_m^0 & 0 & 0 & -\mu_2 & 0 \\ 0 & 0 & \beta_6 S_m^0 & 0 & 0 & 0 & -\mu_2 \end{pmatrix}.$$

The eigenvalues for the matrix $J(E_0)$ are $-\mu_1$ (multiplicity 2), $-\mu_2$ (multiplicity 2), $-(\mu_1+\alpha)$, $-(\beta_5+\mu_1)$, and $\beta_2 S_h^0 - (\beta_3+\beta_4+\mu_1+\gamma)$. An obvious negative sign appears in the first six eigenvalues. As this is the case, the DFE E_0 is locally asymptotically stable if

$$\beta_2 S_h^0 - (\beta_3+\beta_4+\mu_1+\gamma) < 0,$$

$$\beta_2 S_h^0 < (\beta_3+\beta_4+\mu_1+\gamma),$$

$$\frac{\beta_2 S_h^0}{(\beta_3+\beta_4+\mu_1+\gamma)} < 1,$$

$$\frac{\beta_2 \Lambda_1}{(\mu_1+\alpha)(\beta_3+\beta_4+\mu_1+\gamma)} < 1,$$

$$R_0 < 1.$$

So, E_0 is locally asymptotically stable if $R_0 < 1$, otherwise; it is unstable.

Theorem 4.1.2. System (2.1)'s E_1 is locally asymptotically stable if $R_0 > 1$.

Proof. The Jacobian matrix at the endemic equilibrium point (E_1) is

$$J(E_1) = \begin{pmatrix} -\varphi_1 & 0 & -\beta_2 S_h^* & 0 & 0 & 0 & -\beta_1 S_h^* \\ \beta_1 I_m^* & -\mu_1 & 0 & 0 & 0 & 0 & \beta_1 S_h^* \\ \beta_2 I_{h1}^* & 0 & -\varphi_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_3 & -\beta_5-\mu_1 & 0 & 0 & 0 \\ 0 & 0 & \beta_4+\gamma & \beta_5 & -\mu_1 & 0 & 0 \\ 0 & 0 & -\varphi_3 & 0 & 0 & -\varphi_4-\mu_2 & 0 \\ 0 & 0 & \varphi_3 & 0 & 0 & \varphi_4 & -\mu_2 \end{pmatrix},$$

where, $\varphi_1 = \beta_1 I_m^* + \beta_2 I_{h1}^* + \alpha + \mu_1$, $\varphi_2 = \beta_3 + \beta_4 + \gamma + \mu_1 - \beta_2 S_h^*$, $\varphi_3 = \beta_6 S_m^*$, $\varphi_4 = \beta_{16} I_{h1}^*$.

Four eigenvalues of the above matrix are $-\mu_1$, $-\mu_1$, $-\mu_2$, $-(\beta_5+\mu_1)$, and the other roots may be found by using the cubic equation

$$\lambda^3 + \epsilon_1 \lambda^2 + \epsilon_2 \lambda + \epsilon_3 = 0, \tag{4.1}$$

where,

$$\epsilon_1 = \varphi_1 + \varphi_2 + \varphi_4 + \mu_2,$$

$$\epsilon_2 = \beta_2^2 I_{h1}^* S_h^* + \varphi_1\mu_2 + \varphi_2\mu_2 + \varphi_1\varphi_2\mu_2 + \varphi_1\varphi_2 + \varphi_1\varphi_4 + \varphi_2\varphi_4,$$

$$\epsilon_3 = \varphi_1\varphi_2\varphi_4 + \mu_2\beta_2^2 I_{h1}^* S_h^* + \varphi_3\beta_1\beta_2 I_{h1}^* S_h^*.$$

Here, $\epsilon_1 > 0,\ \epsilon_3 > 0,\ \epsilon_1\epsilon_2 - \epsilon_3 > 0.$

For this

$$\begin{aligned}\epsilon_1 &= \varphi_1 + \varphi_2 + \varphi_4 + \mu_2\\ &= \beta_1 I_m^* + \beta_2 I_{h1}^* + \alpha + \mu_1 + \beta_3 + \beta_4 + \gamma + \mu_1 - \beta_2 S_h^* + \beta_6 I_{h1}^* + \mu_2\\ &= \frac{\Lambda_2\beta_1\beta_6(\mu_{1+}\alpha)\mu_2(\beta_5+\mu_1)(\beta_6+\mu_2)(R_0-1)}{\mu_2(\beta_6+\mu_2)\beta_1\beta_6\Lambda_2 + \beta_2\mu_2(\beta_6+\mu_2)}\\ &+ \frac{\beta_2(\mu_{1+}\alpha)\mu_2(\beta_5+\mu_1)(\beta_6+\mu_2)(R_0-1)}{\beta_1\beta_6\Lambda_2 + \beta_2\mu_2(\beta_6+\mu_2)}\\ &+ \frac{\beta_6(\mu_{1+}\alpha)\mu_2(\beta_5+\mu_1)(\beta_6+\mu_2)(R_0-1)}{\beta_1\beta_6\Lambda_2 + \beta_2\mu_2(\beta_6+\mu_2)}\\ &+ \mu_1 + \alpha + \gamma + \mu_2 > 0,\end{aligned}$$

if $R_0 > 1$.

Hence, the Routh-Hurwitz criterion $\epsilon_1 > 0$, $\epsilon_3 > 0$, $\epsilon_1\epsilon_2 - \epsilon_3 > 0$ ensues the local asymptotically stability for $R_0 > 1$.

4.2. Global stability of dengue-free equilibrium (E0) & endemic equilibrium (E1)

Lemma 4.2.1. The region $\omega_1 = \{X\epsilon\omega: S_h \le S_h^0, S_m \le S_m^0\}$ is a positively invariant for the model (1), where $X = \{S_h,\ I_{h0},\ I_{h1},\ H_h,\ R_h,\ S_m,\ I_m\}$.

