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Research Article

Sensitivity Analysis of Vector-host Dynamic Dengue Epidemic Model

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Abstract. A global health hazard, dengue fever causes or contributes to the deaths of 10,000 people and 100 million cases of symptomatic cases every year in more than half of the globe. The goal of this work is to construct a compartmental vector-borne dengue model that takes into account the typical incidence connection between infected humans and susceptible vectors in order to examine the impact of model parameters that are within our control on the basic reproduction number. In order to determine the basic reproduction number R_0, the next-generation matrix is used. The theoretical study reveals that disease-free equilibrium occurs as a locally asymptotically stable if $R_0 < 1$. To measure the disease-free and endemic equilibrium points' global stability, LaSalle's concept is applied. The normalized forward sensitivity index methods show that the epidemic spread can reduce by increasing the rate of symptomatically infected humans to isolated infected humans and the rate of recovery of symptomatically infected humans.

Keywords. Basic reproduction number (R_0), Dengue fever, Metzler matrix, Numerical simulation, Sensitivity analysis, Stability

Mathematics Subject Classification (2020). 34D20, 92D30, 93D05, 97M60

1. Introduction

Dengue fever is an endemic illness in many tropical nations, particularly in their metropolitan regions (Valencia *et al*. [39]). More than half of the globe is affected by this virus, which places a heavy cost on public health systems everywhere (Shragai *et al*. [35], and Oladipo *et al*. [24]).

Aedes mosquitoes, especially Aedes aegypti, are the primary vectors for the Dengue virus, which is a member of the Flavivirus family and causes dengue fever (Orellano *et al*. [27]). According to the *World Health Organization* (WHO), Aedes mosquitoes have a lifespan of around 10 days and may fly up to 100 meters. The eggs of mosquitoes mature every 5-8 days. After a mosquito bite carries the dengue virus, symptoms might take 3-14 days to manifest (Huy *et al*. [13]). Adults and teenagers are the most afflicted by viral dengue (Huy *et al*. [12]). *Dengue Fever* (DF) symptoms include high fevers, headaches, pains around the eye socket, and muscular problems (Agusto and Khan [2], and Ndii *et al*. [22]). *Dengue Hemorrhagic Fever* (DHF) can cause severe symptoms such as nosebleeds, vomiting blood, and plasma leakage, as well as hypotension, anuria, and shock, which is known as dengue shock syndrome (Zou *et al*. [42]).

Until now, the only effective treatment against dengue virus has been fluid replacement therapy, which can be initiated at an early stage, along with some traditional treatments (Rodrigues *et al*. [32]). Aside from the lack of treatment options for dengue virus-infected persons, there is currently no viable vaccine on the market to vaccinate vulnerable individuals (Nie and Xue [23]). The WHO proposed several vaccine developments for dengue, even though there is no such effective immunization against dengue on the market.

Mathematical modeling has been demonstrated to be an effective method for better understanding specific diseases and formulating treatment approaches (Hasan *et al*. [11], Rawson *et al*. [30], and Sepulveda-Salcedo *et al*. [33]). The formulation of the model, and the capability of a simulation with parameter estimates, allow for sensitivity testing and conjuncture comparisons (Shim [34]). Numerous mathematical models have been utilized and researched to better understand the mechanics of vector-borne diseases (Ullah *et al*. [38]). Abidemi *et al*. [1] introduced and evaluated a compartmentalized mathematical model for a dengue disease transmission model that describes Lyapunov stability analysis. The influence of extreme climates on Dengue Fever infection was investigated, and improved planning of *Dengue Fever* management strategies in response to climate change was advised by Wang *et al*. [40]. Li-Martín *et al*. [17] studied the dengue dynamics transmission model with a two-stage structure in humans as an age risk factor. The co-dynamics mathematical model of COVID-19 and malaria were examined and evaluated its optimal control by Omame *et al*. [26]. In their study of the optimal vaccine method for dengue epidemics in Kupang City, Indonesia, Ndii *et al*. [21] used a global sensitivity analysis. Claypool *et al*. [7] conducted research on the cost-effectiveness of dengue and chikungunya control in Colombia.

Pandey *et al*. [28] used Bayesian Markov chain Monte Carlo estimate to investigate vector-host and SIR models. An analysis of the internal dengue epidemic model using fractional piecewise derivatives was done numerically by Ahmad *et al*. [3]. Ndii [20] implemented an effective media campaign dengue dynamic model, which influences the reduction of dengue illnesses by raising individual awareness. Hasan *et al*. [10] analyzed a vector-host SEIR-SEI Dengue epidemiological model that took panic, tension, or anxiety into consideration. Tay *et al*. [37] developed an SI-SIR dengue epidemiological characteristics model for dengue control in Malaysia. An analysis of a dynamic transmission model was carried out by Knerer *et al*. [15] in Thailand to determine the economic benefits and costs of combining vector-control and dengue vaccine techniques.

The research article is systematized as follows. In Section 2, the formulation of the model is presented. The qualitative analysis of the model is given in Section 3 and stability of the equilibrium points is in Section 4. In Section 5, the sensitivity analysis is performed. Section 6 contains the model's numerical simulation done to validate the theoretical analysis shown in Sections 3, 4 and 5. Lastly, the conclusion is in Section 7.

2. Formulation of the Dengue Model

The compartmental epidemic models explain how the epidemic spreads and the numerous preventative actions that may be implemented to stop it (Srivastav *et al*. [36]). On the basis of the study, we presented a Dengue vector-host mathematical model. The total human (host) population categorizes into five groups: susceptible human, S_h (individuals who can contract the disease), infected human, I_{h0} (infected individuals who are not capable to transmit to others), symptomatically infected human, I_{h1} (individuals who are able to transmit to others), isolated infected human, I_{h2} (individuals who tested positive and isolated from others), and recovered human, R_h (individuals who acquired immunity). Thus, the total host population

$$N_h = S_h + I_{h0} + I_{h1} + I_{h2} + R_h\,.$$

Also, the vector(mosquito) population categorizes into two groups: susceptible vector (S_m), and infected vector (I_m). Therefore, the total vector population

$$N_m = S_m + I_m\,.$$

The dynamics Dengue virus may be depicted by the following non-linear system of differential equations based on the assumptions and flow diagram Figure 1

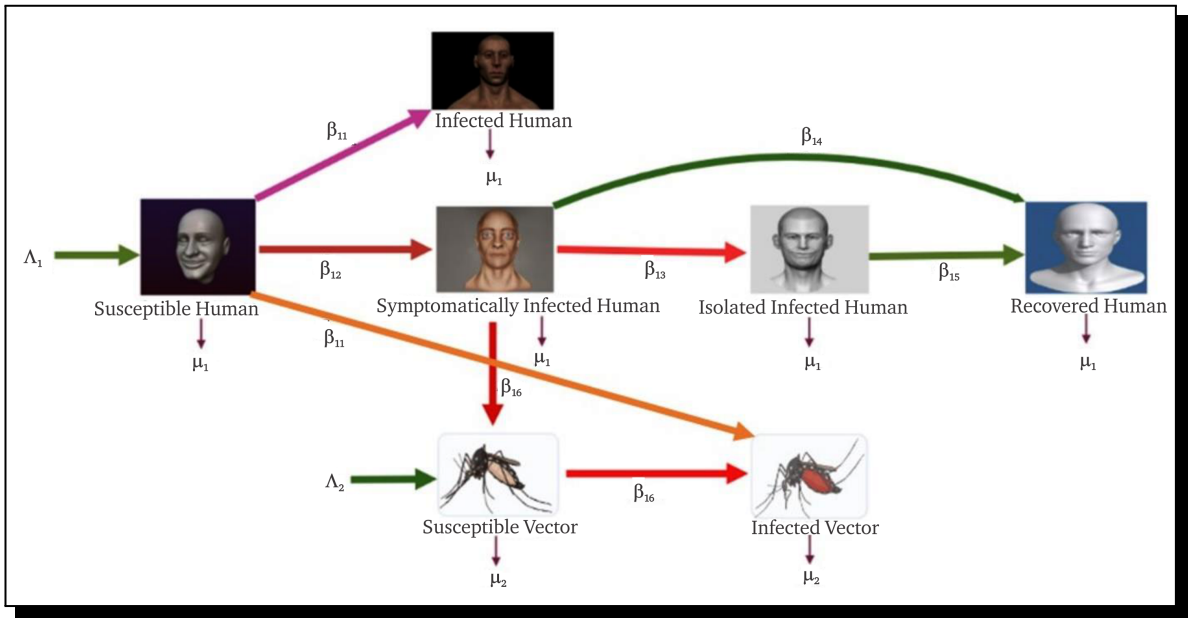

Figure 1. Dengue virus transmission dynamics in different population stages

Human population (h)

