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A Mathematical Model of Logistic Human Population Growth and Vector Population for Dengue Transmission Dynamics

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Abstract: Both physical and mental health can be impacted by diseases, since a person's outlook on life may change as a result of acquiring and managing a health condition. Understanding the dynamics of diseases can be greatly appreciated in dealing and maintaining the endemic strategically. In this paper, a mathematical model based on the SEIR (Susceptible, Exposed, Infectious, Recovered) framework is presented for the dengue transmission dynamics. The mosquito population, which serves as the vector population and depends on the human population for subsistence, is represented in the model by a logistic function. To evaluate the model's capacity for spreading disease, the fundamental reproduction number R_0 is calculated. The disease-free equilibrium is determined to be locally stable if R_0 is less than one and unstable if R_0 is more significant than one. A stability analysis of the endemic and disease-free equilibria is carried out. The findings of this study offer insightful information about dengue transmission dynamics and can guide the development of effective strategies for disease control and prevention.

Keywords: Transmission Dynamics; Dengue; Disease-free Equilibrium; Stability Analysis.

2020 AMS Subject Classifications: 92B05.

1. Introduction

Dengue, a widespread viral disease transmitted by mosquitoes, poses a significant global health concern due to its potential for severe symptoms and occasional fatalities. Mathematical models

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offer valuable insights into understanding dengue transmission dynamics, aiding the development of effective control strategies. This study introduces a SEIR mathematical model focusing on dengue transmission dynamics, integrating a logistic function to portray mosquito population growth and survival. The analysis determines the basic reproduction number R_0, and evaluates the stability of disease equilibria, aiming to provide critical perspectives on dengue transmission patterns for enhanced disease control strategies.

Research in disease transmission modelling, notably by LaSalle (1976) and Singh *et al.* (2019, 2016a), has provided fundamental insights into stability analysis and disease dynamics, such as malaria and HIV/AIDS transmission. Studies by Van den Driessche and Watmough (2002) further explored reproduction numbers' implications in disease models, shedding light on endemic and disease-free equilibrium stability. Several researchers investigated the nexus between disease dynamics and climate factors. Bal and Sodoudi (2020) integrated climate variables into dengue models, elucidating transmission dynamics. Baylis (2017) emphasized climate change's impact on vector-borne diseases, stressing the need for its consideration in risk assessments. Benedum *et al.* (2018) analyzed rainfall's influence on dengue transmission, highlighting the role of environmental variables. Globally, Bhatt *et al.* (2013) provided an overview of dengue distribution and its public health impact. Studies by Butterworth *et al.* (2017), Caldwell *et al.* (2021) and Davis *et al.* (2021) addressed climate change's implications on disease dynamics, focusing on specific regions and forecasting disease adaptation to changing climates.

Das *et al.* (2023) has used basic reproduction number as a controlling parameter to the coronavirus pandemic. Akinwande*et al.* (2024), has demonstrated the prediction of a disease evolvement using the basic reproduction number. Recently, a dispersal strategy for infected individuals in a spatial susceptible-infected-susceptible (SIS) epidemic model has been proposed by Choi and Ahn (2024). A novel fractional model for simulating the coronavirus spread have been designed considering susceptible, infected, treated, and recovered classes in which the susceptible class is further subdivided into two subcategories (Adel *et al.* 2024). El-Mesady *et al.* (2024) simulate the spread of a novel Lumpy skin disease virus using a novel Caputo fractional nonlinear model. They investigated the equilibrium and stability points of the model and the basic reproduction number. A similar study has been conducted to simulate the monkey pox virus spread in the human host and rodent populations (El-Mesady *et al.* 2022).

Higazy *et al.* (2021) studied Infectious diseases in pregnant women know as Lassa hemorrhagic fever. The study found the approximate solution to the fractional-order model pf the disease using Laplace transforms and the Adomian decomposition method. Adel *et al.* (2023) investigate the dynamics of a novel fractional-order monkey pox epidemic model with optimal control. They established stability of the disease-free points. El-Mesady *et al.* (2023) studied monkey pox virus infection model considering stability analysis and optimal control strategies. The interaction between human and rodent populations along with the effects of control signals are investigated in the model. A similar study has been conducted introducing factors such as imperfect vaccination and nonlinear incidence rates (Elsonbaty *et al.* 2024).

Further investigations by Gutierrez *et al.* (2022), Huber *et al.* (2018), Kakarla *et al.* (2020) and Liu-Helmersson *et al.* (2016) delved into climate-related factors influencing dengue

transmission, forecasting future risks and transmission patterns. Marino *et al.* (2008) and Mordecai *et al.* (2017) explored uncertainties and temperature effects on disease spread, while Morin *et al.* (2013) reviewed climate's impact on dengue. Recent studies by Nuraini *et al.* (2021), Wang *et al.* (2022) and Xu *et al.* (2020) continued examining climate-driven disease dynamics, exploring predictive models and environmental influences on disease transmission. This literature review highlights various studies' contributions, covering stability analysis, disease spread models, co-infection dynamics, environmental factors, climate change impacts, and mathematical modelling. These studies offer a thorough comprehension of the intricate interactions between climate, vectors, and disease dynamics in dengue transmission. Further research is crucial to address remaining uncertainties, enhance modeling approaches, and create efficient plans for controlling and preventing dengue. This study's contributions and pertinent articles from the literature are encapsulated in Table 1. This table elucidates the gaps existing in prior research, and our paper aims to address these gaps by offering innovative insights and solutions within the field.

Table 1: Summary of key features of the present study and other relevant articles

Author	**Specific features**						
	SIR Model	Dengue Fever	Reproduction Number	Disease-Free Equilibrium	Endemic Equilibrium	Stability Analysis	Bifurcation
Chen & Hsieh (2012)	×	×	×	✓	✓	✓	×
Syafruddin & Noorani (2012)	✓	✓	×	✓	×	×	×
Chanprasopchai *et al*. (2018)	✓	✓	✓	✓	×	✓	×
Huber *et al.* (2018)	✓	✓	×	×	×	✓	×
Nur *et al.* (2018)	✓	✓	✓	✓	✓	✓	×
Singh *et al*. (2019)	×	×	×	✓	✓	✓	×
Chamnan *et al.* (2021)	✓	✓	✓	✓	✓	×	×
Khalid *et al*. (2015)	✓	✓	✓	✓	×	×	×
Asmaidi (2014)	✓	✓	✓	✓	✓	×	×
Proposed Model	✓	✓	✓	✓	✓	✓	✓

2. Methods

This section provides a concise model description outlining the critical architecture and components. Follow it with a brief analysis, highlighting its strengths and limitations and emphasizing its applicability to the research objectives.

2.1 Model Description

The proposed work will analyze the population dynamics for the spread of dengue disease using the SEIR model. The overall population is categorized into two distinct groups: human and

vector populations. The total human population is represented by $N_h(t)$ and subdivided into four classes: Susceptible Humans (S_h), Exposed Humans (E_h), Infected Humans (I_h), and Recovered Humans (R_h). Also, the overall vector population at time t is represented by $N_v(t)$ and subdivided into three classes: the Susceptible Vector (S_v), Exposed Vector (E_v), and Infected Vector (I_v).

In this model, we assume that individuals enter the susceptible human class through processes like birth. Upon being bitten by an infected vector, a susceptible human undergoes the stages of exposure and subsequent infection at variable rates. Recovered individuals emerge from the infected class after a specified duration. Similarly, susceptible vectors are introduced into the population at a specific rate. The vector initially shifts to the exposed class, and over time, individuals progress from the exposed class to the infected class. Susceptible human populations are recruited at a rate $\left(\lambda - \frac{\gamma N_h}{k}\right)N_h$, where λ is the birth rate, γ is the growth rate, and k is the carrying capacity of the human population. Susceptible humans get the virus from an infected vector following an effective constant at the rate $\frac{b\beta_1 I_v S_h}{N_h}$. The dengue virus will likely infect humans through its vector population, represented by number β_1. Susceptible mosquitoes recruited at rate A and become infectious at rate $\frac{b\beta_2 I_h S_v}{N_h}$ after being contacted by infected humans. The likelihood that the dengue virus will spread through humans into the mosquito population is shown by β_2. ξ_h represents a development rate from exposed class E_h to contaminated class I_h, and ξ_v represents a development rate from exposed class E_v to contaminated class I_v. The improvement rate for the human population is δ_h. μ_h denotes the inherent death estimate within the person inhabitants, and μ_v represents the inherent death estimate within the vector population. Figure 1 illustrates the transmission diagram for the dengue fever model, while Table 2 provides definitions of the state parameters used in the model, offering clarity on the variables influencing the system's dynamics.

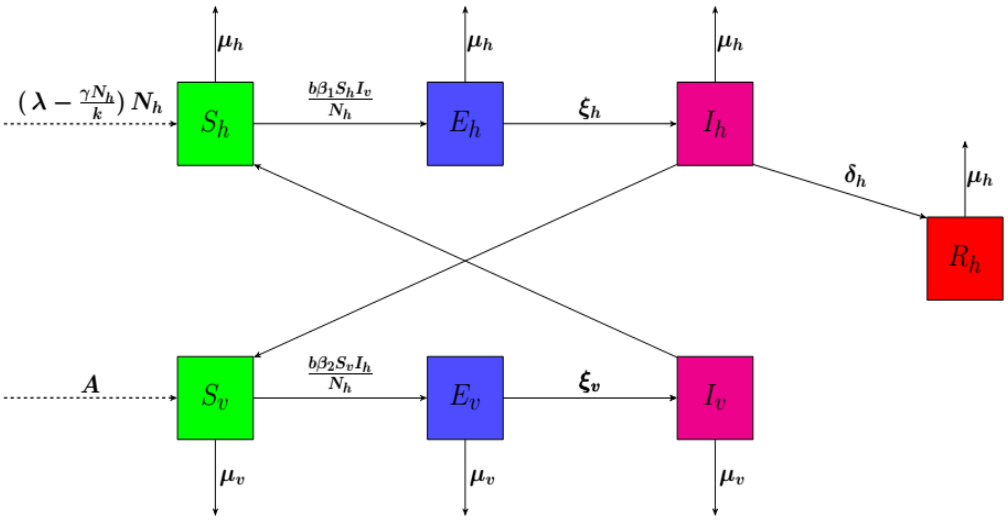

Figure 1: Transmission Dynamic Diagram.

