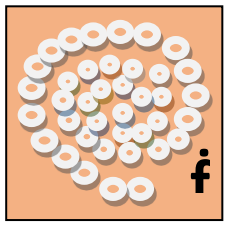

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Sensitivity analysis of Dengue transmission: Impact of integrated pathways and control interventions

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Abstract

Dengue fever remains a significant global health challenge, driven by complex interactions between human and mosquito populations. This study develops and analyzes a compartmental mathematical model to investigate the transmission dynamics of dengue virus, incorporating human and mosquito populations. The model captures key disease progression stages, including susceptible, exposed, infectious, and recovered compartments for humans, and susceptible, exposed, and infectious compartments for mosquitoes. Numerical simulations reveal critical trends in the human and mosquito populations, highlighting peak infection periods and the interplay between host and vector dynamics. Sensitivity analysis using Partial Rank Correlation Coefficients (PRCC) identifies key parameters influencing the basic reproduction number, such as transmission probabilities, mosquito biting rates, and progression rates. The results emphasize the importance of integrated vector management, personal protective measures, and healthcare interventions to mitigate dengue outbreaks. These findings provide actionable insights for designing targeted public health strategies and inform future research to improve dengue control and prevention.

Keywords and phrases: Dengue transmission dynamics; mathematical modeling; sensitivity analysis; basic reproduction number ; vector-borne diseases

1 Introduction

Dengue virus (DENV) continues to be a major public health concern, with an estimated 390 million infections globally each year, and approximately 52% of the world's population residing in areas where DENV transmission occurs [1]. This widespread burden highlights that nearly one-third of the global population is at risk, particularly in tropical and subtropical regions where the virus is endemic [2, 3]. Recent outbreaks in regions such as Spain and France, alongside the growing prevalence of DENV in non-traditional areas, underscore the expanding geographical spread of the disease [4, 5]. As the dynamics of DENV serotypes continue to evolve, understanding their propagation, competition, and coexistence in endemic regions has become essential [6].

The transmission of DENV is primarily mediated by the Aedes aegypti mosquito, with other mosquito species such as Aedes albopictus contributing to its spread [7, 8, 9]. While Aedes aegypti remains the dominant vector in tropical climates, the adaptability of Aedes albopictus allows for transmission in temperate zones, where Aedes aegypti is absent [10]. Recent studies emphasize the importance of integrated vector control strategies to mitigate DENV transmission[11, 12]. Immunity to one serotype does not confer cross-protection, leaving individuals susceptible to severe manifestations such

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as Dengue Hemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS) upon secondary infection [13, 14, 15, 16].

Dengue transmission pathways extend beyond the traditional mosquito-host route. Emerging evidence suggests that vertical transmission in mosquitoes, horizontal transmission within mosquito populations during mating, and host-to-host transmission through sexual contact all play roles in sustaining the virus, even during periods of low mosquito activity [17, 18, 19]. This expanded understanding of transmission routes necessitates more comprehensive models that include these non-vector-based mechanisms [20, 21].

Recent advancements in mathematical modeling have significantly enhanced our understanding of DENV transmission. While traditional models have focused on vector-to-host transmission, newer models incorporate multiple transmission pathways such as vertical and host-to-host transmission, thus offering more detailed insights into the impact of control strategies, including vaccination, mosquito bite prevention, and behavioral interventions [22, 23, 24, 25]. Machine learning techniques and fractional-order models have further improved predictive capabilities, providing more robust frameworks for outbreak prediction and response [26, 27]. Integrated approaches, particularly in co-infection models involving diseases such as malaria, underscore the importance of considering multiple pathogens in disease management strategies [28]. Climate variables, such as temperature and rainfall, are also being increasingly recognized for their role in influencing dengue transmission dynamics [29].

A critical component of these models is the sensitivity analysis (SA), which helps to identify key parameters influencing disease dynamics. Sensitivity analysis is an essential tool for understanding how variations in parameters such as transmission rates, control measures, and human behavior can affect the course of an epidemic. By identifying the most sensitive parameters, researchers can prioritize interventions that are likely to have the greatest impact on controlling the spread of the disease[30, 31] . In the context of DENV, sensitivity analysis can highlight the effectiveness of strategies like vector control, vaccination, and social interventions.