$$\frac{dS_h}{dt} = \Lambda_1 - \beta_{11} S_h I_m - \beta_{12} S_h I_{h1} - \mu_1 S_h\,,$$

$$\frac{dI_{h0}}{dt} = \beta_{11} S_h I_m - \mu_1 I_{h0},$$
$$\frac{dI_{h1}}{dt} = \beta_{12} S_h I_{h1} - \beta_{13} I_{h1} - \beta_{14} I_{h1} - \mu_1 I_{h1},$$
$$\frac{dI_{h2}}{dt} = \beta_{13} I_{h1} - \beta_{15} I_{h2} - \mu_1 I_{h2},$$
$$\frac{dR_h}{dt} = \beta_{14} I_{h1} + \beta_{15} I_{h2} - \mu_1 R_h.$$

Vector population (m)

$$\frac{dS_m}{dt} = \Lambda_2 - \beta_{16} S_m I_{h1} - \mu_2 S_m,$$
$$\frac{dI_m}{dt} = \beta_{16} S_m I_{h1} - \mu_2 I_m. \tag{2.1}$$

With initial condition

$S_h(0) \geq 0,\ I_{h0}(0) \geq 0,\ I_{h1}(0) \geq 0,\ I_{h2}(0) \geq 0,\ R_h(0) \geq 0,\ S_m(0) \geq 0$ and $I_m(0) \geq 0$.

The biological description of the parameters is itemized in Table 1.

Table 1. Values for baseline parameters with definitions and biological descriptions of Dengue model

Parameter	Biological descriptions
Λ_1	Recruitment rates of human population
β_{11}	Rate of infectious from vector to host
β_{12}	Rate of infectious with in host
μ_1	Natural death rate of human population
β_{13}	Rate of symptomatically infected to isolated infected humans
β_{14}	Recovery rate of symptomatically infected humans
β_{15}	Recovery rate of isolated infected human
Λ_2	Recruitment rates of vector population
β_{16}	Infection rate from human to vector
μ_2	Natural death rate of vector population

3. Qualitative Analyses of Model

3.1 Positivity and Boundedness of Solutions

An epidemiological model must have solutions that are both non-negative and bounded. As a result, it is crucial to show that all variables are positive at all times $t > 0$.

Theorem 3.1. *The feasible region defined by*

$$\tau = \left\{ S_h(t), I_{h0}(t), I_{h1}(t), I_{h2}(t), R_h(t), S_m(t), I_m(t) \in \mathbb{R}_7^+ : N_h(t) \leq \frac{\Lambda_1}{\mu_1}, N_m(t) \leq \frac{\Lambda_2}{\mu_2} \right\}$$

is positively invariant for the system (2.1) *with the initial condition defined by* $\mathbb{R}_7^+$.

Proof. The system (2.1) can be written as

$$\frac{dY}{dt} = KY + Z, \tag{3.1}$$

$$Y = (S_h, I_{h0}, I_{h1}, I_{h2}, R_h, S_m, I_m)^t,$$

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

where

$$k_1 = \beta_{11} I_m + \beta_{12} I_{h1} + \mu_1,\ k_2 = \beta_{12} S_h,\ k_3 = \beta_{13} I_{h1},\ k_4 = \beta_{13} + \beta_{14} + \mu_1,\ k_5 = \beta_{13} I_{h1},$$

$$k_6 = \beta_{15} + \mu_1,\ k_7 = \beta_{14},\ k_8 = \beta_{15},\ k_9 = \beta_{16} I_{h1} + \mu_2,\ k_{10} = \beta_{14} I_{h1}$$

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

Here, all the off-diagonal entries of the matrix $K(Y)$ are non-negative. Hence, the matrix is the Metzler matrix (Maiga and Hugo [18]). Also, the vector Z has positive in nature. Therefore, it implies that system (3.1) is positively invariant in $\mathbb{R}^+_7$, which means that any trajectory of (3.1) starting from an initial state remains in $\mathbb{R}^+_7$ forever. □

3.2 Disease-Free Equilibrium

The *Disease-Free Equilibrium* (DFE) of the system (2.1) is produced by setting each system of model system (2.1) to zero. Furthermore, there are no infections or recovery at the DFE. Thus, the Dengue model's DFE (2.1) is provided by

$$E_0 = (S_h^0, I_{h0}^0, I_{h1}^0, I_{h2}^0, R_h^0, S_m^0, I_m^0) = \left(\frac{\Lambda_1}{\mu_1}, 0, 0, 0, 0, \frac{\Lambda_2}{\mu_2}, 0\right).$$

3.3 Basic Reproduction Number

In the research of mathematical epidemiology, the basic reproduction number is a crucial threshold (Zheng and Nie [41]). It aids in forecasting the disease transmission potential. To determine R_0 of the system (2.1) with help of the next-generation matrix, the following result

$$F = \begin{pmatrix} 0 & 0 & \beta_{11} S_h \\ 0 & \beta_{12} S_h & 0 \\ 0 & \beta_{16} & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \mu_1 & 0 & 0 \\ 0 & \beta_{13} + \beta_{14} + \mu_1 & 0 \\ 0 & 0 & \mu_2 \end{pmatrix}.$$

The Dengue model's basic reproduction number is the dominating eigenvalue of the next-generation matrix FV^{-1} generated by

$$R_0 = \frac{\beta_{12} \Lambda_1}{\mu_1 (\beta_{13} + \beta_{14} + \mu_1)}.$$

3.4 Endemic Equilibria

The endemic equilibria point for the Dengue dynamical system (2.1)

$$E_1 = (S_h^*, I_{h0}^*, I_{h1}^*, I_{h2}^*, R_h^*, S_m^*, I_m^*),$$

where

$$S_h^* = \frac{\beta_{13}+\beta_{14}+\mu_1}{\beta_{12}},$$
$$I_{h0}^* = \frac{\beta_{11}\beta_{16}\Lambda_2(\beta_{13}+\beta_{14}+\mu_1)I_{h1}^*}{\mu_1\mu_2(\beta_{16}+\mu_2)},$$
$$I_{h1}^* = \frac{\mu_1\mu_2(\beta_{15}+\mu_1)(\beta_{16}+\mu_2)(R_0-1)}{\beta_{11}\beta_{16}\Lambda_2(\beta_{15}+\mu_1)+\beta_{11}\beta_{16}\mu_2(\beta_{16}+\mu_2)},$$
$$I_{h2}^* = \frac{\beta_{13}I_{h1}^*}{\beta_{15}+\mu_1},$$
$$R_h^* = \frac{(\beta_{13}\beta_{14}+\beta_{14}\beta_{15}+\beta_{14}\mu_1)I_{h1}^*}{\mu_1(\beta_{15}+\mu_1)},$$
$$S_m^* = \frac{\Lambda_2}{\beta_{16}I_{h1}^*+\mu_2},$$
$$I_m^* = \frac{\Lambda_2\beta_{16}I_{h1}^*}{\mu_2(\beta_{16}+\mu_2)}.$$

The endemic equilibria exist if $R_0 > 1$.