Table 2: Definition of state parameters used in the model

Symbols	Description
λ	Recruitment rate of human population
γ	Growth rate of human population
k	Environment can support the maximum human population
b	Biting rate of vector population
A	Constant recruitment rate of vector population
β_1	Transmission probability from vector to human population
β_2	Transmission probability from human to vector population
ξ_h	Progression rate from exposed class E_h to infected class I_h
ξ_v	Progression rate from exposed class E_v to infected class I_v
δ_h	Recovery rate for human population
μ_h	Natural death rates of human population
μ_v	Natural death rates of vector population

2.2 Governing Equations

The model's governing differential equations are formulated by taking into account the relevant inflow and outflow rates for each compartment:

(I) Human population

$$S_h' = \left(\lambda - \frac{\gamma N_h}{k}\right)N_h - \left(\frac{b\beta_1 I_v}{N_h} + \mu_h\right)S_h,$$

$$E_h' = \frac{b\beta_1 I_v}{N_h} S_h - (\mu_h + \xi_h)E_h, \tag{1}$$

$$I_h' = \xi_h E_h - (\mu_h + \delta_h) I_h,$$

$$R_h' = \delta_h I_h - \mu_h R_h.$$

(II) Vector population

$$S_v' = A - \left(\frac{b\beta_2 I_h}{N_h} + \mu_v \right) S_v,$$

$$E_v' = \frac{b\beta_2 I_h}{N_h} S_v - (\mu_v + \xi_v) E_v, \tag{2}$$

$$I_v' = \xi_v E_v - \mu_v I_v.$$

with initial conditions;

$S_h(t) \geq 0, E_h(t) \geq 0, I_h(t) \geq 0, R_h(t) \geq 0, S_v(t) \geq 0, E_v(t) \geq 0, I_v(t) \geq 0$. (Ali, *et al.,* 2019)

Therefore, the total human population and vector population is given by

$N_h = S_h + E_h + I_h + R_h \Rightarrow R_h = N_h - S_h - E_h - I_h$ and

$$N_v = S_v + E_v + I_v = \frac{A}{\mu_v} \Rightarrow S_v = \frac{A}{\mu_v} - E_v - I_v.$$

The system of equations (1) and (2) can be simplified by letting

$$s_h = S_h N_h^{-1}, e_h = E_h N_h^{-1}, i_h = I_h N_h^{-1}, r_h = R_h N_h^{-1}, s_v = S_v N_v^{-1}, e_v = E_v N_v^{-1} \text{ and } i_v = I_v N_v^{-1}. \tag{3}$$

Differentiating the above system of equation (3) w.r.t, time

$$\frac{ds_h}{dt} = \frac{1}{N_h}\left(\frac{dS_h}{dt} - s_h \frac{dN_h}{dt} \right) = \lambda - \frac{\gamma N_h}{k} - nb\beta_1 s_h i_v - \gamma\left(1 - \frac{N_h}{k}\right) s_h - \mu_h s_h,$$

$$\frac{de_h}{dt} = \frac{1}{N_h}\left(\frac{dE_h}{dt} - e_h \frac{dN_h}{dt} \right) = nb\beta_1 s_h i_v - (\mu_h + \xi_h) e_h - \gamma\left(1 - \frac{N_h}{k}\right) e_h,$$

$$\frac{di_h}{dt} = \frac{1}{N_h}\left(\frac{dI_h}{dt} - i_h \frac{dN_h}{dt} \right) = \xi_h e_h - (\mu_h + \delta_h) i_h - \gamma\left(1 - \frac{N_h}{k}\right) i_h,$$

$$\frac{dr_h}{dt} = \frac{1}{N_h}\left(\frac{dR_h}{dt} - r_h \frac{dN_h}{dt} \right) = \delta_h i_h - \mu_h r_h - \gamma\left(1 - \frac{N_h}{k}\right) r_h,$$

$$\frac{ds_v}{dt} = \frac{1}{N_v}\left(\frac{dS_v}{dt} - s_v \frac{dN_v}{dt} \right) = \mu_v - b\beta_2 s_v i_h - \mu_v s_v,$$

$$\frac{de_v}{dt} = \frac{1}{N_v}\left(\frac{dE_v}{dt} - e_v \frac{dN_v}{dt} \right) = b\beta_2 s_v i_h - (\mu_v + \xi_v) e_v,$$

$$\frac{di_v}{dt} = \frac{1}{N_v}\left(\frac{dI_v}{dt} - i_v \frac{dN_v}{dt}\right) = \xi_v e_v - \mu_v i_v.$$

Hence, the simplified system of equations becomes

$$\frac{ds_h}{dt} = \lambda - \gamma\alpha - nb\beta_1 s_h i_v - \gamma(1-\alpha)s_h - \mu_h s_h,$$

$$\frac{de_h}{dt} = nb\beta_1 s_h i_v - (\mu_h + \xi_h)e_h - \gamma(1-\alpha)e_h,$$

$$\frac{di_h}{dt} = \xi_h e_h - (\mu_h + \delta_h)i_h - \gamma(1-\alpha)i_h,$$

$$\frac{dr_h}{dt} = \delta_h i_h - \mu_h r_h - \gamma(1-\alpha)r_h, \tag{4}$$

$$\frac{ds_v}{dt} = \mu_v - b\beta_2 s_v i_h - \mu_v s_v,$$

$$\frac{de_v}{dt} = b\beta_2 s_v i_h - (\mu_v + \xi_v)e_v,$$

$$\frac{di_v}{dt} = \xi_v e_v - \mu_v i_v.$$

Where, $\alpha = \frac{N_h}{k}$.

3. Basic properties

This section explores essential principles pivotal for the subsequent mathematical examination of the provided model.

3.1 Invariant region

Considering the model's tracking of swap in the person inhabitants, we assume all variables are changeable and constructive for $t \geq 0$. Thus, the structure of calculations (4) is analyzed within a biologically relevant and feasible region denoted as Ω. The following lemma outlines the feasible region for the system (4).

Lemma 1: The solution of the normalized model structure (4) is contained in the region $\Omega = \Omega_h \cup \Omega_v \subset \Re_+^4 \times \Re_+^3$.

Proof: To demonstrate that in a proper subset of $\Omega \subset \Re_+^4 \times \Re_+^3$, all possible solutions are uniformly bounded. Splitting the system into a human component n_h and a vector component n_v such that

$$n_h = s_h + e_h + i_h + r_h = 1 \text{and } n_v = s_v + e_v + i_v = 1. \tag{5}$$

Consider any solution $\{s_h, e_h, i_h, r_h\}$ in $\Re_+^4$ with non-negative initial conditions. By applying Birkhoff and Rota's theorem (1989) to differential inequalities, it can be inferred that

$$\lim_{t\to\infty} S_h(t) \le 1. \tag{6}$$

It is also similar for the solution of the vector population. $\{s_v, e_v, i_v\} \in \Re_+^3$ that

$$\lim_{t\to\infty} S_v(t) \le 1. \tag{7}$$

From the system of equation (4), we have:

$n_h' = s_h' + e_h' + i_h' + r_h', n_h' = 0.$

Integrating, we get $\Rightarrow n_h = k_1$, where k_1 constant. But, from $n_h = s_h + e_h + i_h + r_h = 1$, it follows that $k_1 = 1.$

Hence; $\Omega_h = \{(s_h, e_h, i_h, r_h) \in \Re_+^4 : s_h + e_h + i_h + r_h = 1\}.$

Similarly: $n_v' = s_v' + e_v' + i_v'$, $n_v' = 0$.

Integrating, we get $\Rightarrow n_v = k_2$. Also, from $n_v = s_v + e_v + i_v = 1$, it follows that $k_2 = 1.$

Hence; $\Omega_v = \{(s_v, e_v, i_v) \in \Re_+^3 : s_v + e_v + i_v = 1\}.$

As a result, we can affirm that the region Ω remains positively invariant, confirming that the model is well-defined and holds biological significance. Consequently, we can focus on the dynamics produced by the normalized model (4) within the Ω region.

3.2 Positivity of solutions

Lemma 2: With the initial conditions proposed in the model to lie in Ω, where

$\Omega = \{(s_h, e_h, i_h, r_h, s_v, e_v, i_v) \in \Re_+^7 : s_h \ge 0, e_h \ge 0, i_h \ge 0, r_h \ge 0, s_v \ge 0, e_v \ge 0, i_v \ge 0\}.$

Then the solution set $\{s_h(t), e_h(t), i_h(t), r_h(t), s_v(t), e_v(t), i_v(t)\}$ of the simplified model system (4) is positive for all time $t \ge 0.$

Proof: By using the simplified model system (4), from the first equation we have

$$\frac{ds_h}{dt} = \lambda - \gamma\alpha - nb\beta_1 s_h i_v - \gamma(1-\alpha)s_h - \mu_h s_h,$$

$$= \lambda - \gamma\alpha - nb\beta_1 s_h i_v - \gamma s_h - \gamma\alpha s_h - \mu_h s_h,$$

$$\ge -(\gamma + \mu_h)s_h.$$

Integrating with initial condition, we have

$t = 0, s(0) = s_0,$

$s_h(t) \geq s_h(0)e^{-(\gamma+\mu_h)t} \geq 0.$

The second equation of the simplified model system (4), we have

$$\frac{de_h}{dt} = nb\beta_1 s_h i_v - (\mu_h + \xi_h)e_h - \gamma(1-\alpha)e_h,$$

$$= nb\beta_1 s_h i_v - \mu_h e_h - \xi_h e_h - \gamma e_h - \gamma\alpha e_h,$$

$$\geq -(\gamma + \mu_h + \xi_h)e_h.$$

Integrating with initial condition, we have

$t = 0, e(0) = e_0,$

$e_h(t) \geq e_h(0)e^{-(\gamma+\mu_h+\xi_1)t} \geq 0.$

Similarly, the remaining equation of the simplified system (4) are also positive for all $t > 0.$ Thus if we consider the third equation

$$\frac{di_h}{dt} = \xi_h e_h - (\mu_h + \delta_h)i_h - \gamma(1-\alpha)i_h,$$

$$= \xi_h e_h - \mu_h i_h - \delta_h i_h - \gamma i_h - \alpha i_h,$$

$$\geq -(\gamma + \mu_h + \delta_h)i_h.$$

The integration will give $i_h(t) \geq i_h(0)e^{-(\gamma+\mu_h+\delta_h)t} \geq 0.$

Now we consider the fourth equation

$$\frac{dr_h}{dt} = \delta_h i_h - \mu_h r_h - \gamma r_h + \gamma\alpha r_h,$$

$$\geq -(\gamma + \mu_h)r_h.$$

The integration will give $r_h(t) \geq r_h(0)e^{-(\gamma+\mu_h)t} \geq 0.$

also

$$\frac{ds_v}{dt} = \mu_v - b\beta_2 s_v i_h - \mu_v s_v \geq -\mu_v s_v.$$

The integration will give $s_v(t) \geq s_v(0)e^{-\mu_v t} \geq 0.$

and

$$\frac{de_v}{dt} = b\beta_2 s_v i_h - (\mu_v + \xi_v)e_v \geq -(\mu_v + \xi_v)e_v.$$

The integration will give $e_v(t) \geq e_v(0)e^{-(\mu_v+\xi_v)t} \geq 0.$

Lastly;

$$\frac{di_v}{dt} = \xi_v e_v - \mu_v i_v \geq -\mu_v i_v.$$

The integration will give $i_v(t) \geq i_v(0)e^{-\mu_v t} \geq 0$.

Therefore, the solution set $\{s_h(t), e_h(t), i_h(t), r_h(t), s_v(t), e_v(t), i_v(t)\}$ of the simplified model system (4) is positive for all time $t \geq 0$.

i.e $s_h(t) \geq 0, e_h(t) \geq 0, i_h(t) \geq 0, r_h(t) \geq 0, s_v(t) \geq 0, e_v(t) \geq 0, i_v(t) \geq 0$.

4. Analysis of the Model

This segment is dedicated to computing the symmetry states, particularly the disease-free equilibrium (DFE) and the endemic equilibrium (EE). Additionally, we conduct a firmness examination by calculating the fundamental reproduction number.

4.1 Disease Free Equilibrium (DFE) and Basic Reproduction Number

The normalized system of equation (4) has a disease-free equilibrium given by

$$E_0 = (s_h^0, e_h^0, i_h^0, r_h^0, s_v^0, e_v^0, i_v^0) = (1,0,0,0,1,0,0).$$

The assessment of the direct firmness of the ailment-free symmetry state relies on the procreation figure, following the methodology outlined in Anderson and May (1988). To delve into the regional firmness of this symmetry, we employ the next-generation concept, as elucidated by Alexander *et al.* (2005) and Mushayabasa *et al.* (2011). To facilitate this, we introduce matrices F^* and V^*, designed to introduction recently developed infections and the transition of persons out of contaminated sections. The derivations proceed as follows:

$$F^* = \begin{pmatrix} nb\beta_1 s_h i_v \\ \xi_h e_h \\ b\beta_2 s_v i_h \\ \xi_v e_v \end{pmatrix}, \quad \text{and} \quad V^* = \begin{pmatrix} \gamma(1-\alpha)e_h + (\mu_h + \xi_h)e_h \\ \gamma(1-\alpha)i_h + (\mu_h + \delta_h)i_h \\ (\mu_v + \xi_v)e_v \\ \mu_v i_v \end{pmatrix}.$$

Once the partial derivatives of F^* and V^* at E_0 are calculated, the corresponding matrices are

$$F = \begin{pmatrix} 0 & 0 & 0 & nb\beta_1 \\ \xi_h & 0 & 0 & 0 \\ 0 & b\beta_2 & 0 & 0 \\ 0 & 0 & \xi_v & 0 \end{pmatrix}, \text{ and}$$

$$V = \begin{pmatrix} \gamma(1-\alpha)+(\mu_h+\xi_h) & 0 & 0 & 0 \\ 0 & \gamma(1-\alpha)+(\mu_h+\delta_h) & 0 & 0 \\ 0 & 0 & (\mu_v+\xi_v) & 0 \\ 0 & 0 & 0 & \mu_v \end{pmatrix}.$$

Consider the following matrix

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \dfrac{nb\beta_1}{\gamma(1-\alpha)+(\mu_h+\xi_h)} \\ \dfrac{\xi_h}{\gamma(1-\alpha)+(\mu_h+\delta_h)} & 0 & 0 & 0 \\ 0 & \dfrac{b\beta_2}{(\mu_v+\xi_v)} & 0 & 0 \\ 0 & 0 & \dfrac{\xi_v}{\mu_v} & 0 \end{pmatrix}.$$

Thus, the reproduction number R_0, is obtained as

$$R_0 = \sqrt{\frac{nb^2\beta_1\beta_2\xi_h\xi_v}{(\gamma(1-\alpha)+(\mu_h+\delta_h))(\gamma(1-\alpha)+(\mu_h+\xi_h))(\mu_v+\xi_v)\mu_v}}. \tag{8}$$

4.2 Local Stability of Disease-Free Equilibrium (DFE)

In this segment, we consider the local stability of the DFE. They are presented in Theorem 1.