Proof. Using the model's first equation, we obtain

$$\begin{aligned}\frac{dS_h}{dt} &= \Lambda_1 - \beta_1 S_h I_m - \beta_2 S_h I_{h1} - \alpha S_h - \mu_1 S_h \le \Lambda_1 - \alpha S_h - \mu_1 S_h\\ &\le (\mu_1+\alpha)\left(\frac{\Lambda_1}{(\mu_1+\alpha)} - S_h\right) \le (\mu_1+\alpha)\left(S_h^0 - S_h\right).\end{aligned}$$

Implying that, $S_h \le S_h^0 - (S_h^0 - S_h(0))e^{-(\mu_1+\alpha)t}$. Thus $S_h(t) \le S_h^0$ for all $t \ge 0$.

Using the model's fifth equation, we obtain

$$\frac{dS_m}{dt} = \Lambda_2 - \beta_6 S_m I_{h1} - \mu_2 S_m \le \Lambda_2 - \mu_2 S_m \le \mu_2\left(\frac{\Lambda_2}{\mu_2} - S_m\right).$$

Implying that, $S_m \le S_m^0 - (S_m^0 - S_m(0))e^{-\mu_2 t}$. Thus $S_m(t) \le S_m^0$ for all $t \ge 0$.

In summary, we conclude that the ω_1 is positively invariant.

Theorem 4.2.1. The vector-host model may be expressed generally as

$$\frac{d\varkappa_1}{dt} = \mathcal{F}(\varkappa_1,\ \varkappa_2),\ \frac{d\varkappa_2}{dt} = G(\varkappa_1,\ \varkappa_2),\ G(\varkappa_1, 0) = 0. \tag{4.2}$$

Where, $\varkappa_1 = (S_h, R_h, S_m)^t, and\ \varkappa_2 = (I_{h0}, I_h, H_h, I_m)^t$ represent the individuals who are infected and those who are not. Then, the DFE is now represented by the following:

$$E_0 = (\mathcal{H}_1^0, 0) = \left(\frac{\Lambda_1}{(\mu_1+\alpha)}, \frac{\mu_1(\mu_1+\alpha)}{\alpha\Lambda_1}, \frac{\Lambda_2}{\mu_2}, 0,0,0,0\right).$$

The following conditions must be met in order for E_0 to maintain global asymptomatic stability: $H_1: \frac{d\mathcal{H}_1}{dt} = \mathcal{F}(\mathcal{H}_1^0, 0), \mathcal{H}_1^0$ is global asymptomatic stable, H_2: $\hat{G}(\mathcal{H}_1, \mathcal{H}_2) = A\mathcal{H}_2 - G(\mathcal{H}_1, \mathcal{H}_2)$, $\widehat{G}(\mathcal{H}_1, \mathcal{H}_2) \geq 0$, where $A = D_{\mathcal{H}_2}\mathcal{F}(\mathcal{H}_1^0, 0)$ is Metzler matrix.

Theorem 4.2.2. If the criteria of Eq (4.2) are met and $R_0 < 1$, $E_0 = (\mathcal{H}_1^0, 0)$ is a global asymptomatic stable.

Proof. Applying the theorem (4.1.2)

$$\mathcal{F}(\mathcal{H}_1^0, 0) = \Lambda_1 - \alpha S_h - \mu_1 S_h, \hat{G}(\mathcal{H}_1, \mathcal{H}_2) = A\mathcal{H}_2 - G(\mathcal{H}_1, \mathcal{H}_2).$$

Where,

$$A = \begin{bmatrix} -\mu_1 & 0 & 0 & \beta_1 S_h^0 \\ 0 & \beta_2 S_h^0 - \beta_3 + \beta_4 + \gamma + \mu_1 & 0 & 0 \\ 0 & \beta_3 & -(\beta_5 + \mu_1) & 0 \\ 0 & \beta_6 S_m^0 & 0 & -\mu_2 \end{bmatrix}.$$

Now,

$$\hat{G}(\mathcal{H}_1, \mathcal{H}_2) = A\mathcal{H}_2 - G(\mathcal{H}_1, \mathcal{H}_2) = \begin{bmatrix} \beta_1 I_m (S_h^0 - S_h) \\ \beta_2 I_{h1} (S_h^0 - S_h) \\ 0 \\ \beta_6 I_{h1} (S_m^0 - S_m) \end{bmatrix} = \begin{bmatrix} \hat{G}_1 \\ \hat{G}_2 \\ 0 \\ \hat{G}_3 \end{bmatrix}.$$

Clearly, $\hat{G}_1 \geq 0, \hat{G}_2 \geq 0 \; and \; \hat{G}_3 \geq 0$, since $S_h \leq S_h^0, S_m \leq S_m^0$. Therefore, $\hat{G}(\mathcal{H}_1, \mathcal{H}_2) \geq 0$.

Also, A is Metzler matrix. Hence, the dengue-free equilibrium E_0 is global asymptotical stable.

Theorem 4.2.3. System (2.1)'s E_1 is globally asymptotically stable if $R_0 > 1$.