4. Stability Analysis

4.1 Local Stability Around Equilibrium Point

Theorem 4.1. *For $R_0 < 1$ the disease-free equilibrium (E_0) of the system (2.1) is locally asymptotically stable and unstable if $R_0 > 1$.*

Proof. The Jacobian matrix of the system (2.1) at the disease-free equilibrium point (E_0) is

$$J(E_0) = \begin{pmatrix} -\mu_1 & 0 & -\beta_{12}S_h^0 & 0 & 0 & 0 & -\beta_{11}S_h^0 \\ 0 & -\mu_1 & 0 & 0 & 0 & 0 & \beta_{11}S_h^0 \\ 0 & 0 & \beta_{12}S_h^0-(\beta_{13}+\beta_{14}+\mu_1) & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_{13} & -\beta_{15}-\mu_1 & 0 & 0 & 0 \\ 0 & 0 & \beta_{14} & \beta_{15} & -\mu_1 & 0 & 0 \\ 0 & 0 & -\beta_{16}S_m^0 & 0 & 0 & -\mu_2 & 0 \\ 0 & 0 & \beta_{16}S_m^0 & 0 & 0 & 0 & -\mu_2 \end{pmatrix}.$$

The eigenvalues for the matrix $J(E_0)$ are $-\mu_1$ (multiplicity 3), $-\mu_2$ (multiplicity 2), $-(\beta_{15}+\mu_1)$, and $\beta_{12}S_h^0-(\beta_{13}+\beta_{14}+\mu_1)$. Clearly, first six eigenvalues are negative. Therefore, the DFE E_0 is locally asymptotically stable if

$$\beta_{12}S_h^0-(\beta_{13}+\beta_{14}+\mu_1)<0,$$
$$\beta_{12}S_h^0<(\beta_{13}+\beta_{14}+\mu_1),$$
$$\frac{\beta_{12}S_h^0}{(\beta_{13}+\beta_{14}+\mu_1)}<1,$$
$$\frac{\beta_{12}\Lambda_1}{\mu_1(\beta_{13}+\beta_{14}+\mu_1)}<1,$$
$$R_0<1.$$

Hence, the DFE E_0 is locally asymptotically stable if $R_0 < 1$, otherwise, it is unstable. □

Theorem 4.2. *The endemic equilibrium* (E_1) *of the system* (2.1) *is locally asymptotically stable if* $R_0 > 1$.

Proof. The Jacobian matrix of the system (2.1) at the endemic equilibrium point (E_1) is

$$J(E_1) = \begin{pmatrix} -a & 0 & -\beta_{12}S_h^* & 0 & 0 & 0 & -\beta_{11}S_h^* \\ \beta_{11}I_m^* & -\mu_1 & 0 & 0 & 0 & 0 & \beta_{11}S_h^* \\ \beta_{12}I_{h1}^* & 0 & -b & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_{13} & -\beta_{15}-\mu_1 & 0 & 0 & 0 \\ 0 & 0 & \beta_{14} & \beta_{15} & -\mu_1 & 0 & 0 \\ 0 & 0 & -c & 0 & 0 & -d-\mu_2 & 0 \\ 0 & 0 & c & 0 & 0 & d & -\mu_2 \end{pmatrix},$$

where

$$a = \beta_{11}I_m^* + \beta_{12}I_{h1}^* + \mu_1,\ b = \beta_{13} + \beta_{14} + \mu_1 + \beta_{12}S_h^*,\ c = \beta_{16}S_m^*,\ d = \beta_{16}I_{h1}^*.$$

Three eigenvalues of the above matrix are $-\mu_1$, $-\mu_1$, $-(\beta_{15}+\mu_1)$, and the following biquadratic equation will give the rest of the roots

$$\lambda^4 + \epsilon_1\lambda^3 + \epsilon_2\lambda^2 + \epsilon_3\lambda + \epsilon_4 = 0, \tag{4.1}$$

where

$$\epsilon_1 = a + b + d + 2\mu_2, \epsilon_2 = \mu_2^2 + 2a\mu_2 + 2b\mu_2 + d\mu_2 + ab + ad + bd,$$
$$\epsilon_3 = a\mu_2^2 + b\mu_2^2 + 2ab\mu_2 + ad\mu_2 + bd\mu_2 + abd + c\beta_{12}^2 I_{h1}^* S_h^* + c\beta_{11}\beta_{12} I_{h1}^* S_h^*,$$
$$\epsilon_4 = ab\mu_2^2 + abd\mu_2 + c\mu_2\beta_{11}\beta_{12}I_{h1}^*S_h^* + c\mu_2\beta_{12}^2 I_{h1}^* S_h^*.$$

Here $\epsilon_1 > 0$, $\epsilon_1\epsilon_2 - \epsilon_3 > 0$, $\epsilon_1\epsilon_2\epsilon_3 - \epsilon_3^2 - \epsilon_4\epsilon_1^2 > 0$.

For this

$$\begin{aligned} \epsilon_1 &= a + b + d + 2\mu_2 \\ &= \beta_{11}I_m^* + \beta_{12}I_{h1}^* + \mu_1 + \beta_{13} + \beta_{14} + \mu_1 + \beta_{12}S_h^* + \beta_{16}I_{h1}^* + 2\mu_2 \\ &= \beta_{11}I_m^* + \beta_{12}\frac{\mu_1\mu_2(\beta_{15}+\mu_1)(\beta_{16}+\mu_2)(R_0-1)}{\beta_{11}\beta_{16}\Lambda_2(\beta_{15}+\mu_1) + \beta_{11}\beta_{16}\mu_2(\beta_{16}+\mu_2)} + 2\mu_1 + \beta_{13} \\ &\quad + \beta_{14} + \beta_{12}S_h^* + \beta_{16}\frac{\mu_1\mu_2(\beta_{15}+\mu_1)(\beta_{16}+\mu_2)(R_0-1)}{\beta_{11}\beta_{16}\Lambda_2(\beta_{15}+\mu_1) + \beta_{11}\beta_{16}\mu_2(\beta_{16}+\mu_2)} + 2\mu_2 > 0, \quad \text{if } R_0 > 1. \end{aligned}$$

Therefore, Routh-Hurwitz criterion satisfied, and the system (2.1) is locally asymptotically stable for $R_0 > 1$. □

4.2 Global Stability Around Equilibrium Point

In this segment, we will evaluate equilibrium points E_0 and E_1 stability. The next two theorems show the results of the stability analysis of these equilibrium sites.

Theorem 4.3. *If* $R_0 < 1$, *the disease-free equilibrium* (E_0) *is globally asymptotically stable on* τ *with assumption*

$$\beta_{12}S_h^0 + \beta_{16}S_m^0 = \mu_1, \tag{4.2}$$
$$\beta_{11}S_h^0 = \mu_2. \tag{4.3}$$

Proof. We consider the Lyapunov function of the form in

$$G(t) = (S_h - S_h^0\, lnS_h) + I_{h0} + I_{h1} + I_{h2} + R_h + (S_m - S_m^0 \ln S_m) + I_m.$$

Differentiating with respect to t, we get

$$\begin{aligned} G'(t) &= \left(1 - \frac{S_h^0}{S_h}\right) S_h' + I_{h0}' + I_{h1}' + I_{h2}' + R_h' + \left(1 - \frac{S_m^0}{S_m}\right) S_m' + I_m' \\ &= \left(1 - \frac{S_h^0}{S_h}\right)(\Lambda_1 - \beta_{11} S_h I_m - \beta_{12} S_h I_{h1} - \mu_1 S_h) + \beta_{11} S_h I_m - \mu_1 I_{h0} \\ &\quad + \beta_{12} S_h I_{h1} - \beta_{13} I_{h1} - \beta_{14} I_{h1} - \mu_1 I_{h1} + \beta_{13} I_{h1} - \beta_{15} I_{h2} - \mu_1 I_{h2} + \beta_{14} I_{h1} \\ &\quad + \beta_{15} I_{h2} - \mu_1 R_h + \left(1 - \frac{S_m^0}{S_m}\right)(\Lambda_2 - \beta_{16} S_m I_{h1} - \mu_2 S_m) + \beta_{16} S_m I_{h1} - \mu_2 I_m. \end{aligned}$$

On solving further get:

$$\begin{aligned} G'(t) &= \left(1 - \frac{S_h^0}{S_h}\right) \Lambda_1 - \mu_1 S_h + \beta_{11} S_h^0 I_m + \beta_{12} S_h^0 I_{h1} - \mu_1 S_h^0 - \mu_1 I_{h0} - \mu_1 I_{h1} \\ &\quad - \mu_1 I_{h2} - \mu_1 R_h + \left(1 - \frac{S_m^0}{S_m}\right) \Lambda_2 - \mu_2 S_m + \beta_{16} S_m^0 I_{h1} + \mu_2 S_m^0 - \mu_2 I_m. \end{aligned}$$