Theorem 1: The local asymptotic stability of the disease-free equilibrium E_0 in the system (4) is established when $R_0 < 1$, and it becomes unstable otherwise.

Proof: The Jacobian matrix of the model system (4) at E_0 is given by

$$J(E_0) = \begin{pmatrix} -\gamma(1-\alpha)-\mu_h & 0 & 0 & 0 & 0 & 0 & -nb\beta_1 \\ 0 & -\gamma(1-\alpha)-(\mu_h+\xi_h) & 0 & 0 & 0 & 0 & nb\beta_1 \\ 0 & \xi_h & -\gamma(1-\alpha)-(\mu_h+\delta_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta_h & -\gamma(1-\alpha)-\mu_h & 0 & 0 & 0 \\ 0 & 0 & -b\beta_2 & 0 & -\mu_v & 0 & 0 \\ 0 & 0 & b\beta_2 & 0 & 0 & -(\mu_v+\xi_v) & 0 \\ 0 & 0 & 0 & 0 & 0 & \xi_v & -\mu_v \end{pmatrix}$$

Trace $[J(E_0)] = -(2\mu_v + a_1 + a_2 + a_3 + a_4 + a_5) < 0,$

where, $a_1 = -[\gamma(1-\alpha)+\mu_h]$, $a_2 = -[\gamma(1-\alpha)+(\mu_h+\xi_h)]$, $a_3 = -[\gamma(1-\alpha)+(\mu_h+\delta_h)]$, $a_4 = -[\gamma(1-\alpha)+\mu_h]$, and $a_5 = -(\mu_v+\xi_v)$.

$$\text{Det}[J(E_0)] = \left(-\gamma(1-\alpha)-\mu_h-\lambda\right)^2\left(-\mu_v-\lambda\right)\begin{bmatrix}\left(\gamma(1-\alpha)+(\mu_h+\xi_h)+\lambda\right)\left(\gamma(1-\alpha)+(\mu_h+\delta_h)+\lambda\right)\\ \left((\mu_v+\xi_v)+\lambda\right)\left(\mu_v+\lambda\right)-nb^2\beta_1\beta_2\xi_h\xi_v\end{bmatrix}.$$

For $\text{Det}[J(E_0)] > 0,$

$$\Rightarrow \left[\left(\gamma(1-\alpha)+(\mu_h+\xi_h)+\lambda\right)\left(\gamma(1-\alpha)+(\mu_h+\delta_h)+\lambda\right)\left((\mu_v+\xi_v)+\lambda\right)\left(\mu_v+\lambda\right)-nb^2\beta_1\beta_2\xi_h\xi_v\right] > 0,$$

$$\Rightarrow \frac{nb^2\beta_1\beta_2\xi_h\xi_v}{\left(\gamma(1-\alpha)+(\mu_h+\xi_h)+\lambda\right)\left(\gamma(1-\alpha)+(\mu_h+\delta_h)+\lambda\right)\left((\mu_v+\xi_v)+\lambda\right)\left(\mu_v+\lambda\right)} < 1,$$

$$\Rightarrow R_0^2 < 1 \Rightarrow R_0 < 1.$$

This shows that the disease-free equilibrium point E_0 is locally asymptotically stable (LAS) if $R_0 < 1,$ otherwise unstable.

4.3 Global Stability of Disease-Free Equilibrium (DFE)

In this segment, we assess worldwide firmness by applying a comparison theorem outlined in Lakshmikantham *et al.* (1989) and Mushayabasa *et al.* (2011).

Theorem 2: The global asymptotic stability (GAS) of the disease-free equilibrium E_0 in the system (4) is established when $R_0 < 1,$ and it becomes unstable otherwise.

Proof: The behavior of the varying signifying the contaminated part in the order (4) can be stated as follows:

$$\begin{pmatrix}\frac{de_h}{dt}\\ \frac{di_h}{dt}\\ \frac{de_v}{dt}\\ \frac{di_v}{dt}\end{pmatrix} = \left(F-V\right)\begin{pmatrix}e_h\\ i_h\\ e_v\\ i_v\end{pmatrix} - F_i\begin{pmatrix}e_h\\ i_h\\ e_v\\ i_v\end{pmatrix}, \tag{9}$$

where

$$\begin{pmatrix}\frac{de_h}{dt}\\ \frac{di_h}{dt}\\ \frac{de_v}{dt}\\ \frac{di_v}{dt}\end{pmatrix} = \left(F-V\right)\begin{pmatrix}-\left(\mu_h+\xi_h\right)-\gamma\left(1-\alpha\right)+nb\beta_1\\ -\left(\mu_h+\delta_h\right)-\gamma\left(1-\alpha\right)+\xi_h\\ -\left(\mu_v+\xi_v\right)+b\beta_2\\ \xi_v-\mu_v\end{pmatrix} - F_i\begin{pmatrix}e_h\\ i_h\\ e_v\\ i_v\end{pmatrix}.$$ Thus

$$\begin{pmatrix} \frac{de_h}{dt} \\ \frac{di_h}{dt} \\ \frac{de_v}{dt} \\ \frac{di_v}{dt} \end{pmatrix} \leq (F - V) \begin{pmatrix} e_h \\ i_h \\ e_v \\ i_v \end{pmatrix}. \tag{10}$$

Given that every eigenvalue of the matrix $F - V$ possesses a negative real component, the stability of the linearized differential inequality (10) is ensured when R_0 is less than 1. Therefore $(e_h, i_h, e_v, i_v) \to (0, 0, 0, 0)$ as $t \to \infty$. Substituting $e_h = i_h = e_v = i_v = 0$, in (4) gives $s_h(t) \to s_h(0)$ as $t \to \infty$ and $s_v(t) \to s_v(0)$ as $t \to \infty$.

Hence, the DFE (E_0) is globally asymptotically stable for $R_0 < 1$ and unstable if $R_0 > 1$.

4.4 Forward Bifurcation Analysis

The occurrence of forward bifurcation is established by applying center-manifold criteria to the system (4). Utilizing the Center Manifold Theorem, as referenced in Castillo-Chavez and Song (2004), Okosun and Makinde (2014), and Carr, J. (1981), we analyze forward bifurcation. Two key parameters, denoted as a and b, are pivotal in determining the direction of forward bifurcation. Specifically, when a is less than 0 and b is greater than 0, the system experiences forward bifurcation. Using this theorem, we arrive at the following conclusion:

Theorem 3: Consider the following general system of ordinary differential equations with a parameter ϕ

$$\frac{dx}{dt} = f(x, \phi), \; f : \Re^n \times \Re \to \Re^n \text{ and } f \in C^2(\Re^n \times \Re). \tag{11}$$

Without sacrificing generality, we assumed that 0 is an equilibrium for system (11) for all values of the parameter ϕ, meaning $f(0, \phi) = 0$ for all ϕ. Additionally, we assume the following conditions:

A1: $A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0) \right)$ represents the linearization of system (11) around the equilibrium 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of A, and other eigenvalues of A have negative real parts.

A2: Matrix A possesses a right eigenvector w and a left eigenvector v corresponding to the eigenvalue of zero.

Let f_k represent the Kth component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \tag{12}$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial \phi \partial x_i}(0,0). \tag{13}$$

The local dynamic of (11) around 0 are totally governed by parameters *a* and *b*.

(i) $a > 0, b > 0$, when $\phi < 0$ with $|\phi| < 1$, 0 is LAS, and there exists a positive unstable equilibrium, when $0 < \phi < 1$, 0 is unstable and there exists a negative and LAS equilibrium.

(ii) $a < 0, b < 0$, when $\phi < 0$ with $|\phi| < 1$, 0 is unstable, when $0 < \phi < 1$, 0 is asymptotically stable and there exists a positive unstable equilibrium.

(iii) $a > 0, b < 0$, when $\phi < 0$ with $|\phi| < 1$, 0 is unstable, and there exists a LAS negative equilibrium, when $0 < \phi < 1$, 0 is stable, and a positive unstable equilibrium appears.

(iv) $a < 0, b > 0$, when ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Corresponding, a negative equilibrium becomes positive and LAS. [Sing *et al.* (2016b)]

To apply theorem 3 above, the following simplifications and change of variables are made first. Let $S_h = x_1$, $E_h = x_2$, $I_h = x_3$, $R_h = x_4$, $S_V = x_5$, $E_v = x_6$ and $I_v = x_7$. So that $N_h = x_1 + x_2 + x_3 + x_4$ and $N_v = x_5 + x_6 + x_7$. Further, by using vector notation $X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T$, model system (4) can be written in the form $\frac{dX}{dt} = F(X)$, with $F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)^T$,

$$\frac{dx_1}{dt} = f_1 = \lambda - \gamma\alpha - nb\beta_1 x_1 x_7 - \gamma(1-\alpha)x_1 - \mu_h x_1, \tag{14}$$

$$\frac{dx_2}{dt} = f_2 = nb\beta_1 x_1 x_7 - (\mu_h + \xi_h)x_2 - \gamma(1-\alpha)x_2, \tag{15}$$

$$\frac{dx_3}{dt} = f_3 = \xi_h x_2 - (\mu_h + \delta_h)x_3 - \gamma(1-\alpha)x_3, \tag{16}$$

$$\frac{dx_4}{dt} = f_4 = \delta_h x_3 - \mu_h x_4 - \gamma(1-\alpha)x_4, \tag{17}$$

$$\frac{dx_5}{dt} = f_5 = \mu_v - b\beta_2 x_5 x_3 - \mu_v x_5, \tag{18}$$

$$\frac{dx_6}{dt} = f_6 = b\beta_2 x_5 x_3 - (\mu_v + \xi_v)x_6, \tag{19}$$

$$\frac{dx_7}{dt} = f_7 = \xi_v x_6 - \mu_v x_7, \tag{20}$$

with $\lambda = \gamma + \mu_h$.