Proof. The form's Lyapunov function in

$$W(t) = \frac{1}{2}(S_h - S_h^*)^2 + \frac{1}{2}(I_{h0} - I_{h0}^*)^2 + \frac{1}{2}(I_{h1} - I_{h1}^*)^2 + \frac{1}{2}(H_h - H_h^*)^2$$

$$+\frac{1}{2}(R_h - R_h^*)^2 + \frac{1}{2}(S_m - S_m^*)^2 + \frac{1}{2}(I_m - I_m^*)^2.$$

Differentiating with respect to time t, we get:

$$\begin{aligned} W'(t) &= (S_h - S_h^*)S_h' + (I_{h0} - I_{h0}^*)I_{h0}' + (I_{h1} - I_{h1}^*)I_{h1}' + (H_h - H_h^*)H_h' \\ &+(R_h - R_h^*)R_h' + (S_m - S_m^*)S_m' + (I_m - I_m^*)I_m' \\ &= (S_h - S_h^*)(\Lambda_1 - \beta_1 S_h I_m - \beta_2 S_h I_{h1} - \alpha S_h - \mu_1 S_h) + (I_{h0} - I_{h0}^*)(\beta_1 S_h I_m - \mu_1 I_{h0}) \\ &+(I_{h1} - I_{h1}^*)(\beta_2 S_h I_{h1} - \beta_3 I_{h1} - \beta_4 I_{h1} - \gamma I_{h1} - \mu_1 I_{h1}) \\ &+(H_h - H_h^*)(\beta_3 I_{h1} - \beta_5 H_h - \mu_1 H_h) \end{aligned}$$

$$+(R_h - R_h^*)(\beta_4 I_{h1} + \beta_5 H_h + \alpha S_h + \gamma I_{h1} - \mu_1 R_h)$$
$$+(S_m - S_m^*)(\Lambda_2 - \beta_6 S_m I_{h1} - \mu_2 S_m) + (I_m - I_m^*)(\beta_6 S_m I_{h1} - \mu_2 I_m).$$

Using the equilibrium conditions $\Lambda_1 = \mu_1 S_h^* + \mu_1 I_{h0}^* + \mu_1 I_{h1}^* + \mu_1 H_h^* + \mu_1 R_h^*$ and $\Lambda_2 = \mu_2 S_m^* + \mu_2 I_m^*$ into the above equation

$$W'(t) = (S_h - S_h^*)(\mu_1 S_h^* + \mu_1 I_{h0}^* + \mu_1 I_{h1}^* + \mu_1 H_h^* + \mu_1 R_h^*)$$
$$-(S_h - S_h^*)(\beta_1 S_h I_m + \beta_2 S_h I_{h1} + \alpha S_h + \mu_1 S_h) + (I_{h0} - I_{h0}^*)(\beta_1 S_h I_m - \mu_1 I_{h0})$$
$$+(I_{h1} - I_{h1}^*)(\beta_2 S_h I_{h1} - \beta_3 I_{h1} - \beta_4 I_{h1} - \gamma I_{h1} - \mu_1 I_{h1})$$
$$+(H_h - H_h^*)(\beta_3 I_{h1} - \beta_5 H_h - \mu_1 H_h)$$
$$+(R_h - R_h^*)(\beta_4 I_{h1} + \beta_5 H_h + \alpha S_h + \gamma I_{h1} - \mu_1 R_h)$$
$$+(S_m - S_m^*)(\mu_2 S_m^* + \mu_2 I_m^* - \beta_6 S_m I_{h1} - \mu_2 S_m) + (I_m - I_m^*)(\beta_6 S_m I_{h1} - \mu_2 I_m)$$
$$= -\mu_1 (S_h - S_h^*)^2 + \mu_1 I_{h0}^* (S_h - S_h^*) + \mu_1 I_{h1}^* (S_h - S_h^*) - \beta_2 S_h I_{h1}^* (S_h - S_h^*)$$
$$+\mu_1 H_h^* (S_h - S_h^*) + \mu_1 R_h^* (S_h - S_h^*) - \beta_1 S_h I_m (S_h - S_h^*) - \alpha S_h (S_h - S_h^*)$$
$$+\beta_1 S_h I_m (I_{h0} - I_{h0}^*) - \mu_1 I_{h0} (I_{h0} - I_{h0}^*) + \beta_2 S_h I_{h1} (I_{h1} - I_{h1}^*) - \gamma I_{h1} (I_{h1} - I_{h1}^*)$$
$$-(\beta_3 + \beta_4) I_{h1} (I_{h1} - I_{h1}^*) - \mu_1 I_{h1} (I_{h1} - I_{h1}^*) + \beta_3 I_{h1} (H_h - H_h^*)$$
$$-\mu_1 H_h (H_h - H_h^*) + \beta_4 I_{h1} (R_h - R_h^*) + \beta_5 H_h (R_h - R_h^*)$$
$$+\alpha S_h (R_h - R_h^*) + \gamma I_{h1} (R_h - R_h^*) - \mu_1 R_h (R_h - R_h^*) - \mu_2 (S_m - S_m^*)^2$$
$$+\mu_2 I_m^* (S_m - S_m^*) - \beta_6 S_m I_{h1} (S_m - S_m^*) + \beta_6 S_m I_{h1} (I_m - I_m^*) - \mu_2 S_m (I_m - I_m^*)$$
$$= -\mu_1 (S_h - S_h^*)^2 - \mu_1 \{I_{h0} (I_{h0} - I_{h0}^*) - I_{h0}^* (S_h - S_h^*)\}$$
$$-\beta_2 S_h \{I_{h1}^* (S_h - S_h^*) - I_{h1} (I_{h1} - I_{h1}^*)\} - \mu_1 \{I_{h1} (I_{h1} - I_{h1}^*) - I_{h1}^* (S_h - S_h^*)\}$$
$$-\mu_1 \{H_h (H_h - H_h^*) - H_h^* (S_h - S_h^*)\} - \alpha S_h (S_h - S_h^* - R_h + R_h^*)$$
$$-\gamma I_{h1} (I_{h1} - I_{h1}^* - R_h + R_h^*) - \beta_1 S_h I_m (S_h - S_h^* - I_{h0} + I_{h0}^*)$$
$$-\beta_3 I_{h1} (I_{h1} - I_{h1}^* - H_h + H_h^*) - \beta_4 I_{h1} (I_{h1} - I_{h1}^* - R_h + R_h^*)$$
$$-\beta_5 H_h (H_h - H_h^* - R_h + R_h^*) - \mu_1 \{R_h (R_h - R_h^*) - R_h^* (S_h - S_h^*)\}$$
$$-\mu_2 (S_m - S_m^*)^2 - \mu_2 \{(I_m - I_m^*) S_m - I_m^* (S_m - S_m^*)\}$$
$$-\beta_6 S_m I_{h1} (S_m - S_m^* - I_m + I_m^*).$$