Using the equilibrium condition $\mu_1 S_h^0 = \Lambda_1$ and $\mu_2 S_m^0 = \Lambda_2$ into the above equation

$$\begin{aligned} G'(t) &= \left(2 - \frac{S_h^0}{S_h} - \frac{S_h}{S_h^0}\right) \Lambda_1 + \left(2 - \frac{S_m^0}{S_m} - \frac{S_m}{S_m^0}\right) \Lambda_2 + \beta_{11} S_h^0 I_m + \beta_{12} S_h^0 I_{h1} \\ &\quad + \beta_{16} S_m^0 I_{h1} - \mu_1 I_{h0} - \mu_1 I_{h1} - \mu_1 I_{h2} - \mu_1 R_h - \mu_2 I_m \\ &= -\Lambda_1 \frac{(S_h - S_h^0)^2}{S_h S_h^0} - \Lambda_2 \frac{(S_m - S_m^0)^2}{S_m S_m^0} - \mu_1 I_{h0} - \mu_1 I_{h2} - \mu_1 R_h \\ &\quad + (\beta_{12} S_h^0 + \beta_{16} S_m^0 - \mu_1) I_{h1} + (\beta_{11} S_h^0 - \mu_2) I_m. \end{aligned}$$

The condition (4.2) and (4.3) ensure that $G'(t) \le 0$ and $G'(t) = 0$ for $S_h = S_h^0$, $I_{h0} = 0$, $I_{h2} = 0$, $R_h = 0$, $S_m = S_m^0$. So, the largest invariance set is the singleton set $\{E_0\}$. Therefore, by using the principle of LaSalle's invariance the disease-free equilibrium (E_0) is globally asymptotically stable. □

Theorem 4.4. *If $R_0 > 1$, the endemic equilibrium (E_1) is globally asymptotically stable.*

Proof. We consider the Lyapunov function of the form in

$$\begin{aligned} W(t) &= \frac{1}{2}(S_h - S_h^*)^2 + \frac{1}{2}(I_{h0} - I_{h0}^*)^2 + \frac{1}{2}(I_{h1} - I_{h1}^*)^2 + \frac{1}{2}(I_{h2} - I_{h2}^*)^2 + \frac{1}{2}(R_h - R_h^*)^2 \\ &\quad + \frac{1}{2}(S_m - S_m^*)^2 + \frac{1}{2}(I_m - I_m^*)^2. \end{aligned}$$

Differentiating with respect to time t, we get

$$\begin{aligned} W'(t) &= (S_h - S_h^*) S_h' + (I_{h0} - I_{h0}^*) I_{h0}' + (I_{h1} - I_{h1}^*) I_{h1}' + (I_{h2} - I_{h2}^*) I_{h2}' + (R_h - R_h^*) R_h' \\ &\quad + (S_m - S_m^*) S_m' + (I_m - I_m^*) I_m' \\ &= (S_h - S_h^*)(\Lambda_1 - \beta_{11} S_h I_m - \beta_{12} S_h I_{h1} - \mu_1 S_h) + (I_{h0} - I_{h0}^*) \end{aligned}$$

$$
\begin{aligned}
&(\beta_{11}S_hI_m-\mu_1I_{h0})+(I_{h1}-I_{h1}^*)(\beta_{12}S_hI_{h1}-\beta_{13}I_{h1}-\beta_{14}I_{h1}-\mu_1I_{h1})\\
&+(I_{h2}-I_{h2}^*)(\beta_{13}I_{h1}-\beta_{15}I_{h2}-\mu_1I_{h2})+(R_h-R_h^*)(\beta_{14}I_{h1}+\beta_{15}I_{h2}-\mu_1R_h)\\
&+(S_m-S_m^*)(\Lambda_2-\beta_{16}S_mI_{h1}-\mu_2S_m)+(I_m-I_m^*)(\beta_5E_m-\mu_2I_m).
\end{aligned}
$$

Using the equilibrium conditions

$$\Lambda_1=\mu_1S_h^*+\mu_1I_{h0}^*+\mu_1I_{h1}^*+\mu_1I_{h2}^*+\mu_1R_h^*$$

and

$$\Lambda_2=\mu_2S_m^*+\mu_2I_m^*$$

into the above equation

$$
\begin{aligned}
W'(t)&=(S_h-S_h^*)(\mu_1S_h^*+\mu_1I_{h0}^*+\mu_1I_{h1}^*+\mu_1I_{h2}^*+\mu_1R_h^*-\beta_{11}S_hI_m-\beta_{12}S_hI_{h1}-\mu_1S_h)\\
&\quad+(I_{h0}-I_{h0}^*)(\beta_{11}S_hI_m-\mu_1I_{h0})\\
&\quad+(I_{h1}-I_{h1}^*)(\beta_{12}S_hI_{h1}-\beta_{13}I_{h1}-\beta_{14}I_{h1}-\mu_1I_{h1})\\
&\quad+(I_{h2}-I_{h2}^*)(\beta_{13}I_{h1}-\beta_{15}I_{h2}-\mu_1I_{h2})+(R_h-R_h^*)(\beta_{14}I_{h1}+\beta_{15}I_{h2}-\mu_1R_h)\\
&\quad+(S_m-S_m^*)(\mu_2S_m^*+\mu_2I_m^*-\beta_{16}S_mI_{h1}-\mu_2S_m)+(I_m-I_m^*)(\beta_{16}S_mI_{h1}-\mu_2I_m)\\
&=-\mu_1(S_h-S_h^*)^2+\mu_1I_{h0}^*(S_h-S_h^*)+\mu_1I_{h1}^*(S_h-S_h^*)-\beta_{12}S_hI_{h1}^*(S_h-S_h^*)\\
&\quad+\mu_1I_{h2}^*(S_h-S_h^*)+\mu_1R_h^*(S_h-S_h^*)-\beta_{11}S_hI_m(S_h-S_h^*)+\beta_{11}S_hI_m(I_{h0}-I_{h0}^*)\\
&\quad-\mu_1I_{h0}(I_{h0}-I_{h0}^*)+\beta_{12}S_hI_{h1}(I_{h1}-I_{h1}^*)-(\beta_{13}+\beta_{14})I_{h1}(I_{h1}-I_{h1}^*)\\
&\quad-\mu_1I_{h1}(I_{h1}-I_{h1}^*)+\beta_{13}I_{h1}(I_{h2}-I_{h2}^*)-\beta_{15}I_{h2}(I_{h2}-I_{h2}^*)-\mu_1I_{h2}(I_{h2}-I_{h2}^*)\\
&\quad+\beta_{14}I_{h1}(R_h-R_h^*)+\beta_{15}I_{h2}(R_h-R_h^*)-\mu_1R_h(R_h-R_h^*)-\mu_2(S_m-S_m^*)^2\\
&\quad+\mu_2I_m^*(S_m-S_m^*)-\beta_{16}S_mI_{h1}(S_m-S_m^*)+\beta_{16}S_mI_{h1}(I_m-I_m^*)-\mu_2S_m(I_m-I_m^*)\\
&=-\mu_1(S_h-S_h^*)^2-\mu_1\{I_{h0}(I_{h0}-I_{h0}^*)-I_{h0}^*(S_h-S_h^*)\}\\
&\quad-\beta_{12}S_h\{I_{h1}^*(S_h-S_h^*)-I_{h1}(I_{h1}-I_{h1}^*)\}-\mu_1\{I_{h1}(I_{h1}-I_{h1}^*)-I_{h1}^*(S_h-S_h^*)\}\\
&\quad-\mu_1\{I_{h2}(I_{h2}-I_{h2}^*)-I_{h2}^*(S_h-S_h^*)\}-\beta_{11}S_hI_m(S_h-S_h^*-I_{h0}+I_{h0}^*)\\
&\quad-\beta_{13}I_{h1}(I_{h1}-I_{h1}^*-I_{h2}+I_{h2}^*)-\beta_{14}I_{h1}(I_{h1}-I_{h1}^*-R_h+R_h^*)\\
&\quad-\beta_{15}I_{h2}(I_{h2}-I_{h2}^*-R_h+R_h^*)-\mu_1\{R_h(R_h-R_h^*)-R_h^*(S_h-S_h^*)\}\\
&\quad-\mu_2(S_m-S_m^*)^2-\mu_2\{(I_m-I_m^*)S_m-I_m^*(S_m-S_m^*)\}\\
&\quad-\beta_{16}S_mI_{h1}(S_m-S_m^*-I_m+I_m^*).
\end{aligned}
$$

The above equation shows that $W'(t)\le 0$ and $W'(t)=0$ for $S_h=S_h^0$, $I_{h0}=0$, $I_{h1}=0$, $I_{h2}=0$, $R_h=0$, $S_m=S_m^0$, $I_m=0$. So, the largest invariance set is the singleton set $\{E_1\}$. Therefore, by using the principle of LaSalle's invariance the endemic equilibrium E_1 is globally asymptotically stable. □

5. Sensitivity Analysis

Sensitivity analysis reveals the significance of each parameter on the transmission of disease (Chien and Yu [6]). A complicated nonlinear model's data minimization and assimilation both depend on this knowledge, which is also essential for experimental design. Due to

frequent mistakes in the collection of data and presumption parameter values, sensitivity analysis can be frequently used to assess how robust the model predictions of the parameter values (Lee *et al*. [16]). It serves to identify parameters with a large influence on R_0 that should be addressed by intervention techniques.