This study focuses on conducting a detailed sensitivity analysis of a comprehensive dengue transmission model that integrates multiple transmission routes, including vector-to-host, host-to-vector, and vertical transmission, along with the effects of behavioral interventions. The novelty of this study lies in its use of a dynamic, multi-route approach that includes both traditional and emerging transmission pathways. By examining how sensitive the model is to variations in key parameters, this research aims to identify the most effective intervention strategies for controlling dengue outbreaks, particularly in endemic regions. The objective is to provide insights into how various interventions such as vector control and behavioral measures can reduce the spread of dengue and inform future public health policies.

2 Methodology/Model formulation

2.1 DENV Mathematical Model Formulation and Analysis

This model is formulated based on the following assumptions;

1. **Transmission Dynamics**: Dengue is transmitted between humans and mosquitoes, human-to-human and mosquito-to-mosquito transmission.

2. **Population Structure**: Humans are divided into **susceptible**, **exposed**, **infectious**, and **recovered** compartments and Mosquitoes are divided into **susceptible**, **exposed**, and **infectious** compartments.

3. **Homogeneous Mixing**: Humans and mosquitoes mix homogeneously, meaning all individuals have an equal probability of contact.

4. **Latent Period**: Both humans and mosquitoes experience a non-infectious incubation period after acquiring the virus.

5. **Immunity**: Recovered humans are assumed to have temporal immunity to reinfection.
6. **Biting and Transmission**: The mosquito biting rate is constant.
7. **Disease-Induced Mortality**: Both humans and mosquitoes experience a small disease-related mortality rate.

2.2 DENV Model Description

The dengue transmission dynamics in this study are represented using a compartmental model that captures the interactions between human and mosquito populations. The model incorporates key biological and epidemiological processes driving dengue transmission. The compartments are divided into two categories: humans and mosquitoes, with each category further subdivided into states that reflect disease progression.

Human Compartments

1. **Susceptible Humans** (T_H): Individuals who are at risk of becoming infected .
2. **Exposed Humans** (D_H): Individuals who have been exposed to dengue virus and are in the incubation period.
3. **Infectious Humans** (G_H): Individuals who have developed symptoms and are capable of transmitting the virus.
4. **Recovered Humans** (Q_H): Individuals who have recovered from the infection and are assumed to have temporary immunity.

Mosquito Compartments

1. **Susceptible Mosquitoes** (T_M): Mosquitoes that are capable of acquiring the virus.
2. **Exposed Mosquitoes** (D_M): Mosquitoes that have exposed to the virus and are in the incubation period, during which they cannot transmit the virus.
3. **Infectious Mosquitoes** (G_M): Mosquitoes that have completed the incubation period and are capable of transmitting the virus .

2.3 DENV Model Equations

The dynamics of the DENV model are described by the following system of ordinary differential equations (ODEs):

$$\left.\begin{array}{ll}
(1) & \frac{dT_H}{dt} = \phi_H + \tau_H Q_H - (\varepsilon_H + \lambda_H)\, T_H \\
(2) & \frac{dD_H}{dt} = \lambda_H T_H - (\varepsilon_H + \sigma_H)\, D_H \\
(3) & \frac{dG_H}{dt} = \sigma_H D_H - (\varepsilon_H + \theta_H + \alpha_H)\, G_H \\
(4) & \frac{dQ_H}{dt} = \alpha_H G_H - (\varepsilon_H + \tau_H)\, Q_H \\
(5) & \frac{dT_M}{dt} = \phi_M - (\varepsilon_M + \lambda_M)\, T_M \\
(6) & \frac{dD_M}{dt} = \lambda_M T_M - (\varepsilon_M + \sigma_M)\, D_M \\
(7) & \frac{dG_M}{dt} = \sigma_M D_M - (\varepsilon_M + \theta_M)\, G_M
\end{array}\right\} \qquad (1)$$

With initial conditions;

$$T_H > 0,\ D_H \geq 0, G_H \geq 0, Q_H \geq 0,\ \ T_M > 0,\ D_M \geq 0\ and\ G_M \geq 0 \qquad (2)$$

And the dengue rate of infectivity for both populations λ_H and $\lambda_M$defined as follows;$\lambda_H = \beta_1 G_H + \beta_2 \mu G_M\ and\ \lambda_M = \gamma_1 \mu G_H + \gamma_2 G_M \qquad (3)$

2.4 DENV Model Variables and Parameter Descriptions

The DENV model variables and parameter descriptions are in Table 1 and Table 2 respectively.