Now, $W'(t) \leq 0 \ and \ \ W'(t) = 0$ for

$$S_h = S_h^*, I_{h0} = I_{h0}^*, I_{h1} = I_{h1}^*, H_h = H_h^*, R_h = R_h^*, S_m = S_m^*, , I_m = I_m^*.$$

So, the largest invariance set is the singleton set $\{E_1\}$. Therefore, using the principle of LaSalle's invariance, the endemic equilibrium E_1 is globally asymptotically stable.

5. Dengue model's sensitivity analysis

To mitigate and manage dengue outbreaks, a sensitivity analysis is performed to pinpoint the factors that exert the most significant influence on the transmission and dissemination of dengue within the population, as outlined in the model (2.1), employing the same methodology as previous studies [46–48]. The normalized forward sensitivity index of R_0 relates to the system (2.1) parameter ϑ, which is signified by $\zeta_{R_0}^{\vartheta} = \frac{\partial R_0}{\partial \vartheta} . \frac{\vartheta}{R_0}$.

Table 2 and Figure 2 display the dengue fever model's sensitivity index in relation to R_0. When an index has a positive sign, R_0 increases; when it has a negative sign, R_0 declines. If the other parameters are maintained constantly and the negative indices $(\alpha, \mu_1, \gamma, \beta_3, \beta_4)$ are increased, the value of R_0 falls, suggesting a reduction in the rate of disease transmission. Conversely, if all other parameters remain constant and the indices with positive values (Λ_1, β_2) are raised, R_0 will also rise, resulting in a higher rate of disease transmission among humans. Furthermore, the sensitivity index shows that the infectious rate within the host has the highest positive value (+1), suggesting that changes in the host's infectious rate will affect R_0. In addition, the negative sensitivity index of -0.99324 for the rate of immune system of susceptible host by consumption of natural foods $(\boldsymbol{\alpha})$ indicates that a rise in $\boldsymbol{\alpha}$ leads to a reduction in R_0 and vice versa. So, the best control approach to keep diseases under control is to increase the immune system rate of susceptible hosts by eating natural foods.

Figure 3–6 display the R_0 of the proposed system as it varies with different input parameters. Visualizing these parameters' effect on the outcomes of R_0 is the primary goal of this endeavor. Dengue fever in the population decreases as the rate of the immune system in the susceptible host grows due to the consumption of natural foods (α), while the rate of infection within the host (β_2) remains constant (refer to Figure 3). It is indeed possible to take R_0 to a satisfactory level of $R_0 < 1$ by enhancing the immune system of susceptible hosts by consuming natural foods, with an α value of approximately 0.42.

Table 2. Sensitivity measures of R_0.

Parameter	Sensitivity index
Λ_1	+1.0
β_2	+1.0
α	-0.993245927
μ_1	-0.0084993573
γ	-0.3573103612
β_3	-0.2793059925
β_4	-0.3616383535

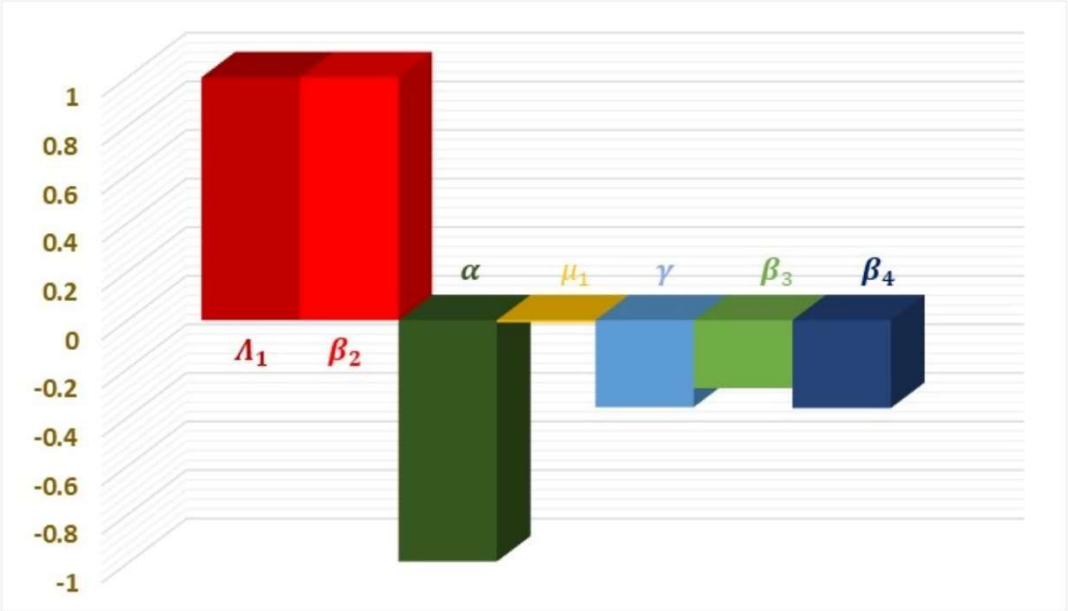

Figure 2. Sensitivity measures of R_0.