The Jacobian of the above normalized model system (14)-(20) is given as

$$J(E_0) = \begin{pmatrix} -(\lambda-\gamma\alpha) & 0 & 0 & 0 & 0 & 0 & -nb\beta_1 \\ 0 & -(\lambda-\gamma\alpha+\xi_h) & 0 & 0 & 0 & 0 & nb\beta_1 \\ 0 & \xi_h & -(\lambda-\gamma\alpha+\delta_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta_h & -(\lambda-\gamma\alpha) & 0 & 0 & 0 \\ 0 & 0 & -b\beta_2 & 0 & -\mu_v & 0 & 0 \\ 0 & 0 & b\beta_2 & 0 & 0 & -(\mu_v+\xi_v) & 0 \\ 0 & 0 & 0 & 0 & 0 & \xi_v & -\mu_v \end{pmatrix},$$

$$|J(E_0)| = -\mu_v(\lambda-\gamma\alpha)^2\left[\mu_v(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)(\mu_v+\xi_v) - nb^2\beta_1\beta_2\xi_h\xi_v\right].$$

$\Rightarrow |J(E_0)| = 0$. Either $-\mu_v = 0$, $(\lambda-\gamma\alpha)^2 = 0$,

or $\mu_v(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)(\mu_v+\xi_v) - nb^2\beta_1\beta_2\xi_h\xi_v = 0.$

$$\Rightarrow \mu_v(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)(\mu_v+\xi_v) = nb^2\beta_1\beta_2\xi_h\xi_v,$$

$$\Rightarrow \frac{nb^2\beta_1\beta_2\xi_h\xi_v}{\mu_v(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)(\mu_v+\xi_v)} = 1,$$

$$\Rightarrow R_0^2 = 1,$$

$$\Rightarrow R_0 = 1.$$

Let $\beta_1 = \beta^* = \dfrac{(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)(\mu_v+\xi_v)\mu_v}{nb^2\beta_1\beta_2\xi_h\xi_v}$.

Eigen vectors of $J(E_0)$: It can be shown that the Jacobian of (14)-(20) has a right eigenvector associated with the zero eigenvalues given by $W = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T$, which is obtained from

$$\begin{pmatrix} -(\lambda-\gamma\alpha) & 0 & 0 & 0 & 0 & 0 & -nb\beta_1 \\ 0 & -(\lambda-\gamma\alpha+\xi_h) & 0 & 0 & 0 & 0 & nb\beta_1 \\ 0 & \xi_h & -(\lambda-\gamma\alpha+\delta_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta_h & -(\lambda-\gamma\alpha) & 0 & 0 & 0 \\ 0 & 0 & -b\beta_2 & 0 & -\mu_v & 0 & 0 \\ 0 & 0 & b\beta_2 & 0 & 0 & -(\mu_v+\xi_v) & 0 \\ 0 & 0 & 0 & 0 & 0 & \xi_v & -\mu_v \end{pmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \\ w_7 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

where, $w_1 = -\dfrac{nb\beta_1 w_7}{(\lambda-\gamma\alpha)}$, $w_2 = \dfrac{nb\beta_1 w_7}{(\lambda-\gamma\alpha+\xi_h)}$, $w_3 = \dfrac{nb\beta_1\xi_h w_7}{(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)}$,

$w_4 = \dfrac{nb\beta_1\xi_h\delta_h w_7}{(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)(\lambda-\gamma\alpha)}$, $w_5 = -\dfrac{nb^2\beta_1\beta_2\xi_h w_7}{(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}$,

$w_6 = \dfrac{nb^2\beta_1\beta_2\xi_h w_7}{(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)(\mu_v+\xi_v)}$, and $w_7 > 0$.

Furthermore, the Jacobian of (14)-(20) has a left eigenvector associated with the zero eigenvalues given by $V = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)^T$, such that

$$\begin{pmatrix} -(\lambda-\gamma\alpha) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(\lambda-\gamma\alpha+\xi_h) & \xi_h & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\lambda-\gamma\alpha+\delta_h) & \delta_h & -b\beta_2 & b\beta_2 & 0 \\ 0 & 0 & 0 & -(\lambda-\gamma\alpha) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_v & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_v+\xi_v) & \xi_v \\ -nb\beta_1 & nb\beta_1 & 0 & 0 & 0 & 0 & -\mu_v \end{pmatrix} \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \\ v_6 \\ v_7 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

where, $v_2 = \dfrac{\mu_v}{nb\beta_1} v_7$, $v_3 = \dfrac{(\lambda-\gamma\alpha+\xi_h)\mu_v}{nb\beta_1\xi_h} v_7$, $v_6 = \dfrac{\xi_v}{(\mu_v+\xi_v)} v_7$,

$v_1 = v_4 = v_5 = 0$, and $v_7 > 0$.

4.5 Computation of parameters a and b

Given the normalised model system (14)-(20), the partial derivatives of F that are not zero are as follows:

$$\frac{\partial f_1}{\partial x_1} = -nb\beta_1 x_7 - (\gamma(1-\alpha)+\mu_h), \frac{\partial^2 f_1}{\partial x_1 \partial x_7} = -nb\beta_1, \frac{\partial f_2}{\partial x_1} = nb\beta_1 x_7, \frac{\partial^2 f_2}{\partial x_1 \partial x_7} = nb\beta_1,$$

$$\frac{\partial f_5}{\partial x_3} = -b\beta_2 x_5, \quad \frac{\partial^2 f_5}{\partial x_3 \partial x_5} = -b\beta_2, \quad \frac{\partial f_6}{\partial x_3} = b\beta_2 x_5, \quad \frac{\partial^2 f_6}{\partial x_3 \partial x_5} = b\beta_2.$$

Consequently, based on the aforementioned expression,

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$

$$= v_1 \sum_{i,j=1}^{7} w_i w_j \frac{\partial^2 f_1}{\partial x_i \partial x_j} + v_2 \sum_{i,j=1}^{7} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j} + v_5 \sum_{i,j=1}^{7} w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j} + v_6 \sum_{i,j=1}^{7} w_i w_j \frac{\partial^2 f_6}{\partial x_i \partial x_j},$$

$$= 2v_1 w_1 w_7 \frac{\partial^2 f_1}{\partial x_1 \partial x_7} + 2v_2 w_1 w_7 \frac{\partial^2 f_2}{\partial x_1 \partial x_7} + 2v_5 w_3 w_5 \frac{\partial^2 f_5}{\partial x_3 \partial x_5} + 2v_6 w_3 w_5 \frac{\partial^2 f_6}{\partial x_3 \partial x_5},$$

$$= 2v_1 w_1 w_7 (-nb\beta_1) + 2v_2 w_1 w_7 (nb\beta_1) + 2v_5 w_3 w_5 (-b\beta_2) + 2v_6 w_3 w_5 (b\beta_2).$$

Since $v_1 = v_5 = 0.$

$$\Rightarrow a = 2(nb\beta_1 v_2 w_1 w_7 + b\beta_2 v_6 w_3 w_5),$$

$$= 2\left(- \frac{n^2 b^2 \beta_1^2 v_2 w_7^2}{(\lambda - \gamma\alpha)} - \frac{n^2 b^4 \beta_1^2 \beta_2^2 v_6 \xi_h^2 w_7^2}{(\lambda - \gamma\alpha + \xi_h)^2 (\lambda - \gamma\alpha + \delta_h)^2} \right),$$

$$= -2\left(\frac{n^2 b^2 \beta_1^2 v_2 w_7^2}{(\lambda - \gamma\alpha)} + \frac{n^2 b^4 \beta_1^2 \beta_2^2 v_6 \xi_h^2 w_7^2}{(\lambda - \gamma\alpha + \xi_h)^2 (\lambda - \gamma\alpha + \delta_h)^2} \right),$$

$\Rightarrow a < 0.$

It can be shown that the pertinent non-zero partial derivatives of f are in order to ascertain the symbol of b.

$$\frac{\partial f_1}{\partial \beta_1} = -nbx_1 x_7, \quad \frac{\partial^2 f_1}{\partial x_7 \partial \beta_1} = -nbx_1, \quad \frac{\partial f_2}{\partial \beta_1} = nbx_1 x_7, \quad \frac{\partial^2 f_2}{\partial x_7 \partial \beta_1} = nbx_1.$$

So that,

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial \beta \partial x_i}(0,0),$$

$$= \frac{\partial^2 f_1}{\partial x_7 \partial \beta_1} v_1 w_7 + \frac{\partial^2 f_2}{\partial x_7 \partial \beta_1} v_2 w_7,$$

$$= v_1 w_7 (-nb) + v_2 w_7 (nb).$$

Since $v_1 = 0.$

$$\Rightarrow b = nbv_2 w_7 = \frac{\mu_v}{\beta_1} v_7 w_7.$$

Since $v_7 > 0$, and $w_7 > 0$, then $b > 0$.

Since, by using item (iv) of theorem 3, we have established forward bifurcation.

4.6 Endemic Equilibrium (EE)

The system (4) attains an endemic equilibrium point, which is given by:

$$\lambda-\gamma\alpha-nb\beta_1 s_h^* i_v^* - \gamma(1-\alpha)s_h^* - \mu_h s_h^* = 0, \tag{21}$$

$$nb\beta_1 s_h^* i_v^* - (\mu_h+\xi_h)e_h^* - \gamma(1-\alpha)e_h^* = 0, \tag{22}$$

$$\xi_h e_h^* - (\mu_h+\delta_h)i_h^* - \gamma(1-\alpha)i_h^* = 0, \tag{23}$$

$$\delta_h i_h^* - \mu_h r_h^* - \gamma(1-\alpha)r_h^* = 0, \tag{24}$$

$$\mu_v - b\beta_2 s_v^* i_h^* - \mu_v s_v^* = 0, \tag{25}$$

$$b\beta_2 s_v^* i_h^* - (\mu_v+\xi_v)e_v^* = 0, \tag{26}$$

$$\xi_v e_v^* - \mu_v i_v^* = 0. \tag{27}$$

Adding (21) and (22), we get

$$e_h^* = \frac{(\lambda-\gamma\alpha)(1-s_h^*)}{(\lambda-\gamma\alpha+\xi_h)}. \tag{28}$$

From (23), $e_h^* = \dfrac{(\lambda-\gamma\alpha+\delta_h)i_h^*}{\xi_h}$. (29)

From (24), $r_h^* = \dfrac{\delta_h i_h^*}{(\lambda-\gamma\alpha)}$. (30)

From (25), $i_h^* = \dfrac{\mu_v i_v^*}{b\beta_2(1-i_v^*)}$. (31)

From (21), $i_v^* = \dfrac{(\lambda-\gamma\alpha)(1-s_h^*)}{nb\beta_1 s_h^*}$. (32)

From (27), $e_v^* = \dfrac{\mu_v i_v^*}{\xi_v}$. (33)

Put the value of i_v^* and i_h^* in equation (31) and (29), we get

Therefore, $i_h^* = \dfrac{\mu_v(\lambda-\gamma\alpha)(1-s_h^*)}{nb^2\beta_1\beta_2 s_h^* - b\beta_2(\lambda-\gamma\alpha)(1-s_h^*)}$, (34)

and $e_h^* = \dfrac{(\lambda-\gamma\alpha+\xi_h)\mu_v(\lambda-\gamma\alpha)(1-s_h^*)}{nb^2\beta_1\beta_2\xi_h s_h^* - b\beta_2\xi_h(\lambda-\gamma\alpha)(1-s_h^*)}$. (35)

Equating equations (28) and (35), we get

$$\frac{(\lambda-\gamma\alpha)(1-s_h^*)}{(\lambda-\gamma\alpha+\xi_h)} = \frac{(\lambda-\gamma\alpha+\delta_h)\mu_v(\lambda-\gamma\alpha)(1-s_h^*)}{nb^2\beta_1\beta_2\xi_h s_h^* - b\beta_2\xi_h(\lambda-\gamma\alpha)(1-s_h^*)},$$

$$\Rightarrow nb^2\beta_1\beta_2\xi_h s_h^*(\lambda-\gamma\alpha)(1-s_h^*) - b\beta_2\xi_h(\lambda-\gamma\alpha)^2(1-s_h^*)^2 = (\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v(\lambda-\gamma\alpha)(1-s_h^*),$$

$$\Rightarrow (\lambda-\gamma\alpha)(1-s_h^*)\left[nb^2\beta_1\beta_2\xi_h s_h^* - b\beta_2\xi_h(\lambda-\gamma\alpha)(1-s_h^*)\right] = (\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v(\lambda-\gamma\alpha)(1-s_h^*),$$

$$\Rightarrow \left[nb^2\beta_1\beta_2\xi_h s_h^* - b\beta_2\xi_h(\lambda-\gamma\alpha)(1-s_h^*)\right] = (\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v,$$

$$\Rightarrow \left[nb^2\beta_1\beta_2\xi_h + b\beta_2\xi_h(\lambda-\gamma\alpha)\right]s_h^* - b\beta_2\xi_h(\lambda-\gamma\alpha) = (\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v,$$

$$\Rightarrow s_h^* = \frac{b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h\left[nb\beta_1+(\lambda-\gamma\alpha)\right]}.$$