Table 1: Dengue Model Variables Description

Variables	Description	Values	Source
T_H	Dengue Susceptible human population	406,250	[32]
D_H	Dengue Exposed human population	369,150	[32]
G_H	Dengue Infectious human population	156,170	[32]
Q_H	Dengue Recovered human population	20,000	[32]
T_M	Dengue Susceptible Mosquitoes population	40,200	[32]
D_M	Dengue Exposed Mosquitoes population	32,000	[32]
G_M	Dengue Infectious Mosquitoes population	21,200	[32]

Table 2: Dengue Model Parameters Description

Parameters	Description	Value	Source
ϕ_H	Human Population recruitment rate	0.0000406	[32]
ϕ_M	Mosquito Population recruitment rate	0.0005789	[22]
σ_H	Human progression rate from exposed to infectious population.	0.1667	[22]
σ_M	Mosquito progression rate from exposed to infectious population.	0.1428	[22]
α_H	Human progression rate from infectious to recovered population	0.14286	[22]
τ_H	Human progression rate from recovered to susceptible population	0.011	[22]
ε_H	Human population natural death rate	0.0000457	[22]
ε_M	Mosquito population natural death rate	0.03	[22]
θ_H	Human population disease death rate	0.33	[32]
θ_M	Mosquito population disease death rate	0.05	[33]
μ	Mosquito biting rate	0.5	[22]
β_1	Transmission probability rate from human -to-human	0.001	[34]
β_2	Transmission probability rate from mosquito -to-human	0.375	[22]
γ_1	Transmission probability rate from human -to-mosquito	0.375	[22]
γ_2	Transmission probability rate from mosquito -to-mosquito	0.02	[35]

2.5 DENV Model Dynamics and Flow

The flow diagram for the DENV model **Figure 1** illustrates the transitions between compartments for both humans and mosquitoes. Susceptible individuals (T_H,T_M) become exposed (D_H,D_M) after contact with an infectious (human or Mosquito) counterpart (G_H,G_M), eventually progressing to the infectious state (G_H,G_M). Recruitment, death, and recovery processes are incorporated for both populations to maintain population stability.