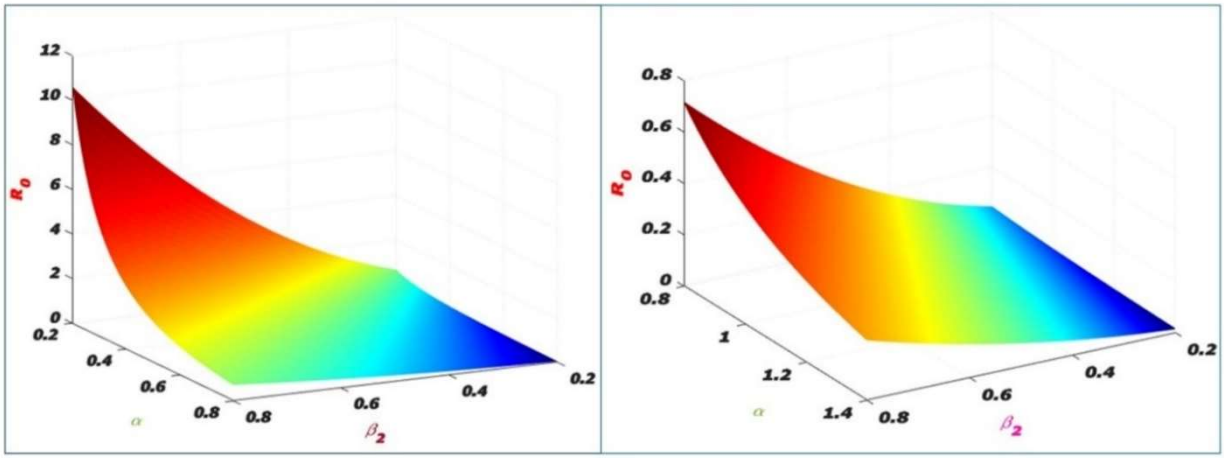

Figure 3. Impact of variability α on R_0.

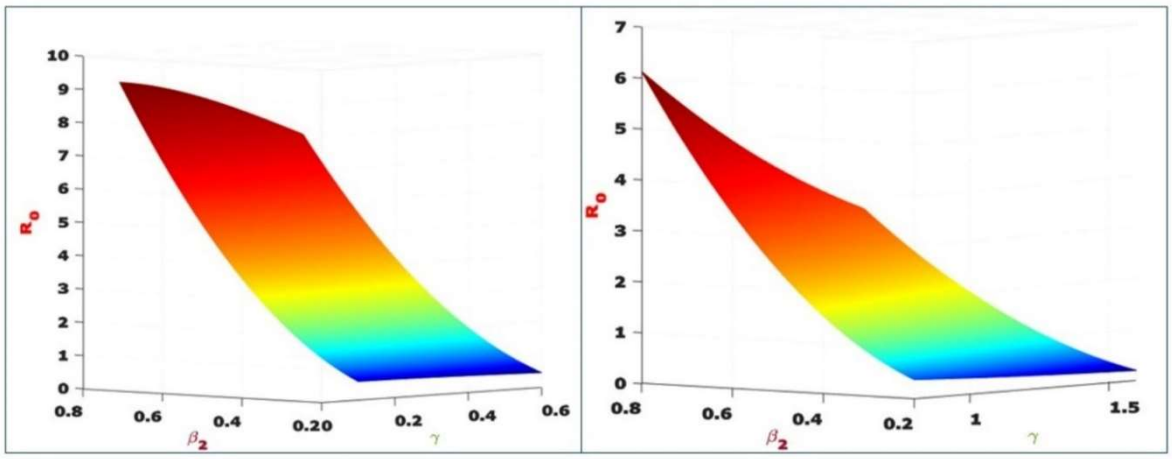

Figure 4. Impact of variability γ on R_0.

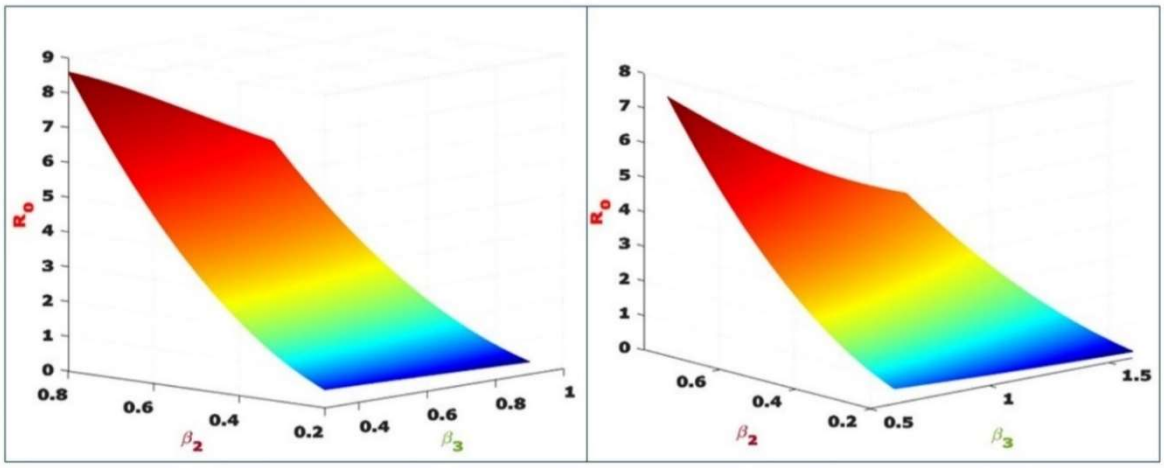

Figure 5. Impact of variability β_3 on R_0.