Put the value of s_h^* in equation (28), we get

$$i_h^* = \frac{(\lambda-\gamma\alpha)}{(\lambda-\gamma\alpha+\xi_h)}\left[1-\frac{b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h\left[nb\beta_1+(\lambda-\gamma\alpha)\right]}\right],$$

$$\Rightarrow i_h^* = \frac{(\lambda-\gamma\alpha)}{(\lambda-\gamma\alpha+\xi_h)}\left[\frac{b\beta_2\xi_h\left[nb\beta_1+(\lambda-\gamma\alpha)\right]-b\beta_2\xi_h(\lambda-\gamma\alpha)-(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h\left[nb\beta_1+(\lambda-\gamma\alpha)\right]}\right],$$

$$\Rightarrow i_h^* = \frac{(\lambda-\gamma\alpha)}{(\lambda-\gamma\alpha+\xi_h)}\left[\frac{b\beta_2\xi_h\left[nb\beta_1+2(\lambda-\gamma\alpha)\right]-(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h\left[nb\beta_1+(\lambda-\gamma\alpha)\right]}\right],$$

$$\Rightarrow i_h^* = \frac{(\lambda-\gamma\alpha)}{(\lambda-\gamma\alpha+\xi_h)}\left[\frac{nb^2\beta_1\beta_2\xi_h+2(\lambda-\gamma\alpha)b\beta_2\xi_h-(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{nb^2\beta_1\beta_2\xi_h+b\beta_2\xi_h(\lambda-\gamma\alpha)}\right].$$

Put the value of i_h^* in equation (29), we get

$$e_h^* = \frac{(\lambda-\gamma\alpha+\delta_h)i_h^*}{\xi_h}\times$$
$$\frac{(\lambda-\gamma\alpha)}{(\lambda-\gamma\alpha+\xi_h)}\left[\frac{nb^2\beta_1\beta_2\xi_h+2(\lambda-\gamma\alpha)b\beta_2\xi_h-(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{nb^2\beta_1\beta_2\xi_h+b\beta_2\xi_h(\lambda-\gamma\alpha)}\right].$$

Put the value of i_h^* in equation (30), we get

$$r_h^* = \frac{\delta_h i_h^*}{(\lambda-\gamma\alpha)}\times\frac{(\lambda-\gamma\alpha)}{(\lambda-\gamma\alpha+\xi_h)}\left[\frac{nb^2\beta_1\beta_2\xi_h+2(\lambda-\gamma\alpha)b\beta_2\xi_h-(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{nb^2\beta_1\beta_2\xi_h+b\beta_2\xi_h(\lambda-\gamma\alpha)}\right],$$

$$\Rightarrow r_h^* = \frac{\delta_h i_h^*}{(\lambda-\gamma\alpha+\xi_h)}\left[\frac{nb^2\beta_1\beta_2\xi_h+2(\lambda-\gamma\alpha)b\beta_2\xi_h-(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{nb^2\beta_1\beta_2\xi_h+b\beta_2\xi_h(\lambda-\gamma\alpha)}\right].$$

Put the value of s_h^* in equation (32),we get

$$i_v^* = \frac{(\lambda-\gamma\alpha)\left[1-\frac{b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h\left[nb\beta_1+(\lambda-\gamma\alpha)\right]}\right]}{nb\beta_1\left[\frac{b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h\left[nb\beta_1+(\lambda-\gamma\alpha)\right]}\right]},$$

$$\Rightarrow i_v^* = \frac{(\lambda-\gamma\alpha)\left[\dfrac{b\beta_2\xi_h[nb\beta_1+(\lambda-\gamma\alpha)]-b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h[nb\beta_1+(\lambda-\gamma\alpha)]}\right]}{nb\beta_1\left[\dfrac{b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h[nb\beta_1+(\lambda-\gamma\alpha)]}\right]},$$

$$\Rightarrow i_v^* = \frac{(\lambda-\gamma\alpha)}{nb\beta_1}\left[\frac{b\beta_2\xi_h[nb\beta_1+(\lambda-\gamma\alpha)]-b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}\right].$$

Put the value of i_v^* in equation (33), we get

$$e_v^* = \frac{(\lambda-\gamma\alpha)\mu_v}{nb\beta_1\xi_v}\left[\frac{b\beta_2\xi_h[nb\beta_1+(\lambda-\gamma\alpha)]-b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}{b\beta_2\xi_h(\lambda-\gamma\alpha)+(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)\mu_v}\right],$$

and

$$s_v^* = \frac{nb\beta_1\mu_v(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)+2b\beta_2\xi_h(\lambda-\gamma\alpha)^2-\mu_v(\lambda-\gamma\alpha)(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h)}{nb\beta_1(b\beta_2\xi_h(\lambda-\gamma\alpha)+\mu_v(\lambda-\gamma\alpha+\xi_h)(\lambda-\gamma\alpha+\delta_h))}.$$

4.7 Global Stability of Endemic Equilibrium

In this subsection, we address the endemic equilibrium's general stability.

Theorem 4: The GAS of the endemic equilibrium E^* in the system (4) is established when $R_0 > 1$, and it becomes unstable otherwise.

Proof: Consider a Lyapunov function V as a means of demonstrating the global stability of endemic equilibrium.

$$V = (s_h - s_h^* - s_h^* \ln\frac{s_h}{s_h^*}) + (e_h - e_h^* - e_h^* \ln\frac{e_h}{e_h^*}) + (i_h - i_h^* - i_h^* \ln\frac{i_h}{i_h^*}) + (r_h - r_h^* - r_h^* \ln\frac{r_h}{r_h^*})$$
$$+ (s_v - s_v^* - s_v^* \ln\frac{s_v}{s_v^*}) + (e_v - e_v^* - e_v^* \ln\frac{e_v}{e_v^*}) + (i_v - i_v^* - i_v^* \ln\frac{i_v}{i_v^*}). \quad (36)$$

The derivative of V is

$$V' = \left(1-\frac{s_h^*}{s_h}\right)s_h' + \left(1-\frac{e_h^*}{e_h}\right)e_h' + \left(1-\frac{i_h^*}{i_h}\right)i_h' + \left(1-\frac{r_h^*}{r_h}\right)r_h' + \left(1-\frac{s_v^*}{s_v}\right)s_v' + \left(1-\frac{e_v^*}{e_v}\right)e_v' + \left(1-\frac{i_v^*}{i_v}\right)i_v' = 0.$$

Since,

$$s_h' = \lambda - \gamma\alpha - nb\beta_1 s_h i_v - (\lambda-\gamma\alpha)s_h,$$

$$e_h' = nb\beta_1 s_h i_v - (\lambda-\gamma\alpha+\xi_h)e_h,$$

$$i_h' = \xi_h e_h - (\lambda-\gamma\alpha+\delta_h)i_h,$$

$$r_h' = \delta_h i_h - (\lambda-\gamma\alpha)r_h,$$

$$s_v' = \mu_v - b\beta_2 s_v i_h - \mu_v s_v,$$

$$e_v' = b\beta_2 s_v i_h - (\mu_v + \xi_v)e_v,$$

$$i_v' = \xi_v e_v - \mu_v i_v.$$

Then

$$V' = \left(\frac{s_h - s_h^*}{s_h}\right)\left(\lambda - \gamma\alpha - nb\beta_1 s_h i_v - (\lambda - \gamma\alpha)s_h\right) + \left(\frac{e_h - e_h^*}{e_h}\right)\left(nb\beta_1 s_h i_v - (\lambda - \gamma\alpha + \xi_h)e_h\right)$$

$$+\left(\frac{i_h - i_h^*}{i_h}\right)\left(\xi_h e_h - (\lambda - \gamma\alpha + \delta_h)i_h\right) + \left(\frac{r_h - r_h^*}{r_h}\right)\left(\delta_h i_h - (\lambda - \gamma\alpha)r\right) + \left(\frac{s_v - s_v^*}{s_v}\right)\left(\mu_v - b\beta_2 s_v i_h - \mu_v s_v\right)$$

$$+\left(\frac{e_v - e_v^*}{e_v}\right)\left(b\beta_2 s_v i_h - (\mu_v + \xi_v)e_v\right) + \left(\frac{i_v - i_v^*}{i_v}\right)\left(\xi_v e_v - \mu_v i_v\right),$$

$$= \left(\frac{s_h - s_h^*}{s_h}\right)\left[\lambda - \gamma\alpha - \left\{nb\beta_1\left(i_v - i_v^*\right) + (\lambda - \gamma\alpha)\right\}\left(s_h - s_h^*\right)\right] + \left(\frac{e_h - e_h^*}{e_h}\right)\left\{\begin{matrix} nb\beta_1\left(s_h - s_h^*\right)\left(i_v - i_v^*\right) \\ -(\lambda - \gamma\alpha + \xi_h)\left(e_h - e_h^*\right)\end{matrix}\right\}$$

$$+\left(\frac{i_h - i_h^*}{i_h}\right)\left\{\xi_h\left(e_h - e_h^*\right) - (\lambda - \gamma\alpha + \delta_h)\left(i_h - i_h^*\right)\right\} + \left(\frac{r_h - r_h^*}{r_h}\right)\left\{\delta_h\left(i_h - i_h^*\right) - (\lambda - \gamma\alpha)\left(r_h - r_h^*\right)\right\}$$

$$+\left(\frac{s_v - s_v^*}{s_v}\right)\left[\mu_v - \left\{b\beta_2\left(i_h - i_h^*\right) + \mu_v\right\}\left(s_v - s_v^*\right)\right] + \left(\frac{e_v - e_v^*}{e_v}\right)\left\{b\beta_2\left(i_h - i_h^*\right)\left(s_v - s_v^*\right) - (\mu_v + \xi_v)\left(e_v - e_v^*\right)\right\}$$

$$+\left(\frac{i_v - i_v^*}{i_v}\right)\left\{\xi_v\left(e_v - e_v^*\right) - \mu_v\left(i_v - i_v^*\right)\right\},$$

$$= -(\lambda - \gamma\alpha)\frac{\left(s_h - s_h^*\right)^2}{s_h} + \frac{\left(s_h - s_h^*\right)}{s_h}\left[(\lambda - \gamma\alpha) - nb\beta_1\left(i_v - i_v^*\right)\left(s_h - s_h^*\right)\right] - (\lambda - \gamma\alpha + \xi_h)\frac{\left(e_h - e_h^*\right)^2}{e_h}$$

$$+\frac{\left(e_h - e_h^*\right)}{e_h}\left\{nb\beta_1\left(s_h - s_h^*\right)\left(i_v - i_v^*\right)\right\} - (\lambda - \gamma\alpha + \delta_h)\frac{\left(i_h - i_h^*\right)^2}{i_h} + \frac{\left(i_h - i_h^*\right)}{i_h}\left\{\xi_h\left(e_h - e_h^*\right)\right\}$$

$$-(\lambda - \gamma\alpha)\frac{\left(r_h - r_h^*\right)^2}{r_h} + \frac{\left(r_h - r_h^*\right)}{r_h}\left\{\delta_h\left(i_h - i_h^*\right)\right\} - \mu_v\frac{\left(s_v - s_v^*\right)^2}{s_v} + \frac{\left(s_v - s_v^*\right)}{s_v}\left[\mu_v - b\beta_2\left(i_h - i_h^*\right)\left(s_v - s_v^*\right)\right]$$

$$-(\mu_v + \xi_v)\frac{\left(e_v - e_v^*\right)^2}{e_v} + \frac{\left(e_v - e_v^*\right)}{e_v}\left\{b\beta_2\left(i_h - i_h^*\right)\left(s_v - s_v^*\right)\right\} - \mu_v\frac{\left(i_v - i_v^*\right)^2}{i_v} + \frac{\left(i_v - i_v^*\right)}{i_v}\left\{\xi_v\left(e_v - e_v^*\right)\right\},$$