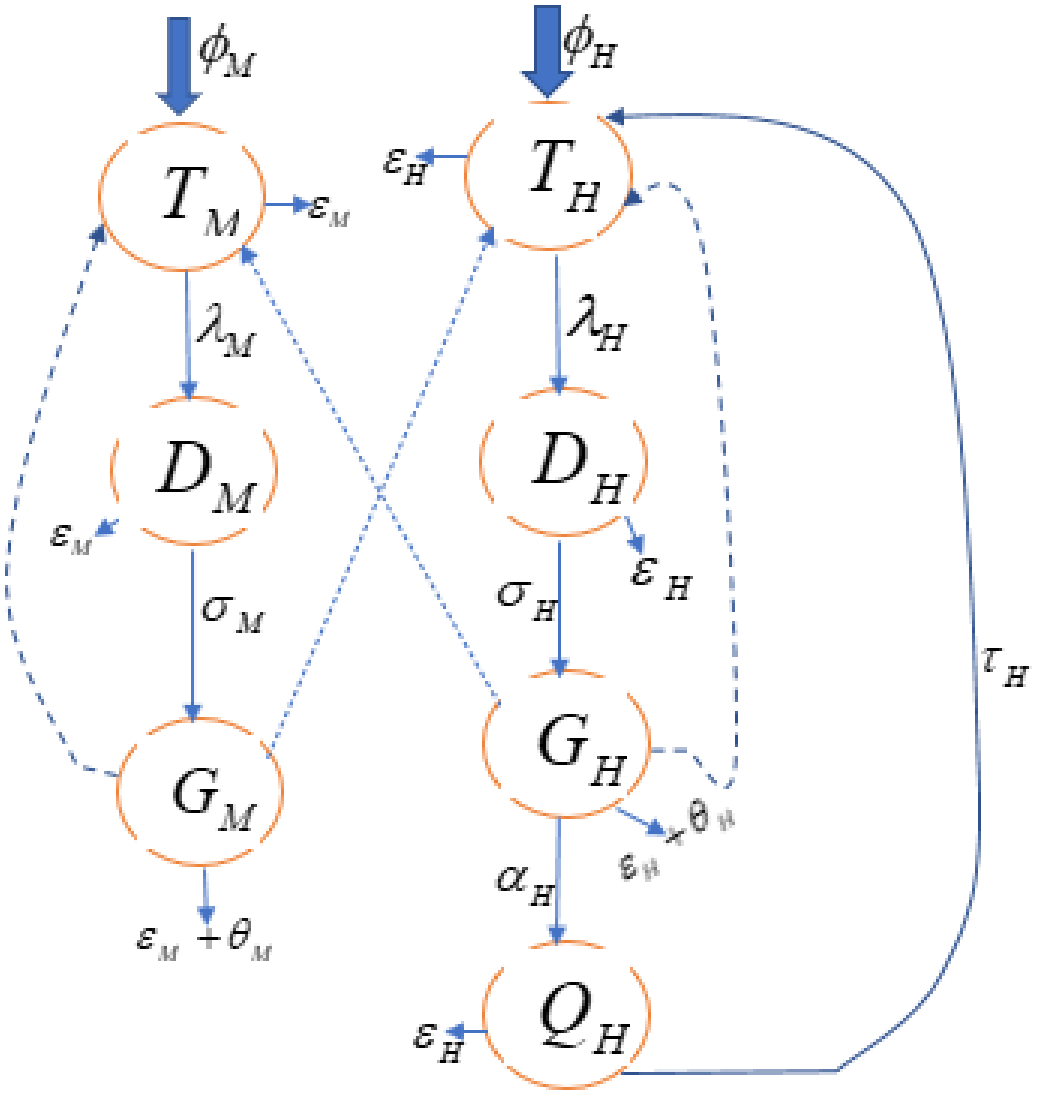

Figure 1: **Dengue Model Flow Diagram**

2.6 Analysis of the DENV Model

This analysis subsection focuses on evaluating the dynamics of the DENV transmission model through mathematical analysis.

2.6.1 Positivity of Solutions

Since the system (1) of the DENV model equations represents human and mosquito populations. We have to consider that the population size cannot be negative. Considering the biological feasible region.

$$\Omega_j = \left\{ (T_H, D_H, G_H, Q_H, T_M, D_M, G_M) \in \mathbb{R}^7_+ : N_H \leq \frac{\phi_H}{\varepsilon_H}, N_M \leq \frac{\phi_M}{\varepsilon_M} \right\}$$

To ensure that all solutions in Ω_j remains in Ω_j for all time;
Whenever $N_H > \frac{\phi_H}{\varepsilon_H}$ then $\frac{dN_H}{dt} < 0$ and whenever $N_M > \frac{\phi_M}{\varepsilon_M}$ then $\frac{dN_M}{dt} < 0$. However, following that $\phi_H - \varepsilon_H N_H$ is a bound of and $\phi_M - \varepsilon_M N_M$ is a bound of $\frac{dN_M}{dt}$ then by recalling the implication of the comparison theorem it can be readily shown that
$N_H(t) \leq \frac{\phi_H}{\varepsilon_H} + \left[N_H(0) - \frac{\phi_H}{\varepsilon_H} \right] e^{\varepsilon_H t}$ and $N_M(t) \leq \frac{\phi_M}{\varepsilon_M} + \left[N_M(0) - \frac{\phi_M}{\varepsilon_M} \right] e^{\varepsilon_M t}$
Obviously, $N_H(t) \leq \frac{\phi_H}{\varepsilon_H}$ if $N_H(0) \leq \frac{\phi_H}{\varepsilon_H}$and $N_M(t) \leq \frac{\phi_M}{\varepsilon_M}$ if $N_M(0) \leq \frac{\phi_M}{\varepsilon_M}$
Hence, we conclude that every solution of the seven equations of the model (1) with initial conditions in Ω_j remains there for all t > 0. Thus, Ω_j is positively invariant and attracting. Therefore, it is sufficient to consider the dynamics of the dengue flow generated by the system (1) in Ω_j and the model can be considered as being epidemiologically and mathematically well posed.

2.6.2 DENV Model Disease Equilibrium points

The DENV disease free equilibrium of the model (1) is given as;
$\zeta^k = \left\{ T_H^k, D_H^k, G_H^k, Q_H^k, T_M^k, D_M^k, G_M^k \right\} = \left\{ \frac{\phi_H}{\varepsilon_H}, 0, 0, 0, \frac{\phi_M}{\varepsilon_M}, 0, 0 \right\}$
while
The DENV disease endemic equilibrium of the model is given as;
$\zeta^m = \{T_H^m, D_H^m, G_H^m, Q_H^m, T_M^m, D_M^m, G_M^m\} \neq \{0,0,0,0,0,0,0\} \quad where$
$T_H^m = \frac{\phi_H + \tau_H Q_H^m}{(\varepsilon_H + \lambda_H)}, D_H^m = \frac{\lambda_H T_H^m}{(\varepsilon_H + \sigma_H)}, G_H^m = \frac{\sigma_H D_H^m}{(\varepsilon_H + \theta_H + \alpha_H)}, Q_H^m = \frac{\alpha_H G_H^m}{(\varepsilon_H + \tau_H)},$
$T_M^m = \frac{\phi_M}{(\varepsilon_M + \lambda_M)}, D_M^m = \frac{\lambda_M T_M^m}{(\varepsilon_M + \sigma_M)} \quad and \quad G_M^m = \frac{\sigma_M D_M^m}{(\varepsilon_M + \theta_M)}$