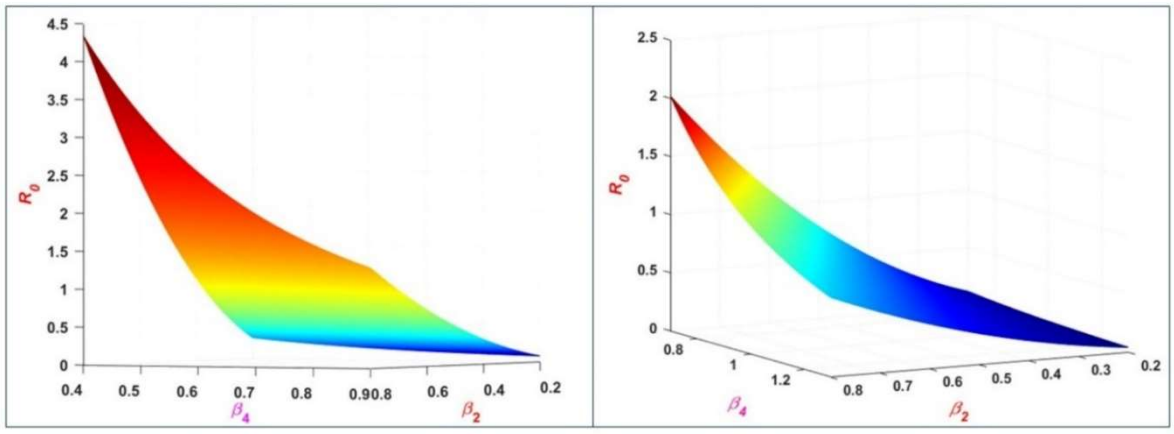

Figure 6. Impact of variability β_4 on R_0.

Figures 4–6 demonstrate that while the infectious disease rate (β_2) stays constant within the host, there is noticeable variation in the awareness rate among symptomatic infected humans (γ), the rate of symptomatic to hospitalized infected (β_3), and symptomatic infection recovery rate (β_4). The results indicate that γ and β_3 have small effects on R_0, but the symptomatic infection recovery rate has a larger impact on R_0 (Figure 6).

6. Numerical simulation

To demonstrate the findings of this study, the model's numerical simulations are performed using the parameter values specified in Table 3. The simulation visually represents the quantities of susceptible, infected, symptomatic infected, and hospitalized infected carriers within the host population, as well as the quantities of susceptible and infected people within the vector population.

Table 3. References and parameters values of system (2.1).

Parameter	Values	Reference
Λ_1	.9999	[49]
β_1	.8500	Assumed
β_2	.6294	[50]
α	.2520	Assumed
μ_1	.003468	[51]
β_3	.555	[52]
β_4	.7186	[53]
γ	.111	[54]
β_5	.0062	Assumed
Λ_2	.00034	[55]
β_6	.009	[56]
μ_2	.000244	Assumed

Figure 7 shows the dynamic behavior of an epidemic model over 150 days, illustrating the impact of fluctuations in the parameter α: the rate of the immune system of susceptible hosts by consumption of natural foods. Figure 7 shows the susceptible human (S_h), symptomatically infected human (I_{h1}), recovered human (R_h), and total infected population $(I_{h0} + I_{h1} + H_h)$ under three conditions: the original α(green), a 10% increase in α(red), and a 10% decrease in α(blue). The susceptible human (S_h) experiences rapid decline and stabilization, with α influencing the initial peak and pace of stabilization (Figure 7(a)). The symptomatically infected human (I_{h1}) shows a dramatic initial surge followed by a steady reduction, with greater peaks and faster falls found with increased α (Figure 7(b)). Increased α leads to a fast increase in the recovered human (R_h) in Figure 7(c), before settling at higher values. In Figure 7(d), the overall infected human follows a similar trend of rapid rise and stabilization, with higher and faster stabilization found with a 10% increase in α levels. Increasing α intensifies and shortens the epidemic, whereas decreasing α leads to a less severe but longer epidemic course.