$$= -(\lambda - \gamma\alpha)\frac{\left(s_h - s_h^*\right)^2}{s_h} + (\lambda - \gamma\alpha) - (\lambda - \gamma\alpha)\frac{s_h^*}{s_h} - \left(nb\beta_1 i_v - nb\beta_1 i_v^*\right)\frac{\left(s_h - s_h^*\right)^2}{s_h}$$

$$-(\lambda-\gamma\alpha+\xi_h)\frac{(e_h-e_h^*)^2}{e_h}+\frac{(e_h-e_h^*)}{e_h}\left(nb\beta_1 s_h i_v-nb\beta_1 s_h i_v^*-nb\beta_1 s_h^* i_v+nb\beta_1 s_h^* i_v^*\right)$$

$$-(\lambda-\gamma\alpha+\delta_h)\frac{(i_h-i_h^*)^2}{i_h}+\frac{(i_h-i_h^*)}{i_h}\left(\xi_h e_h-\xi_h e_h^*\right)-(\lambda-\gamma\alpha)\frac{(r_h-r_h^*)^2}{r_h}+\frac{(r_h-r_h^*)}{r_h}\left(\delta_h i_h-\delta_h i_h^*\right)$$

$$-\mu_v\frac{(s_v-s_v^*)^2}{s_v}+\mu_v-\mu_v\frac{s_v^*}{s_v}-b\beta_2 i_h\frac{(s_v-s_v^*)^2}{s_v}+b\beta_2 i_h^*\frac{(s_v-s_v^*)^2}{s_v}-(\mu_v+\xi_v)\frac{(e_v-e_v^*)^2}{e_v}$$

$$+\frac{(e_v-e_v^*)}{e_v}\left\{b\beta_2\left(i_h s_v-i_h s_v^*-s_v i_h^*+s_v^* i_h^*\right)\right\}-\mu_v\frac{(i_v-i_v^*)^2}{i_v}+\frac{(i_v-i_v^*)}{i_v}\left(\xi_v e_v-\xi_v e_v^*\right),$$

$$=-(\lambda-\gamma\alpha)\frac{(s_h-s_h^*)^2}{s_h}+(\lambda-\gamma\alpha)-(\lambda-\gamma\alpha)\frac{s_h^*}{s_h}-nb\beta_1 i_v\frac{(s_h-s_h^*)^2}{s_h}+nb\beta_1 i_v^*\frac{(s_h-s_h^*)^2}{s_h}$$

$$-(\lambda-\gamma\alpha+\xi_h)\frac{(e_h-e_h^*)^2}{e_h}+nb\beta_1 s_h i_v-nb\beta_1 s_h i_v^*-nb\beta_1 s_h^* i_v+nb\beta_1 s_h^* i_v^*-nb\beta_1 s_h i_v\frac{e_h^*}{e_h}$$

$$+nb\beta_1 s_h i_v^*\frac{e_h^*}{e_h}+nb\beta_1 s_h^* i_v\frac{e_h^*}{e_h}-nb\beta_1 s_h^* i_v^*\frac{e_h^*}{e_h}-(\lambda-\gamma\alpha+\delta_h)\frac{(i_h-i_h^*)^2}{i_h}+\xi_h e_h-\xi_h e_h^*$$

$$-\xi_h e_h\frac{i_h^*}{i_h}+\xi_h e_h^*\frac{i_h^*}{i_h}-(\lambda-\gamma\alpha)\frac{(r_h-r_h^*)^2}{r_h}+\delta_h i_h-\delta_h i_h^*-\delta_h i_h\frac{r_h^*}{r_h}+\delta_h i_h^*\frac{r_h^*}{r_h}-\mu_v\frac{(s_v-s_v^*)^2}{s_v}$$

$$+\mu_v-\mu_v\frac{s_v^*}{s_v}-b\beta_2 i_h\frac{(s_v-s_v^*)^2}{s_v}+b\beta_2 i_h^*\frac{(s_v-s_v^*)^2}{s_v}-(\mu_v+\xi_v)\frac{(e_v-e_v^*)^2}{e_v}+b\beta_2 i_h s_v-b\beta_2 i_h s_v^*$$

$$-b\beta_2 s_v i_h^*+b\beta_2 s_v^* i_h^*-b\beta_2 i_h s_v\frac{e_v^*}{e_v}+b\beta_2 i_h s_v^*\frac{e_v^*}{e_v}+b\beta_2 s_v i_h^*\frac{e_v^*}{e_v}-b\beta_2 s_v^* i_h^*\frac{e_v^*}{e_v}-\mu_v\frac{(i_v-i_v^*)^2}{i_v}$$

$$+\xi_v e_v-\xi_v e_v^*-\xi_v e_v\frac{i_v^*}{i_v}+\xi_v e_v^*\frac{i_v^*}{i_v}=0.$$

Separate positive and negative terms such that

$$G=nb\beta_1 i_v^*\frac{(s_h-s_h^*)^2}{s_h}+b\beta_2 i_h^*\frac{(s_v-s_v^*)^2}{s_v}+nb\beta_1 s_h i_v+nb\beta_1 s_h^* i_v^*+nb\beta_1 s_h i_v^*\frac{e_h^*}{e_h}+nb\beta_1 s_h^* i_v\frac{e_h^*}{e_h}$$

$$+\xi_h e_h+\xi_h e_h^*\frac{i_h^*}{i_h}+\delta_h i_h+\delta_h i_h^*\frac{r_h^*}{r_h}+\mu_v+b\beta_2 i_h s_v+b\beta_2 s_v^* i_h^*+b\beta_2 i_h s_v^*\frac{e_v^*}{e_v}+b\beta_2 s_v i_h^*\frac{e_v^*}{e_v}+\xi_v e_v$$

$$+\xi_v e_v^*\frac{i_v^*}{i_v}+(\lambda-\gamma\alpha),$$

and

$$H = -(\lambda - \gamma\alpha)\frac{(s_h - s_h^*)^2}{s_h} - nb\beta_1 i_v \frac{(s_h - s_h^*)^2}{s_h} - \mu_v \frac{(s_v - s_v^*)^2}{s_v} - b\beta_2 i_h \frac{(s_v - s_v^*)^2}{s_v}$$

$$-(\lambda - \gamma\alpha + \xi_h)\frac{(e_h - e_h^*)^2}{e_h} - (\lambda - \gamma\alpha + \delta_h)\frac{(i_h - i_h^*)^2}{i_h} - (\lambda - \gamma\alpha)\frac{(r_h - r_h^*)^2}{r_h} - (\mu_v + \xi_v)\frac{(e_v - e_v^*)^2}{e_v}$$

$$-\mu_v \frac{(i_v - i_v^*)^2}{i_v} - (\lambda - \gamma\alpha)\frac{s_h^*}{s_h} - nb\beta_1 s_h i_v \frac{e_h^*}{e_h} - nb\beta_1 s_h^* i_v^* \frac{e_h^*}{e_h} - \xi_h e_h \frac{i_h^*}{i_h} - \delta_h i_h \frac{r_h^*}{r_h} - \mu_v \frac{s_v^*}{s_v} - b\beta_2 i_h s_v \frac{e_v^*}{e_v}$$

$$-b\beta_2 s_v^* i_h^* \frac{e_v^*}{e_v} - \xi_v e_v \frac{i_v^*}{i_v} - nb\beta_1 s_h i_v^* - nb\beta_1 s_h^* i_v - \xi_h e_h^* - \delta_h i_h^* - b\beta_2 i_h s_v^* - b\beta_2 s_v i_h^* - \xi_v e_v^*.$$

If G < H then V' will be negative it means that $V' < 0$. It follows that $V' = 0 \Leftrightarrow s_h = s_h^*, e_h = e_h^*, i_h = i_h^*, r_h = r_h^*, s_v = s_v^*, e_v = e_v^*$ and $i_v = i_v^*$. The maximum invariant set of system (4) on the set $\{(s_h^*, e_h^*, i_h^*, r_h^*, s_v^*, e_v^*, i_v^*) : V' = 0\}$ is the singleton (E^*). Thus, for system (4), the endemic equilibrium E^* is GAS if $G < H$ by LaSalle's invariance principle (1976).

5. Results

The study's numerical simulations are illustrated through the application of the Runge-Kutta method to the normalized model system (4), utilizing the estimated parameter values listed in Table 3.

Table 3: Parameter values for the model of the disease dengue fever.

Parameters	Values	Source
λ	0.00002	Chen and Hsieh (2012)
γ	0.000004	Assumed
B	0.33	Adams and Boots (2010)
β_1	0.375	KMTL (2006)
β_2	0.75	KMTL (2006)
ξ_h	0.197	Assumed
ξ_v	0.183	Assumed
μ_h	0.000016	Chen and Hsieh (2012)
μ_v	0.331	Chen and Hsieh (2012)
δ_h	0.142	Adams and Boots (2010)
n	12	Assumed
α	0.637	Assumed

Figures 2 to 7 depict the variations in terms of proportions of susceptible humans, infected humans, recovered humans, susceptible vectors, exposed vectors, and infective vectors for various biting rates denoted as 'b'.

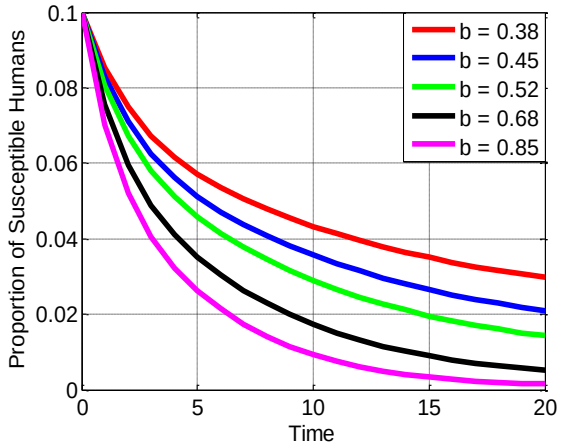

Figure 2: Variation in the susceptible human population for varying biting rates b .

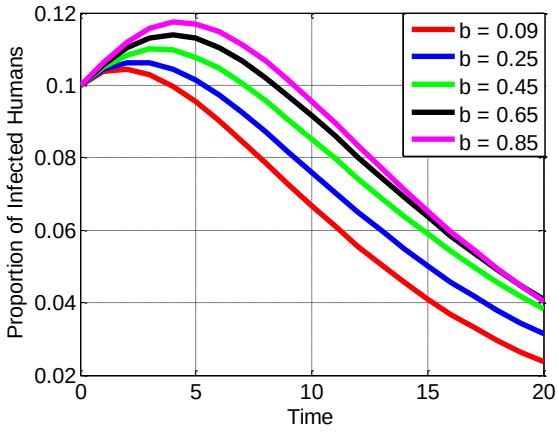

Figure 3: Variation in the infected human population for varying biting rates b .

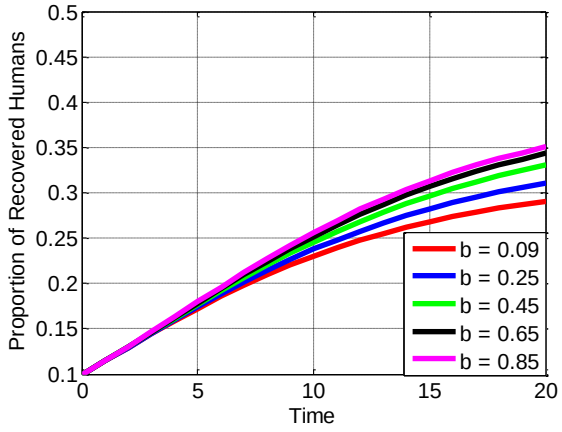

Figure 4: Variation in the recovered human population for varying biting rates b .

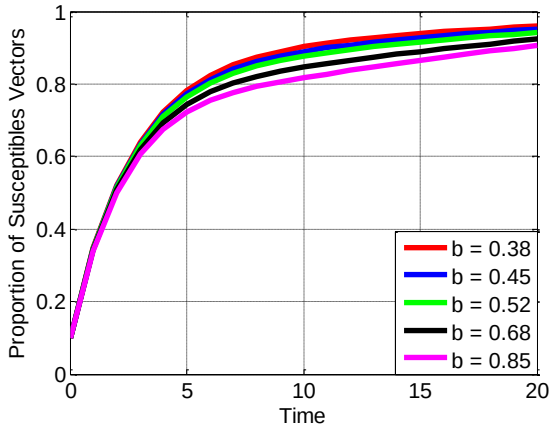

Figure 5: Variation in the susceptible vector population for varying biting rates b .