2.6.3 Dengue Model Basic Reproduction Number (R_0)

The basic reproduction number R_0 here represents the expected number of secondary dengue infection cases produced by a single infected individual in a fully dengue susceptible population. This is a critical threshold parameter that determines whether dengue virus disease can invade and persist in a population. To derive R_0, the next-generation matrix method was applied. The infection matrix (F) and transition matrix (V) for the dengue model are defined as follows:

$$F = \begin{bmatrix} 0 & \beta_1 N_H & 0 & \beta_2 \mu N_M \\ 0 & 0 & 0 & 0 \\ 0 & \gamma_1 \mu N_H & 0 & \gamma_2 N_M \\ 0 & 0 & 0 & 0 \end{bmatrix} \qquad V = \begin{bmatrix} (\varepsilon_H + \sigma_H) & 0 & 0 & 0 \\ -\sigma_H & (\varepsilon_H + \theta_H + \alpha_H) & 0 & 0 \\ 0 & 0 & (\varepsilon_M + \sigma_M) & 0 \\ 0 & 0 & -\sigma_M & (\varepsilon_M + \theta_M) \end{bmatrix}$$

The next-generation matrix is given by:

$$W = FV^{-1}$$

The spectral radius (dominant eigenvalue) of W yields R_0. Simplifying, we obtain:

$$R_0 = \sqrt{\frac{\beta_1 \beta_2 \gamma_1 \gamma_2 \mu^2}{(\varepsilon_H + \sigma_H)(\varepsilon_H + \theta_H + \alpha_H)(\varepsilon_M + \sigma_M)(\varepsilon_M + \theta_M)}}$$

This expression for R_0 highlights the interplay between the human-to-human (β_1) mosquito-to-human (β_2) , human-to-mosquito (γ_1) and mosquito-to-mosquito (γ_2)transmission rates, biting rate (μ), and the dynamics of both human and mosquito populations. The reproduction number increases with

higher biting rates and transmission rates and decreases with higher recovery or death rates. This threshold value serves as a guide for control strategies. For example, reducing transmission rates can effectively lower R_0, helping to interrupt the transmission cycle.

2.6.4 Local Stability of The DENV Model DFE (E^t)

Theorem 1
The DENV disease free equilibrium (DFE) of the system (1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.
Using MATLAB software with the baseline parameter values in **Table 2** we numerically computed the value of R_0 as $R_0 \approx 0.059$. This implies that the DFE is locally asymptotically stable since $R_0 < 1$. This implies that the dengue virus disease will die out of the population in the long run as it will not persist.

2.6.5 Dengue Model Sensitivity Analysis

To assess the local sensitivity of the basic reproduction number R_0 to variations in the model parameters, we computed the partial derivatives of R_0 with respect to each of the key parameters in R_0. These partial derivatives provide insight into how sensitive the basic reproduction number is to changes in each parameter. The computation is as follows;

$$\frac{\partial R_0}{\partial \beta_1} = \frac{\beta_2\gamma_1\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)} \approx 0.00729$$

$$\frac{\partial R_0}{\partial \beta_2} = \frac{\beta_1\gamma_1\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)} \approx 0.00729$$

$$\frac{\partial R_0}{\partial \gamma_1} = \frac{\beta_1\beta_2\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)} \approx 0.00729$$

$$\frac{\partial R_0}{\partial \gamma_2} = \frac{\beta_1\beta_2\gamma_1\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)} \approx 0.00729$$

$$\frac{\partial R_0}{\partial \mu} = \frac{\beta_1\beta_2\gamma_1\gamma_2\mu}{R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)} \approx 0.00729$$

$$\frac{\partial R_0}{\partial \varepsilon_H} = \frac{\beta_1\beta_2\gamma_1\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)^2\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)} \approx -0.00413$$

$$\frac{\partial R_0}{\partial \sigma_H} = \frac{\beta_1\beta_2\gamma_1\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)^2\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)} \approx -0.00413$$

$$\frac{\partial R_0}{\partial \theta_H} = \frac{\beta_1\beta_2\gamma_1\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)^2\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)} \approx -0.00413$$