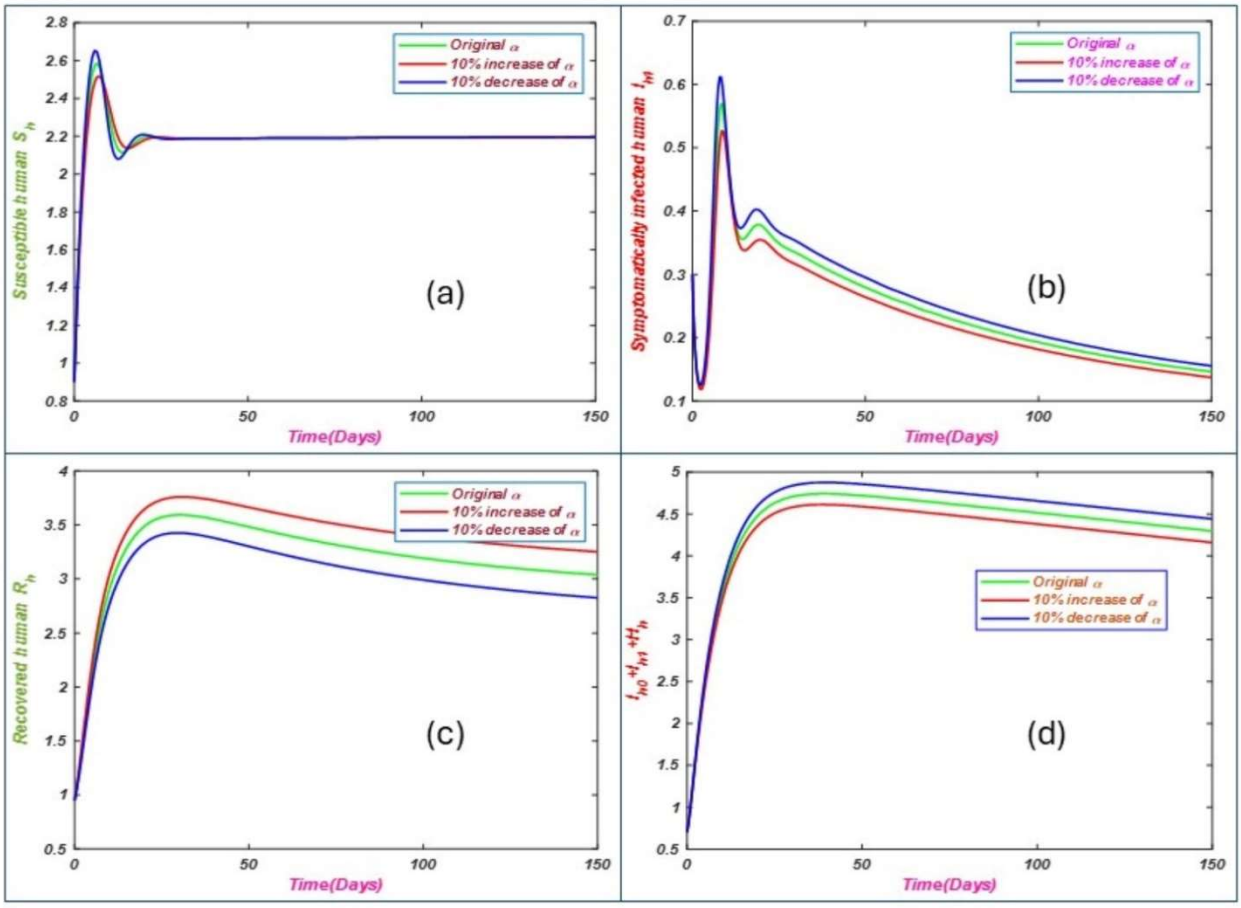

Figure 7. The effects of α.

Figure 8 displays four graphs illustrating the impacts of altering the parameter β_3 on an epidemic model over a span of 150 days. The figure illustrates the fluctuations in the susceptible human population (S_h), hospitalized infected human population (H_h), infected vector population (I_m), and the total population $(S_h + I_{h0} + I_h + H_h)$ across three scenarios: the initial β_3(green), a 10% rise in β_3(red), and a 10% drop in β_3(blue). The population of individuals susceptible to the infection (S_h) initially drops and then reaches a stable state. This is due to a decrease in the transmission rate β_3, which results in a smaller peak in infections and a faster population stability. The population of infected individuals who are hospitalized (H_h) exhibits a notable peak followed by a steady fall, which is caused by a reduced β_3 resulting in a more significant peak and longer duration of hospitalization. The population of infected vectors (I_m), diminishes with time, with a reduced β_3 leading to a more significant initial value and a slower rate of decline. The aggregate population $(S_h + I_{h0} + I_h + H_h)$ experiences a significant increase followed by a period of stability, with negligible influence from changes in β_3 on the overall pattern. Overall, a reduction in β_3 exacerbates and lengthens the epidemic, while an augmentation in β_3 alleviates its severity and duration.