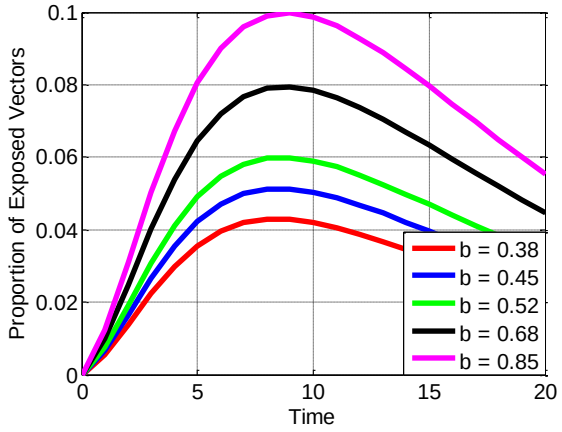

Figure 6: Variation in the exposed vector population for varying biting rates b .

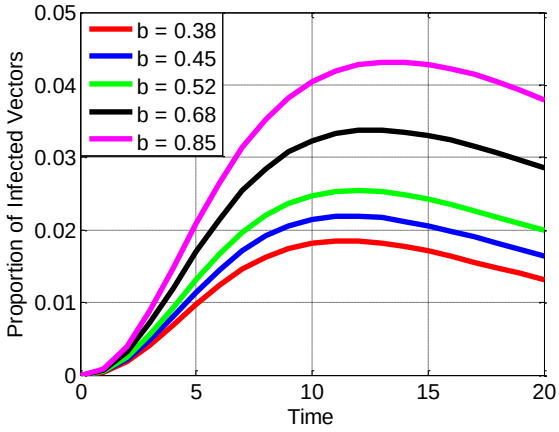

Figure 7: Variation in the infected vector population for varying biting rates b .

In Figure 2, a notable trend is the decline in the susceptible human population as the biting rate rises. In contrast, as we observe in Figures 3 and 4, a rise in the biting rate corresponds to a rise in the proportions of the infected and the recovered human population. Furthermore, when the rate of biting rises, there is a noticeable reduction in the proportion of susceptible vector populations. Conversely, a rise in the biting rate is associated with an increase in the proportions of exposed vector populations and infected vector populations, which is evident in Figures 6 and 7, respectively.

Figures 8 to 14 illustrate the changes in terms of proportions of susceptible, exposed, infected, and recovered human populations concerning susceptible, exposed, and infected vector populations. These variations are observed for different values of the transmission probability from vectors to the human population, denoted as β_1.

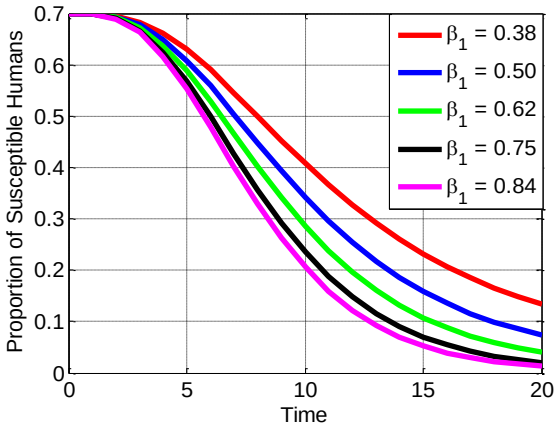

Figure 8: Variation in the proportion of susceptible human for different transmission probability rates β_1.

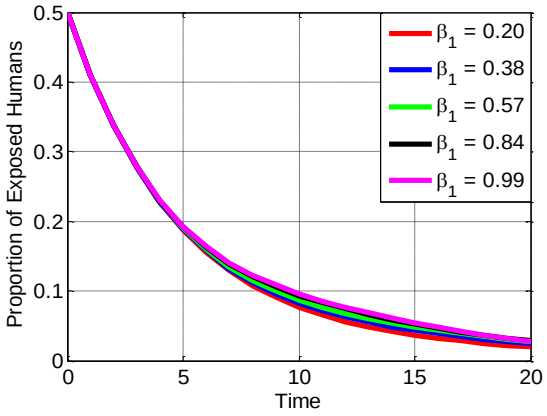

Figure 9: Variation in the proportion of exposed human for different transmission probability rates β_1.

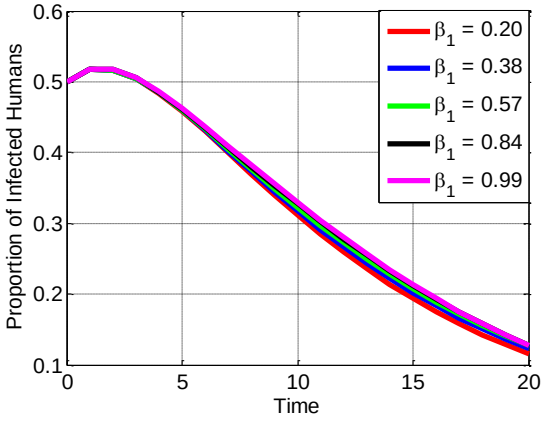

Figure 10: Variation in the proportion of infected human for different transmission probability rates β_1.

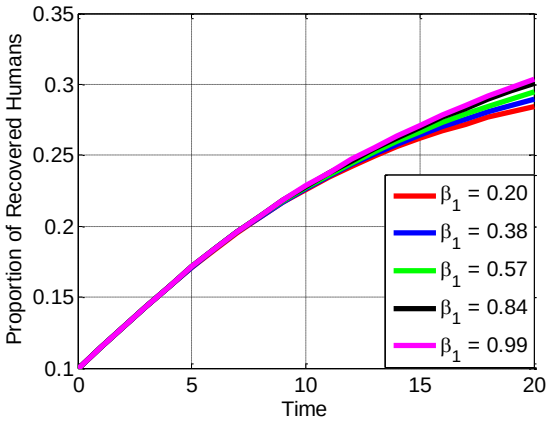

Figure 11: Variation in the proportion of recovered human for different transmission probability rates β_1.

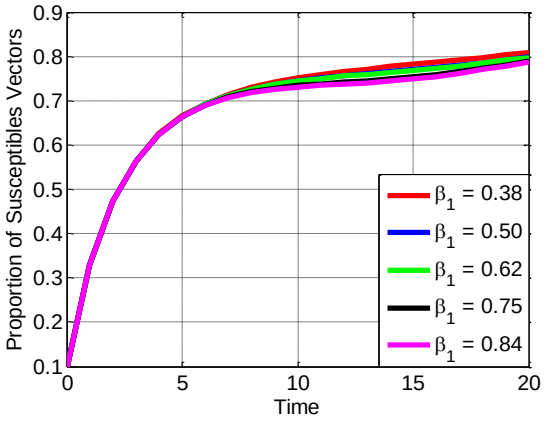

Figure 12: Variation in the proportion of susceptible vector population for different transmission probability rates β_1.

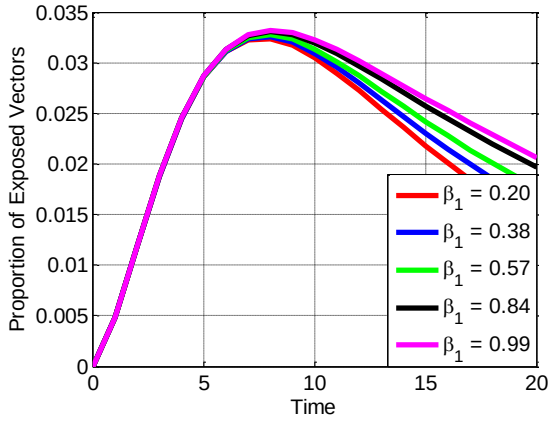

Figure 13: Variation in the proportion of exposed vector population for different transmission probability rates β_1.

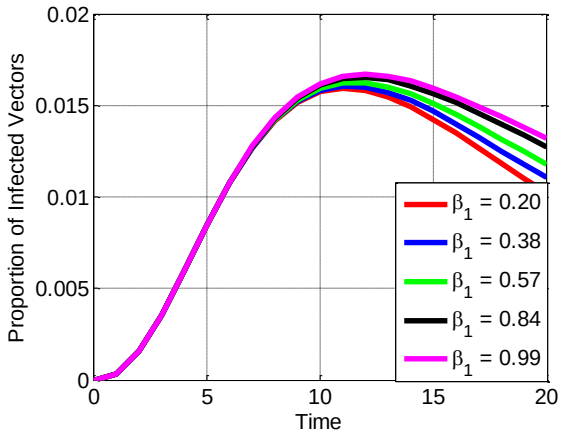

Figure 14: Variation in the proportion of infected vector population for different transmission probability rates β_1.

It is clear from Figures 8, 9, 10, and 11 that a rise in the likelihood of the virus spreading from the vector to the human population causes several noteworthy patterns. The fraction of vulnerable people in the human population falls dramatically as this chance increases and more people contract the disease. The percentages of exposed and infected people in the human population rise simultaneously. In addition, the human recovery rate increases as more people beat the illness.

It is clear from Figure 12 that when the likelihood of viruses spreading from vectors to human populations rises, the percentage of susceptible vectors falls. The likelihood of viruses spreading from vectors to human populations rises simultaneously as the number of exposed and infected vector populations rises. Figures 13 and 14 provide more examples of these tendencies, respectively. The differences between susceptible human, exposed human, infected human, and recovered human populations and susceptible vector, exposed vector, and infected vector populations are shown in Figures 15 through 21. These graphic representations are produced for various distinct values of the likelihood of transmission from the human population to the vector population, which is indicated as β_2.

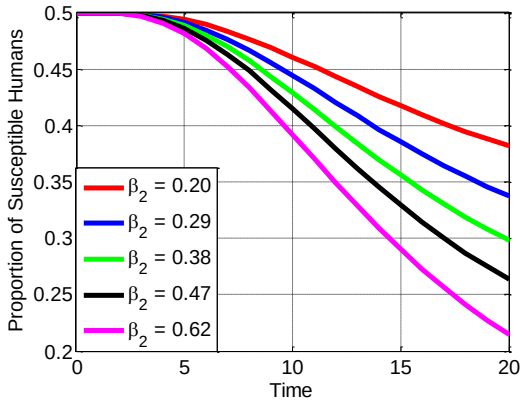

Figure 15: Variation in the proportion of susceptible human population for different transmission probability rates β_2.

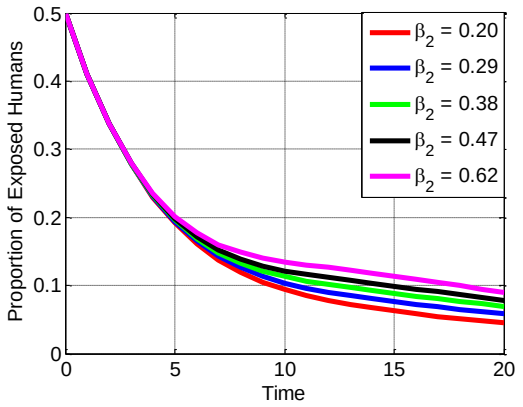

Figure 16: Variation in the proportion of exposed human population for different transmission probability rates β_2.

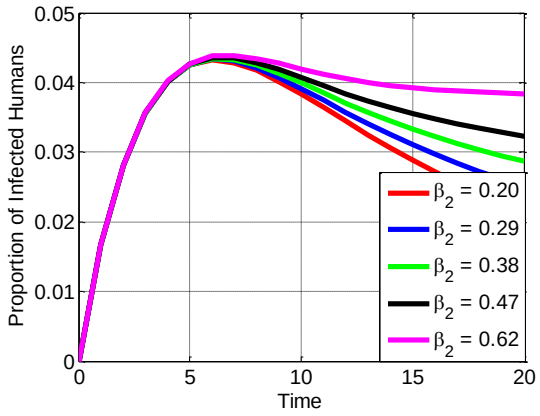

Figure 17: Variation in the proportion of infected human population for different transmission probability rates β_2.