$$\frac{\partial R_0}{\partial \alpha_H} = \frac{\beta_1\beta_2\gamma_1\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)^2\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)} \approx -0.00413$$

$$\frac{\partial R_0}{\partial \varepsilon_M} = \frac{\beta_1\beta_2\gamma_1\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)^2\left(\varepsilon_M+\theta_M\right)} \approx -0.00413$$

$$\frac{\partial R_0}{\partial \sigma_M} = \frac{\beta_1\beta_2\gamma_1\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)^2\left(\varepsilon_M+\theta_M\right)} \approx -0.00413$$

$$\frac{\partial R_0}{\partial \theta_M} = \frac{\beta_1\beta_2\gamma_1\gamma_2\mu^2}{2R_0\left(\varepsilon_H+\sigma_H\right)\left(\varepsilon_H+\theta_H+\alpha_H\right)\left(\varepsilon_M+\sigma_M\right)\left(\varepsilon_M+\theta_M\right)^2} \approx -0.00413$$

The structure of the model leads to symmetry in the partial derivatives for certain pairs of parameters. Specifically, the positive parameters contribute similarly to the numerator, leading to identical magnitudes of the corresponding partial derivatives. Similarly, the negative parameters contribute similarly to the denominator, leading to similar negative partial derivatives. The positive parameters are critical for the spread of the dengue virus disease while the negative parameters reduce the transmission potential of the dengue virus disease.

Using the MATLAB software and parameter values in Table 2 we performed the global sensitivity analysis using Partial Rank Correlation Coefficients (PRCC) for the model parameters and obtained the result in **Figure 3.**

3 Results

Using MATLAB and the values in Table 1 and Table 2 for our DENV model simulation we obtained the following results;