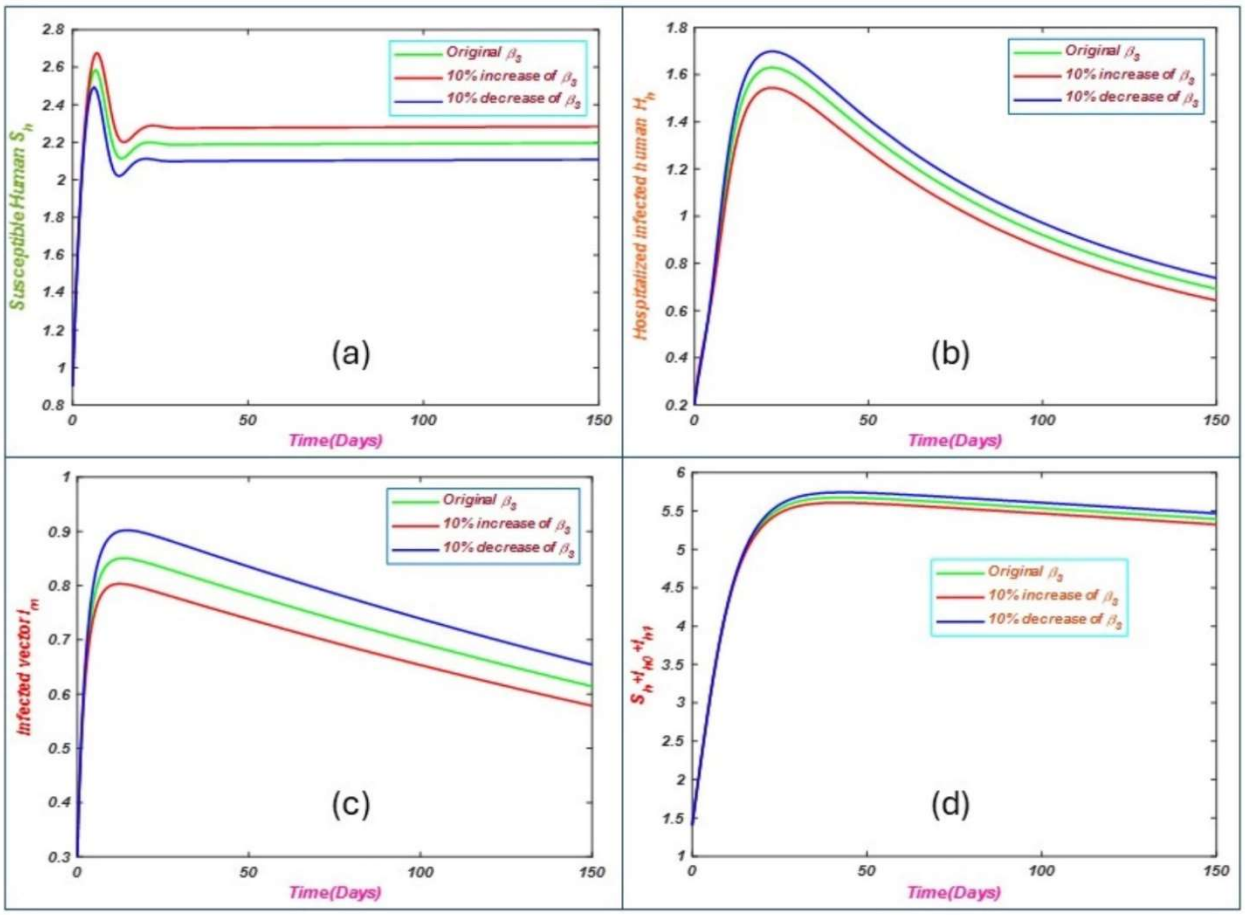

Figure 8. The effects of β_3.

Figure 9 illustrates the impact of changing the parameter β_5 on the number of hospitalized infected and recovered individuals over a period of time, measured in days. The initial β_5(green) of the hospitalized infected exhibits a fast ascent, reaching its maximum around day 40, and subsequently undergoes a slow decrease (Figure 9(a)). An increment of 10% in the value of β_5(red) leads to a decrease in the highest point and a slightly swifter decrease. A reduction of 10% in the value of β_5 (blue) results in an increased peak and a longer duration of hospitalization. In Figure 9(b), the population of the original β_5(green) strain has rapid growth, reaching its highest point around day 50, and then remains stable. A 10% augmentation in the value of β_5(red) leads to a marginally elevated peak and a faster attainment of stability. A reduction of 10% in the value of β_5(blue) results in a marginally diminished highest point and a delayed attainment of stability. In general, raising the value of β_5 has the tendency to decrease the maximum number of patients requiring hospitalization and speed up the process of recovery, leading to a less severe outbreak.

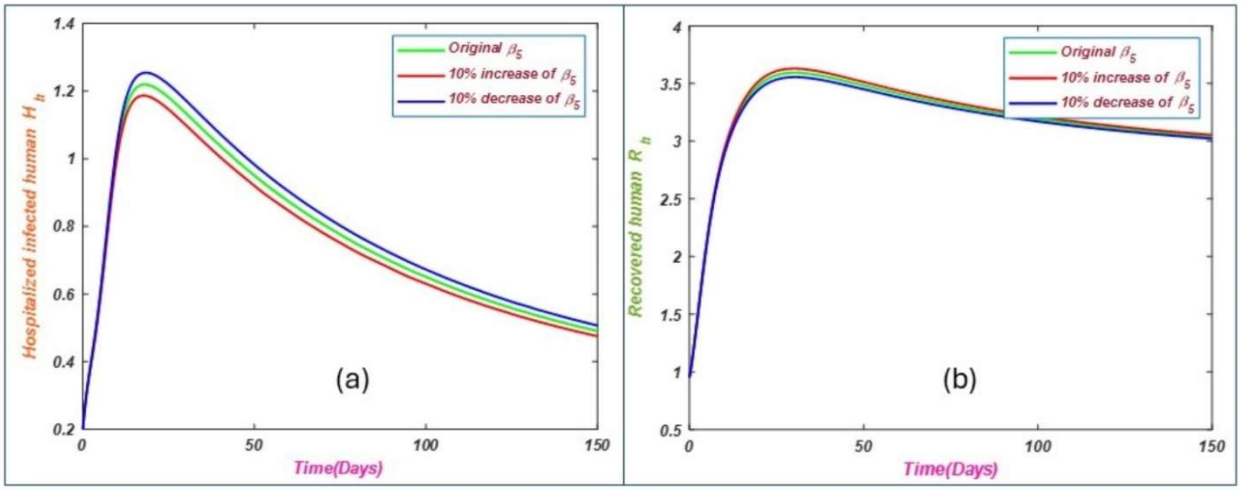

Figure 9. The effects of β_5.

7. Conclusions

This study focuses on developing and examining a nonlinear mathematical model that represents the dynamics of dengue. The model considers different compartments for infected people, individuals with symptoms, and individuals requiring hospitalization. We took a look at the suggested model's fundamental characteristics. The model equilibria were used to produce the control reproduction number, the most essential mathematical quantity with major public health relevance. The results of the model's stability tests were presented. The application of the Jacobian matrix method reveals that the local asymptotic stability of the disease-free steady-state is disrupted. Numerical simulation using several parameter configurations demonstrated the evolution of epidemics, the dynamics of the system, and validated theoretical findings. The numerical simulations illustrate the impact of parameters such as the rate of immune system of susceptible hosts by consuming natural foods, the rate of symptomatic infection to hospitalized infected humans, and the recovery rate of hospitalized infected humans. We also conducted a sensitivity study to evaluate the impact of model parameters on the dynamics and management of the disease. We examined the impact of various parameter rates in order to determine the extent to which this variance influences the epidemic trajectory. The research findings indicate that raising the rate of α has the most significant impact on reducing the reproduction number, with no other parameter having a comparable effect on reducing infection. An investigation of this nature can provide crucial information for policymakers and health experts who may be faced with the harsh reality of infectious diseases.

Author contributions

All authors contributed equal significant contributions to the study's concept, design, methodology, validation, and analysis. All authors have read and approved the final version of the manuscript for publication.

Conflict of interest

The authors declare that the research was conducted in the absence of any conflict of interest.

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