This study will be conducted using the normalized forward sensitivity index of a variable with regard to a parameter, which is calculated as the ratio of the relative variation in the variable to the relative variation in the parameter. Partial derivatives can be used to define the sensitivity index. The normalized forward sensitivity index of the basic reproduction number R_0 regarding the system (2.1) parameter φ, which is signified by $\Gamma_{R_0}^{\varphi} = \frac{\partial R_0}{\partial \varphi} \cdot \frac{\varphi}{R_0}$.

Table 2. Sensitivity indices of R_0 evaluated at the baseline parameter values of the model

Parameter	Sensitivity index
Λ_1	+1.0
β_{12}	+1.0
μ_1	−1.002715595
β_{13}	−0.434589236
β_{14}	−0.5626951736

The sensitivity indices of R_0, Table 2 and Figure 2 indicate that the recruitment rates of human population and rate of infectious with in host is +1, i.e., if the recruitment rates of human population and rate of infectious with in the host increase (decrease) 10%, the value of R_0 increases (decreases) 10%. The sensitivity index rate of symptomatically infected to isolated infected human and recovery rate of symptomatically infected human is negative, which means that if increase β_{13} and β_{14} then R_0 decreases 4.3% and 5.6%, respectively. Therefore, the increases of symptomatically infected to isolated infected human and recovery rate of symptomatically infected human can reduce the epidemic spreads.

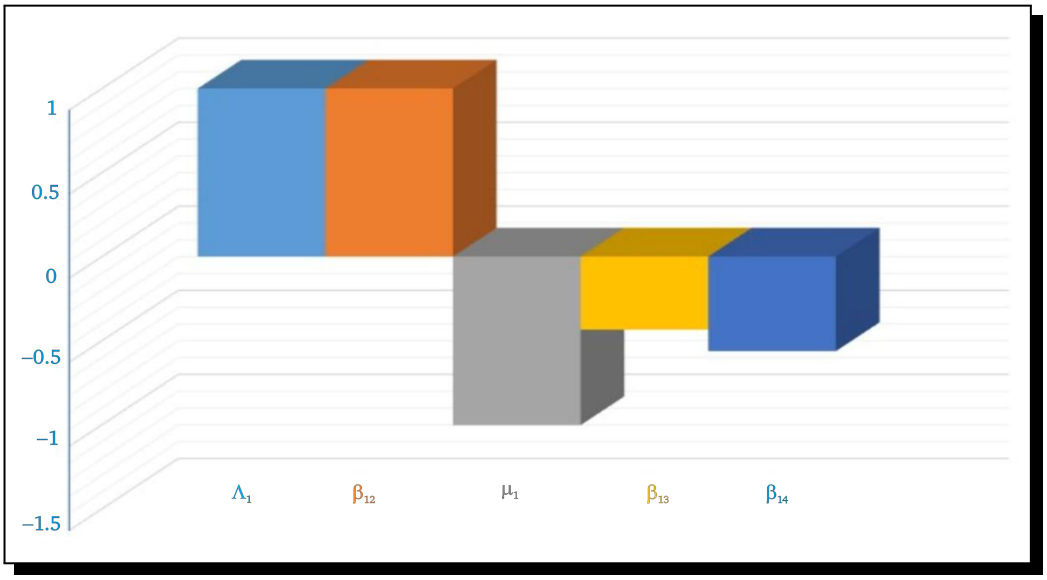

Figure 2. Sensitivity indices of R_0

6. Numerical Simulation

To demonstrate the preceding analysis finding, the model's numerical simulations are performed with help of the parameter values shown in Table 3. Simulated times of susceptible, infected, symptomatically infected, and isolated infected carriers were displayed for the host population, while simulated times of susceptible and infected individuals were presented for the vector population.

Table 3. System (2.1) parameters values

Parameter	Values	Units	Reference
Λ_1	.9999	day^{-1}	[8]
β_{11}	.8500	day^{-1}	[14]
β_{12}	.6294	day^{-1}	[29]
μ_1	.003468	day^{-1}	[19]
β_{13}	.555	day^{-1}	[5]
β_{14}	.7186	day^{-1}	[31]
β_{15}	.0062	day^{-1}	Assumed
Λ_2	.00034	day^{-1}	[25]
β_{16}	.009	day^{-1}	[9]
μ_2	.000244	day^{-1}	[4]

The dynamical system simulation shown in Figures 3–14 exhibits the various parameter's influence on the transmission dynamics model and demonstrates how these parameters are effective in causing epidemics in various human populations as well as vector populations. The population dynamics of the susceptible class are shown in Figures 3 and 4, with varied rates of infection within the host (β_{12}) and rates of symptomatic infection to isolated infected (β_{13}) individuals. Figures show that the first 20 days have a fluctuation, but afterwards, it becomes stable.

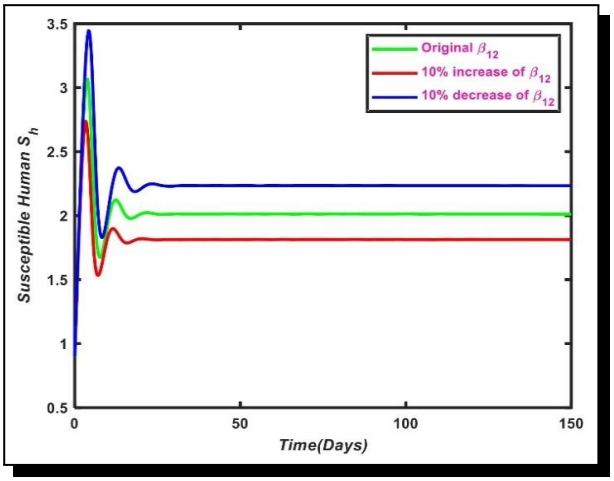

Figure 3. Effect on variation of β_{12} on S_h

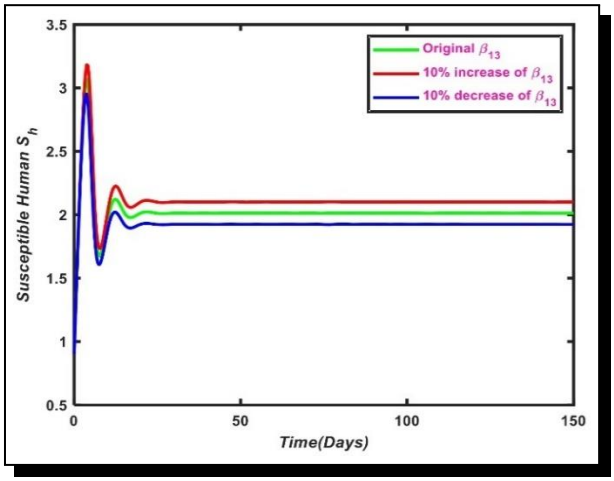

Figure 4. Effect on variation of β_{13} on S_h

Figures 5 and 6, indicate the effect on variation of β_{12} and β_{13} on infected individuals. There

is a slight impact of β_{13}, whereas huge impact on rates of infection within the host (β_{12}) of infected individuals. Both the figures decrease in the first 50 days, and after that they grow exponentially.