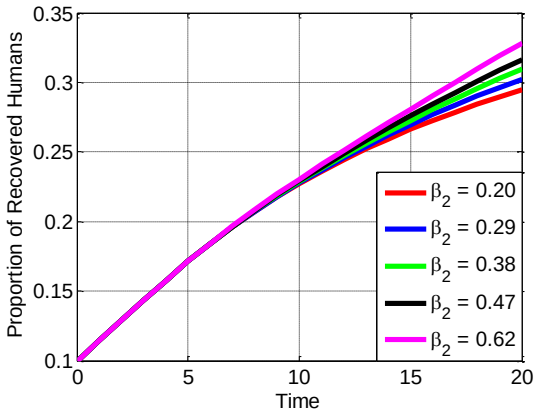

Figure 18: Variation in the proportion of recovered human population for different transmission probability rates β_2.

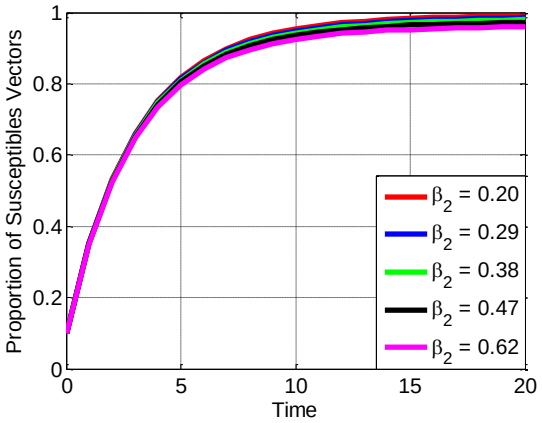

Figure 19: Variation in the proportion of susceptible vector population for different transmission probability rates β_2.

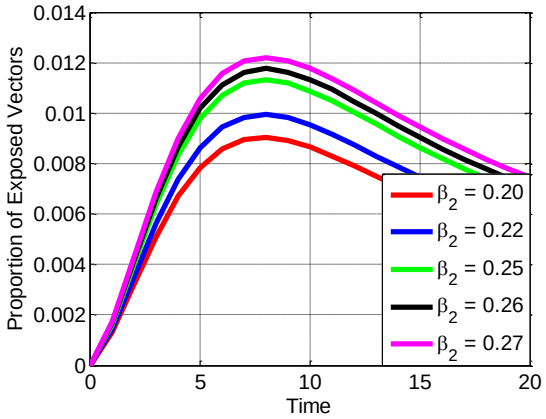

Figure 20: Variation in the proportion of exposed vector population for different transmission probability rates β_2.

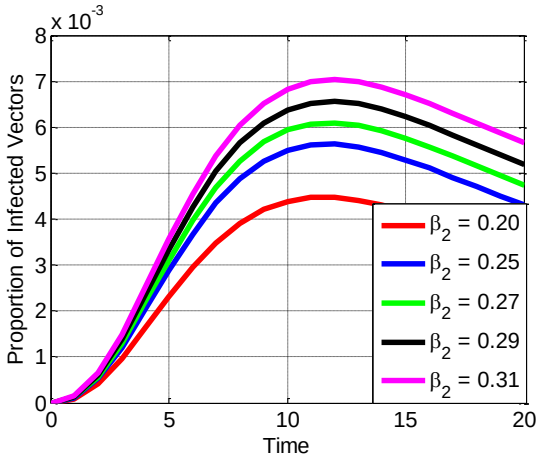

Figure 21: Variation in the proportion of infected vector population for different transmission probability rates β_2.

Figures 15, 16, 17, and 18 clarify that several distinct trends appear as the probability of the virus spreading from humans to vector populations rises. The fraction of vulnerable people in the human population falls dramatically as this likelihood increases because more people contract the disease. The percentages of exposed and sick people in the human population climb simultaneously, and as more people recover from the disease, so does the percentage of the recovered population. We see a similar pattern in Figure 19. The likelihood of a virus spreading from humans to the vector population increases with the fraction of susceptible vectors falling. Figures 20 and 21 further illustrate this relationship, showing that as the risk of the virus spreading from humans to vector populations increases, so does the percentage of exposed and infected vector populations. The distribution of exposed and infected human populations varies, as seen in Figures 22 and 23, respectively. These graphs show these differences across a range of distinct progression rate values, indicated by the symbol ξ_h.

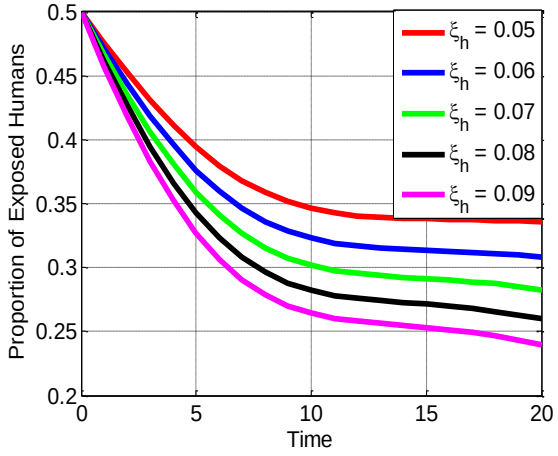

Figure 22: Variation in the proportion of exposed human population for different progression rates ξ_h.

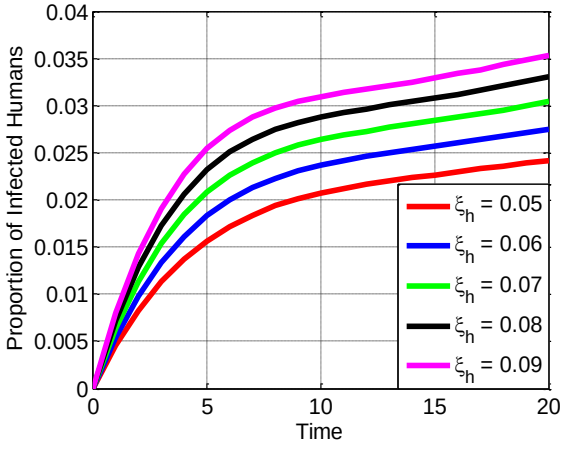

Figure 23: Variation in the proportion of infected human population for different progression rates ξ_h.

In Figure 22, it is observed that the exposed human population decreases as the progression rate, denoted as (ξ_h), increases, whereas with an increase in the progression rate (ξ_h), the proportion of the infected human population also increases, as indicated in Figure 23. Furthermore, Figures 24 and 25 present the variations in the proportions of exposed vector and infected vector populations. These figures demonstrate these variations for a range of different values of the progression rate, referred to as ξ_v.

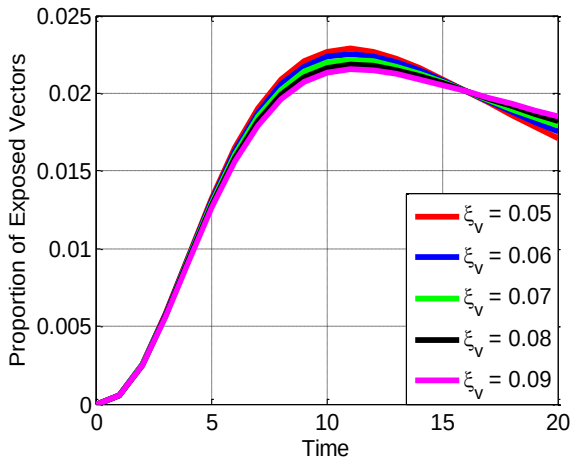

Figure 24: Variation in the proportion of exposed vector population for different progression rates ξ_v.

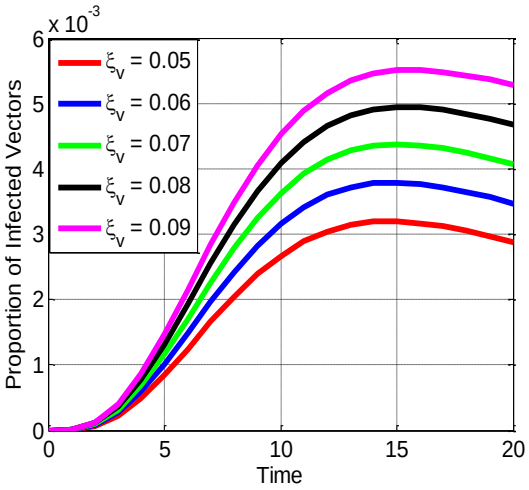

Figure 25: Variation in the proportion of infected vector population for different progression rates ξ_v.

In Figure 24, it is observed that the exposed vector population decreases as the progression rate (ξ_v) increases while the increase of progression rate (ξ_v) the proportion of infected vector population increases as it is indicated in Figure 25.

6. Discussion

In this investigation, we have devised a mathematical framework to depict dengue's transmission dynamics, employing an SEIR model. Our model integrates a logistic function to depict the growth and viability of the mosquito population, which relies on the human populace for sustenance. This approach furnishes a comprehensive comprehension of the intricate interaction between the vector and human populations in disseminating dengue. Through our scrutiny, we have ascertained the fundamental reproduction number, symbolized by R_0, which acts as a pivotal gauge of disease transmission potential. Our observations unveil that the equilibrium devoid of disease retains local stability when R_0 is below one, signifying the feasibility of efficacious control and eradication of the disease under specific circumstances. Conversely, if it surpasses one, the equilibrium void of disease becomes precarious, indicating the potential for persistent transmission within the populace. Furthermore, we thoroughly analyzed stability for both disease-free and endemic equilibria. This scrutiny enabled us to explore the enduring behavior of the system and pinpoint conditions under which the disease may persist or wane. Such discernments are imperative in guiding the formulation of targeted disease control and prevention strategies.

Overall, our research provides valuable contributions to the understanding of dengue transmission dynamics. We have shed light on the complex interplay between the mosquito vector and human host by employing mathematical modelling techniques and considering the logistic human population and exposed class. These insights can assist policymakers, healthcare professionals, and researchers formulate effective measures to combat dengue, mitigate its impact, and ultimately reduce its burden on affected communities. However, it is essential to acknowledge that our model, like any mathematical representation, simplifies the complexity of real-world dynamics. Further research and data collection are necessary to refine and validate the model, incorporating additional factors such as spatial heterogeneity, environmental influences, and intervention strategies. These extensions would contribute to a more comprehensive understanding of dengue transmission and aid in developing targeted interventions to control and prevent this significant public health concern.

7. Conclusion

According to estimates, 40-50% of the world's population is at risk for dengue in tropical, subtropical, and most recently, more temperate regions due to the disease's sharp rise in prevalence in recent decades. Dealing with such a dynamic endemic is challenging and requires scientific resources. If not well and strategically tackled, the disease will eliminate a population. This study demonstrates the potential of mathematical modelling to elucidate the transmission dynamics of dengue. By providing insights into the role of the human population and the interaction with the mosquito vector, our findings contribute to the broader body of knowledge aimed at controlling and mitigating the impact of dengue, ultimately working towards a future with reduced disease prevalence and improved public health outcomes. The list of abbreviations and its meanings as used in this work are presented in Table 4. For future work, we suggest generalizing the studied of SEIR model to show the effect of the infection from other sources other than the mosquito as considered in this study and investigate the effects and stability of the model by introducing additional compartments as recovered and reinfected after contact with infected individual. This study can be extended further to animals that highly fragile with some communicable diseases.

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The authors have contributed equally to the current study and they have all read and agreed with the revised version of the paper.

Conflict of Interest:

The authors declare that they have no known financial or competing interest regarding the authors position and publication of this manuscript.

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Appendix

List of Abbreviations

SEIR	Susceptible, Exposed, Infected, and Recovered
HIV	Human Immunodeficiency Virus
AIDS	Acquired Immunodeficiency Syndrome
DFE	Disease Free Equilibrium
EE	Endemic Equilibrium
GAS	Global Asymptotically Stable
LAS	Locally Asymptotically Stable