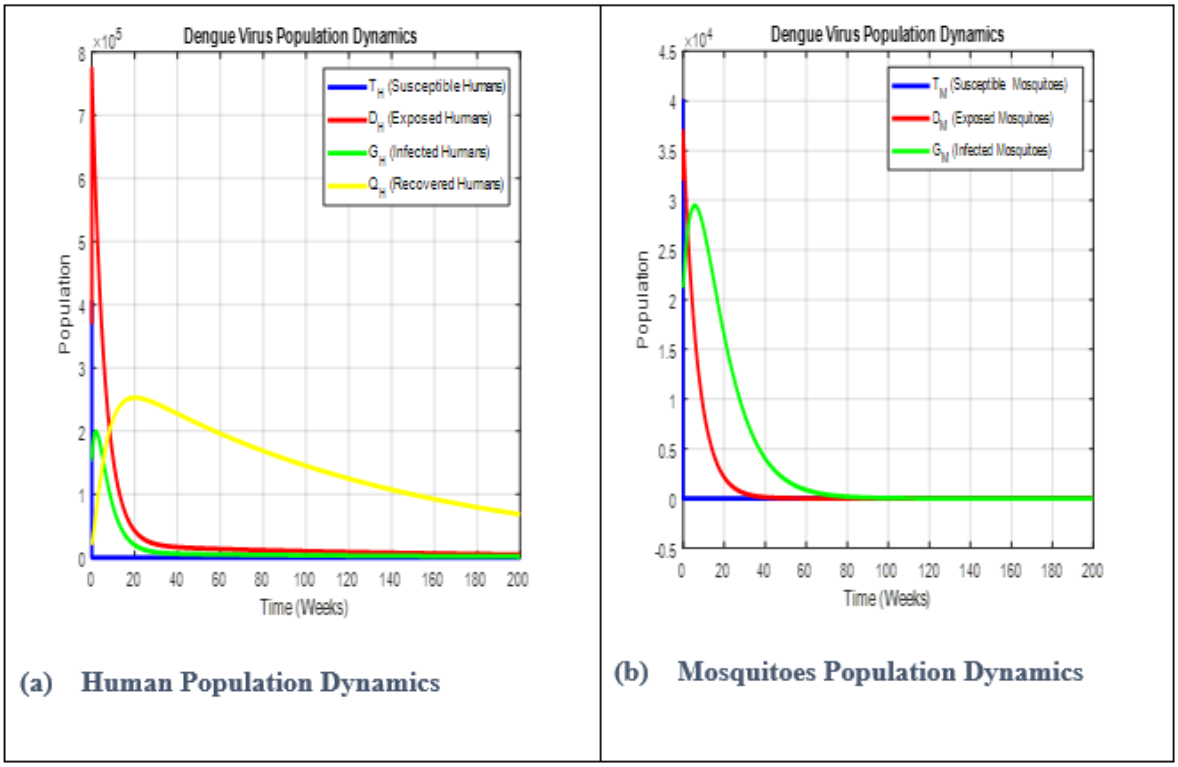

Figure 2: **Dengue Model Flow Diagram**

Figure 2(a) shows the dynamics of the human population under the dengue model. The **Susceptible human's** population decreases continuously over time as individuals become exposed or infected. **Exposed humans'** population initially increases as susceptible individuals become exposed but then decreases due to progression to the infected compartments. **Infectious humans'** population of infectious humans increases after an initial delay, peaks, and then decreases as individuals recover or die. **Recovered humans'** population increases steadily as individuals recover from infection. The decline in susceptible humans shows the effectiveness of transmission pathways converting susceptible into other states. The increase in recovered humans over time reflects the progression of the disease and recovery dynamics, indicating a successful reduction in the infectious pool. The transient peak in exposed and infectious populations suggests control strategies are needed to address this critical time period.

Figure 2(b) depicts the mosquito population dynamics in the dengue model: **Susceptible mosquitoes** decrease over time as mosquitoes become exposed. **Exposed mosquitoes** increase initially as susceptible

mosquitoes encounter infectious humans but decline later as they progress to the infectious state or die. **Infectious mosquitoes** increase after a delay, peak, and eventually decline due to natural or disease-induced death. The decreasing trend in susceptible mosquitoes aligns with the biting and exposure rates modeled. The dynamics of the exposed and infectious mosquitoes closely mirror the human dynamics, as both populations are interdependent through the transmission cycle.