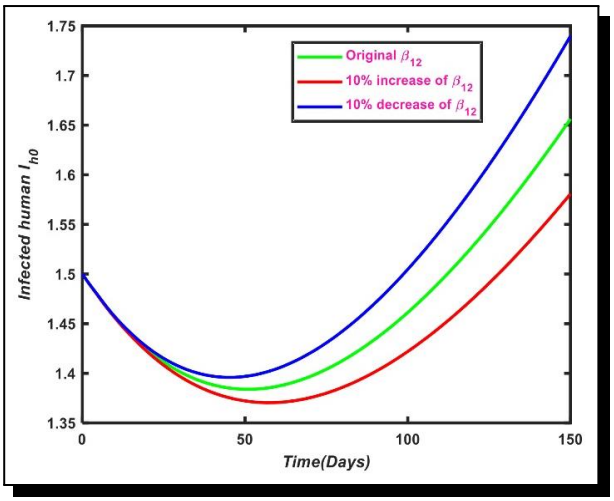

Figure 5. Effect on variation of β_{12} on I_{h0}

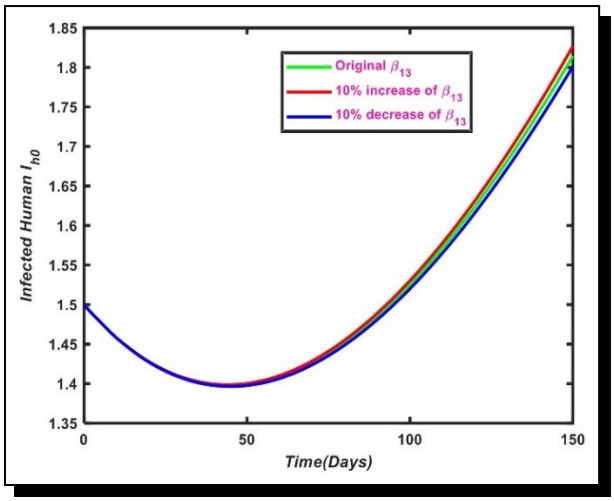

Figure 6. Effect on variation of β_{13} on I_{h0}

The population dynamics of the symptomatic infected individuals are shown in Figures 7 and 8, with variations β_{12} and β_{13}. Within the first 20 days have a fluctuation, but later it becomes stable.

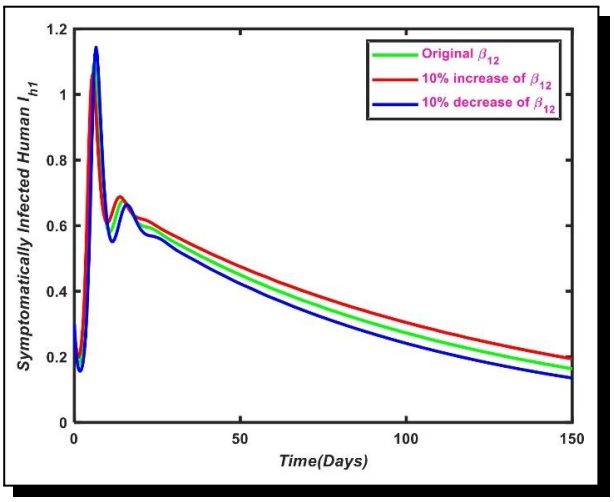

Figure 7. Effect on variation of β_{12} on I_{h1}

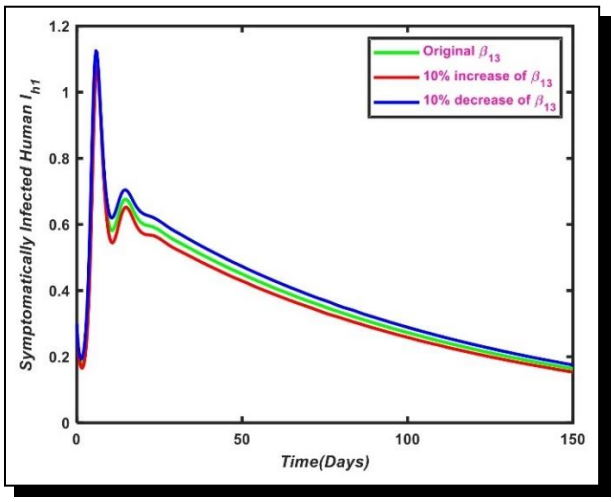

Figure 8. Effect on variation of β_{13} on I_{h1}

The effect on variation of β_{12} and β_{13} on the isolated infected individuals exhibit in Figures 9 and 10. Both the figures grows exponentially up to 100 days, while there is no effect on variation of β_{12} and β_{13} in between 0 to 25 days.

Infection rates from humans to vectors and rates of symptomatic infection to isolated infected are shown in Figures 11–12 with the behavior of a susceptible vector population. In between the first 60 days, the susceptible vector decreases after that it increases. Figure 11 shows when β_{13} increases to 10%, the susceptible vectors slightly up to the original, whereas in Figure 12, increases of β_{16} to 10% down to the original.

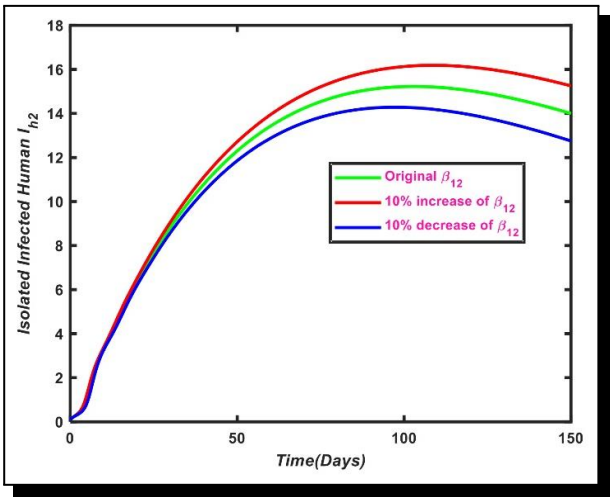

Figure 9. Effect on variation of β_{12} on I_{h2}

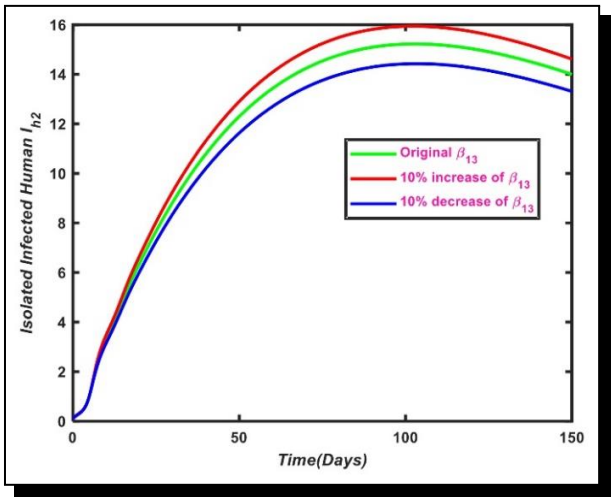

Figure 10. Effect on variation of β_{13} on I_{h2}

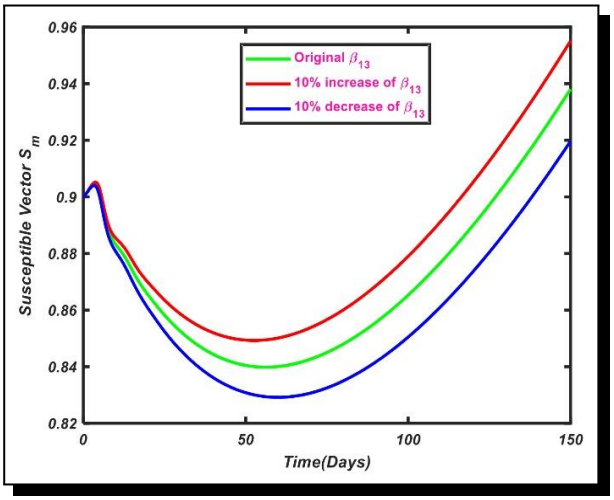

Figure 11. Effect on variation of β_{13} on S_m

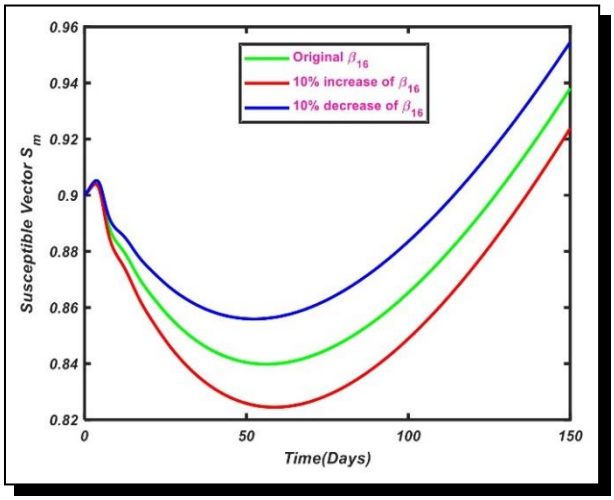

Figure 12. Effect on variation of β_{16} on S_m

Figures 13 and 14 show the variation of the infected vector with respect to time t for various values of β_{13} and β_{16}. Figure 13 demonstrates that if the rates of symptomatic infection to isolated infected increases then the infected vector decreases to the originals. On the other hand, Figure 14 illustrates that the increase of infection rates from humans to vectors increases the infected vector.

7. Conclusion

Dengue fever is a potentially fatal and dangerous illness that affects individuals all around the world. Developing appropriate management approaches for this viral disease is now a challenge for politicians, researchers, and public health experts. This article concentrated on examining the dynamics of dengue disease. We presented a mathematical model that models them while also accounting for the influence of the variable human with exponential growth. We used the Metzler property to investigate the positivity and boundedness of the system, as

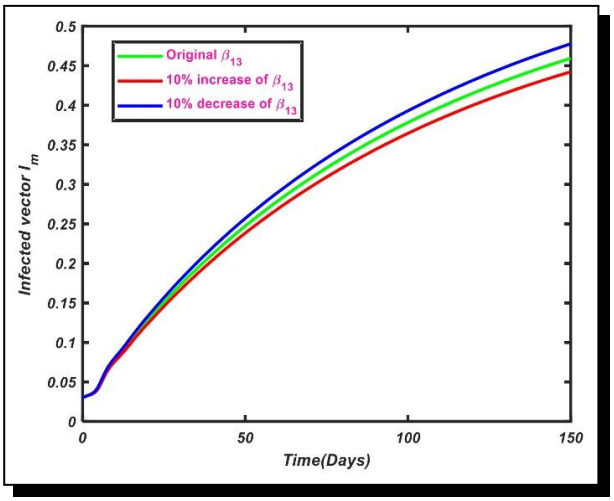

Figure 13. Effect on variation of β_{13} on I_m

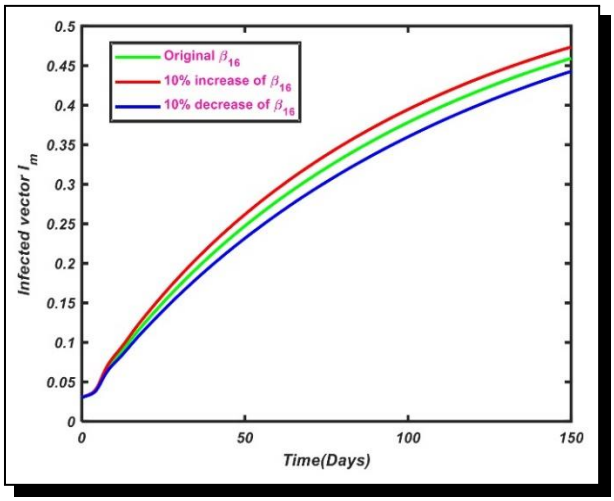

Figure 14. Effect on variation of β_{16} on I_m

well as the basic reproduction number (R_0), which controls disease transmission and the growth rate of the infected human population. The evolution of epidemics, the system's behaviors, and theoretical outcomes were all demonstrated using numerical simulation with varied parameter values. It was demonstrated that the sensitivity of R_0 achieved very high sensitivity for the model's parameters, such as the rate of symptomatically infected individuals to isolated infected individuals, as well as the rate of recovery of symptomatically infected humans can reduce the Dengue epidemic.

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Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

All the authors contributed significantly in writing this article. The authors read and approved the final manuscript.

References

[1] A. Abidemi, J. Ackora-Prah, H. O. Fatoyinbo and J. K. K. Asamoah, Lyapunov stability analysis and optimization measures for a dengue disease transmission model, *Physica A: Statistical Mechanics and its Applications* **602** (2022), 127646, DOI: 10.1016/j.physa.2022.127646.

[2] F. B. Agusto and M. A. Khan, Optimal control strategies for dengue transmission in Pakistan, *Mathematical Biosciences* **305** (2018), 102 – 121, DOI: 10.1016/j.mbs.2018.09.007.

[3] S. Ahmad, M. F. Yassen, M. M. Alam, S. Alkhati, F. Jarad and M. B. Riaz, A numerical study of dengue internal transmission model with fractional piecewise derivative, *Results in Physics* **39** (2022), 105798, DOI: 10.1016/j.rinp.2022.105798.

[4] S. A. Carvalho, S. O. da Silva and I. da C. Charret, Mathematical modeling of dengue epidemic: control methods and vaccination strategies, *Theory in Biosciences* **138** (2019), 223 – 239, DOI: 10.1007/s12064-019-00273-7.

[5] A. Chamnan, P. Pongsumpun, I.-M. Tang and N. Wongvanich, Local and global stability analysis of dengue disease with vaccination and optimal control, *Symmetry* **13**(10) (2021), 1917, DOI: 10.3390/sym13101917.

[6] L.-C. Chien and H.-L. Yu, Impact of meteorological factors on the spatiotemporal patterns of dengue fever incidence, *Environment International* **73** (2014), 46 – 56, DOI: 10.1016/j.envint.2014.06.018.

[7] A. L. Claypool, M. L. Brandeau and J. D. Goldhaber-Fiebert, Prevention and control of dengue and chikungunya in Colombia: A cost-effectiveness analysis, *PLOS Neglected Tropical Diseases* **15**(12) (2021), e0010086, DOI: 10.1371/journal.pntd.0010086.

[8] A. Dwivedi and R. Keval, Analysis for transmission of dengue disease with different class of human population, *Epidemiologic Methods* **10**(1) (2021), 20200046, DOI: 10.1515/em-2020-0046.

[9] A. Dwivedi, V. Baniya and R. Keval, Application of optimal controls on dengue dynamics — a mathematical study, in: *Mathematical Modelling and Computational Intelligence Techniques* (ICMMCIT2021), P. Balasubramaniam, K. Ratnavelu, G. Rajchakit and G. Nagamani (editors), Springer Proceedings in Mathematics & Statistics, Vol. **376**, Springer, Singapore (2021), DOI: 10.1007/978-981-16-6018-4_1.

[10] M. R. Hasan, A. Hobiny and A. Alshehri, Analysis of vector-host SEIR-SEI dengue epidemiological model, *International Journal of Analysis and Applications* **20** (2022), 57, DOI: 10.28924/2291-8639-20-2022-57.

[11] M. R. Hasan, A. A. Alsaiari, B. Z. Fakhurji, M. H. R. Molla, A. H. Asseri, M. A. A. Sumon, M. N. Park, F. Ahammad and B. Kim, Application of mathematical modeling and computational tools in the modern drug design and development process, *Molecules* **27**(13) (2022), 4169, DOI: 10.3390/molecules27134169.

[12] B. V. Huy, L. N. M. Hoa, D. T. Thuy, N. V. Kinh, T. T. D. Ngan, L. V. Duyet, N. T. Hung, N. N. Q. Minh, N. T. Truong and N. V. V. Chau, Epidemiological and clinical features of dengue infection in adults in the 2017 outbreak in Vietnam, *BioMed Research International* **2019** (2019), Article ID 3085827, 6 pages, DOI: 10.1155/2019/3085827.

[13] N. T. Huy, T. Van Giang, D. H. D. Thuy, M. Kikuchi, T. T. Hien, J. Zamora and K. Hirayama, Factors associated with dengue shock syndrome: a systematic review and meta-analysis, *PLOS Neglected Tropical Diseases* **7**(9) (2013), e2412, DOI: 10.1371/journal.pntd.0002412.

[14] M. A. Khan and Fatmawati, Dengue infection modeling and its optimal control analysis in East Java, Indonesia, *Heliyon* **7** (2021), e06023, DOI: 10.1016/j.heliyon.2021.e06023.

[15] G. Knerer, C. S. M. Currie and S. C. Brailsford, The economic impact and cost-effectiveness of combined vector-control and dengue vaccination strategies in Thailand: results from a dynamic transmission model, *PLOS Neglected Tropical Diseases* **14**(10) (2020), e0008805, DOI: 10.1371/journal.pntd.0008805.

[16] J.-S. Lee, M. Carabali, J. K. Lim, V. M. Herrera, I.-Y. Park, L. Villar and A. Farlow, Early warning signal for dengue outbreaks and identification of high risk areas for dengue fever in Colombia using climate and non-climate datasets, *BMC Infectious Diseases* **17** (2017), Article number: 480, DOI: 10.1186/s12879-017-2577-4.

[17] A. Li-Martín, R. Reyes-Carreto and C. Vargas-De-León, Dynamics of a dengue disease transmission model with two-stage structure in the human population, *Mathematical Biosciences and Engineering* **20**(1) (2023), 955 – 974, DOI: 10.3934/mbe.2023044.

[18] K. Maiga and A. Hugo, Modelling the impact of health care providers in transmission dynamics of COVID-19, *Results in Physics* **38**, 105552, DOI: 10.1016/j.rinp.2022.105552.

[19] P. Mutsuddy, S. T. Jhora, A. K. M. Shamsuzzaman, S. M. G. Kaisar and M. N. A. Khan, Dengue situation in Bangladesh: An epidemiological shift in terms of morbidity and mortality, *Canadian Journal of Infectious Diseases and Medical Microbiology* **2019** (2019), Article ID 3516284, 12 pages, DOI: 10.1155/2019/3516284.

[20] M. Z. Ndii, The effects of vaccination, vector controls and media on dengue transmission dynamics with a seasonally varying mosquito population, *Results in Physics* **34** (2022), 105298, DOI: 10.1016/j.rinp.2022.105298.

[21] M. Z. Ndii, A. R. Mage, J. J. Messakh and B. S. Djahi, Optimal vaccination strategy for dengue transmission in Kupang city, Indonesia, *Heliyon* **6**(11) (2020), e05345, DOI: 10.1016/j.heliyon.2020.e05345.

[22] M. Z. Ndii, R. I. Hickson, D. Allingham and G. N. Mercer, Modelling the transmission dynamics of dengue in the presence of Wolbachia, *Mathematical Biosciences* **262** (2015), 157 – 166, DOI: 10.1016/j.mbs.2014.12.011.

[23] L.-F. Nie and Y.-N. Xue, The roles of maturation delay and vaccination on the spread of Dengue virus and optimal control, *Advances in Difference Equations* **2017** (2017), Article number: 278, DOI: 10.1186/s13662-017-1323-y.

[24] H. J. Oladipo, I. Rabiu and Y. A. Tajudeen, Dengue virus and SARS-CoV-2 co-infection dynamics: An emerging threat among African countries, *Annals of Medicine & Surgery* **82** (2022), 104398, DOI: 10.1016/j.amsu.2022.104398.

[25] A. Omame, M. E. Isah and M. Abbas, An optimal control model for COVID-19, zika, dengue, and chikungunya co-dynamics with reinfection, *Optimal Control Applications and Methods* **44**(1) (2022), 170 – 204, DOI: 10.1002/oca.2936.

[26] A. Omame, H. Rwezaura, M. L. Diagne, S. C. Inyama and J. M. Tchuenche, COVID-19 and dengue co-infection in Brazil: optimal control and cost-effectiveness analysis, *The European Physical Journal Plus* **136** (2021), Article number: 1090, DOI: 10.1140/epjp/s13360-021-02030-6.

[27] P. W. Orellano, J. I. Reynoso, H.-C. Stahl and O. D. Salomon, Cost-utility analysis of dengue vaccination in a country with heterogeneous risk of dengue transmission, *Vaccine* **34**(5) (2016), 616 – 621, DOI: 10.1016/j.vaccine.2015.12.040.

[28] A. Pandey, A. Mubayi and J. Medlock, Comparing vector-host and SIR models for dengue transmission, *Mathematical Biosciences* **246**(2) (2013), 252 – 259, DOI: 10.1016/j.mbs.2013.10.007.

[29] L. Pimpi, S. W. Indratno, J. W. Puspita and E. Cahyono, Stochastic and deterministic dynamic model of dengue transmission based on dengue incidence data and climate factors in Bandung city, *Communication in Biomathematical Sciences* **5** (2022), 78 – 89, DOI: 10.5614/cbms.2022.5.1.5.

[30] T. Rawson, K. E. Wilkins and M. B. Bonsall, Optimal control approaches for combining medicines and mosquito control in tackling dengue, *Royal Society Open Science* **7**(4) (2020), 181843, DOI: 10.1098/rsos.181843.

[31] H. S. Rodrigues, M. T. T. Monteiro and D. F. M. Torres, Sensitivity analysis in a dengue epidemiological model, *Conference Papers in Science* **2013** (2013), Article ID 721406, 7 pages, DOI: 10.1155/2013/721406.

[32] H. S. Rodrigues, M. T. T. Monteiro and D. F. M. Torres, Vaccination models and optimal control strategies to dengue, *Mathematical Biosciences* **247** (2014), 1 – 12, DOI: 10.1016/j.mbs.2013.10.006.

[33] L. S. Sepulveda-Salcedo, O. Vasilieva and M. Svinin, Optimal control of dengue epidemic outbreaks under limited resources, *Studies in Applied Mathematics* **144**(2) (2020), 185 – 212, DOI: 10.1111/sapm.12295.

[34] E. Shim, Cost-effectiveness of dengue vaccination in Yucatán, Mexico using a dynamic dengue transmission model, *PLOS One* **12**(4) (2017), e0175020, DOI: 10.1371/journal.pone.0175020.

[35] T. Shragai, J. Pérez-Pérez, M. del Pilar Quimbayo-Forero, R. Rojo, L. C. Harrington and G. Rúa-Uribe, Distance to public transit predicts spatial distribution of dengue virus incidence in Medellín, Colombia, *Scientific Reports* **12** (2022), Article number: 8333, DOI: 10.1038/s41598-022-12115-6.

[36] A. K. Srivastav, P. K. Tiwari and M. Ghosh, Modeling the impact of early case detection on dengue transmission: deterministic vs. stochastic, *Stochastic Analysis and Applications* **39**(3) (2021), 434 – 455, DOI: 10.1080/07362994.2020.1804403.

[37] C. J. Tay, M. Fakhruddin, I. S. Fauzi, S. Y. Teh, M. Syamsuddin, N. Nuraini and E. Soewono, Dengue epidemiological characteristic in Kuala Lumpur and Selangor, Malaysia, *Mathematics and Computers in Simulation* **194** (2022), 489 – 504, DOI: 10.1016/j.matcom.2021.12.006.

[38] S. Ullah, M. F. Khan, S. A. A. Shah, M. Farooq, M. A. Khan, M. Bin Mamat, Optimal control analysis of vector-host model with saturated treatment, *The European Physical Journal Plus* **135** (2020), Article number: 839, DOI: 10.1140/epjp/s13360-020-00855-1.

[39] V. N. Valencia, Y. Díaz, J. M. Pascale, M. F. Boni and J. E. Sanchez-Galan, Assessing the effect of climate variables on the incidence of dengue cases in the metropolitan region of Panama city, *International Journal of Environmental Research and Public Health* **18** (2021), 12108, DOI: 10.3390/ijerph182212108.

[40] Y. Wang, Y. Wei, K. Li, X. Jiang, C. Li, Q. Yue, B. C.-Y. Zee and K. C. Chong, Impact of extreme weather on dengue fever infection in four Asian countries: A modelling analysis, *Environment International* **169** (2022), 107518, DOI: 10.1016/j.envint.2022.107518.

[41] T. T. Zheng and L. F. Nie, Modelling the transmission dynamics of two-strain Dengue in the presence awareness and vector control, *Journal of Theoretical Biology* **443** (2018), 82 – 91, DOI: 10.1016/j.jtbi.2018.01.017.

[42] L. Zou, J. Chen, X. Feng and S. Ruan, Analysis of a dengue model with vertical transmission and application to the 2014 dengue outbreak in Guangdong province, China, *Bulletin of Mathematical Biology* **80** (2018), 2633 – 2651, DOI: 10.1007/s11538-018-0480-9.