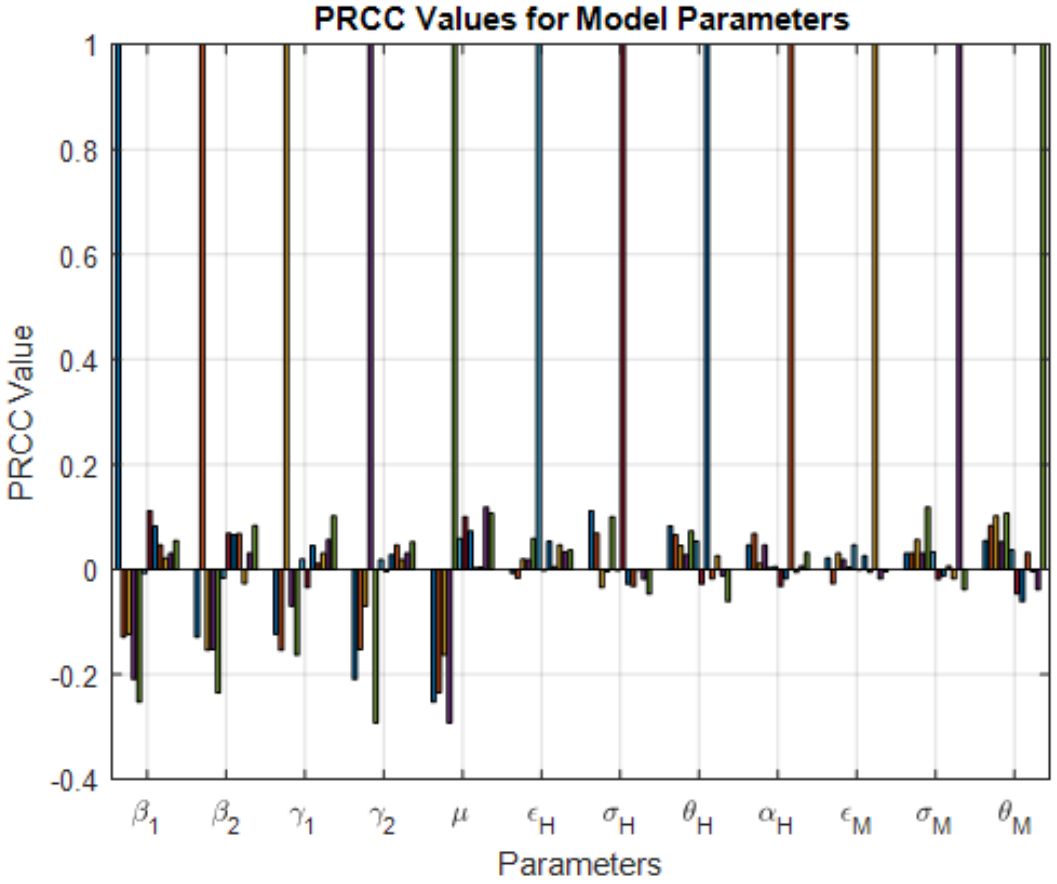

Figure 3: **PCCR DENV Model Global Sensitivity Analysis**

Figure 3 displays the Partial Rank Correlation Coefficients (PRCC) for the model parameters, showing their relative impact on the reproduction number: **Positive PRCC values**: Parameters that increase basic reproduction number when their values are increased (e.g., transmission rates). **Negative PRCC values**: Parameters that decrease basic reproduction number when their values are increased (e.g., recovery or death rates). Thus, Parameters like transmission probabilities and biting rates have a **positive impact**, indicating they are critical drivers of disease spread. Parameters like human natural death rate, progression rates and mosquito death rates show **negative impact**, reflecting their role in reducing the infection potential. The symmetry in the PRCC values for some parameters suggests their equal importance in the transmission process.

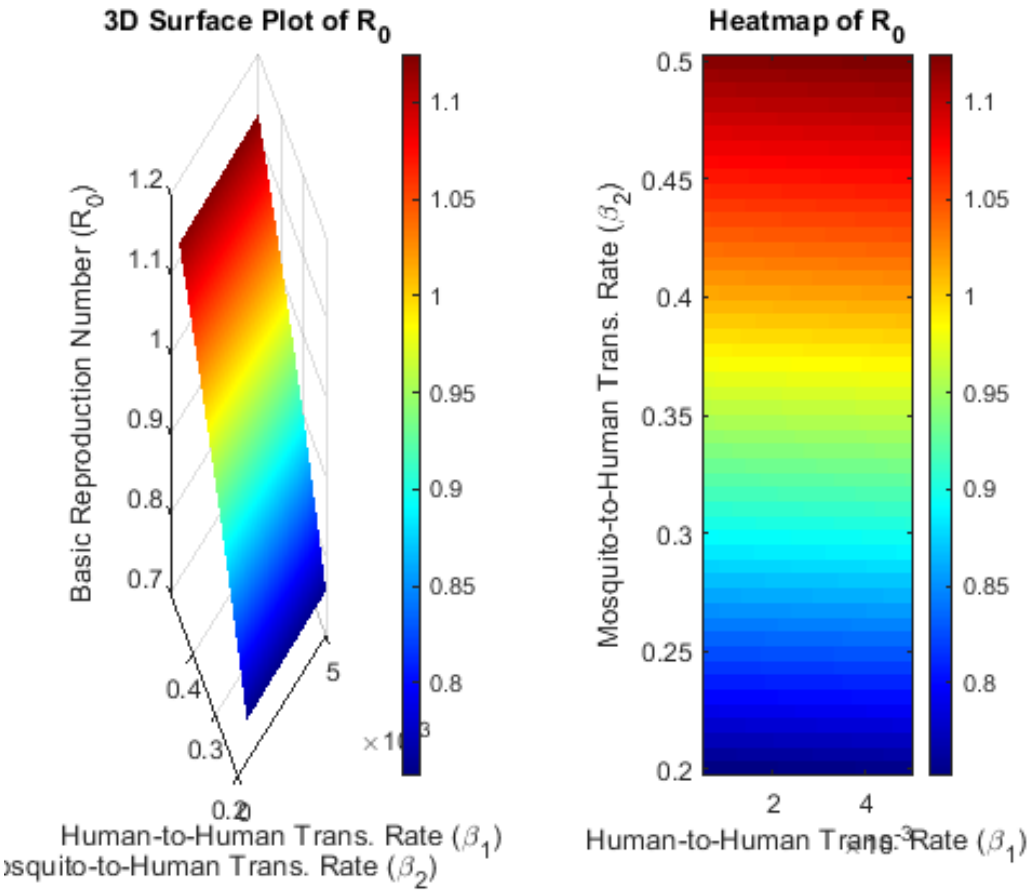

Figure 4: **Compares of 3D Surface Plot and Heatmap of the model R_0 with Human-to-human transmission rate (β_1) and Mosquito-to-human transmission rate (β_2)**

Figure 4 evaluates how changes in the human-to-human and mosquito-to-human transmission probabilities influence R_0.The **3D surface plot** illustrates a proportional relationship: R_0increases as either β_1 or β_2 increases. The **heatmap** provides a top-down view, highlighting areas of high R_0 values, which correspond to combinations of high transmission probabilities. Thus, effective control measures targeting mosquito-to-human transmission (β_2), such as reducing mosquito populations or minimizing contact with humans, will have the most significant impact in reducing R_0

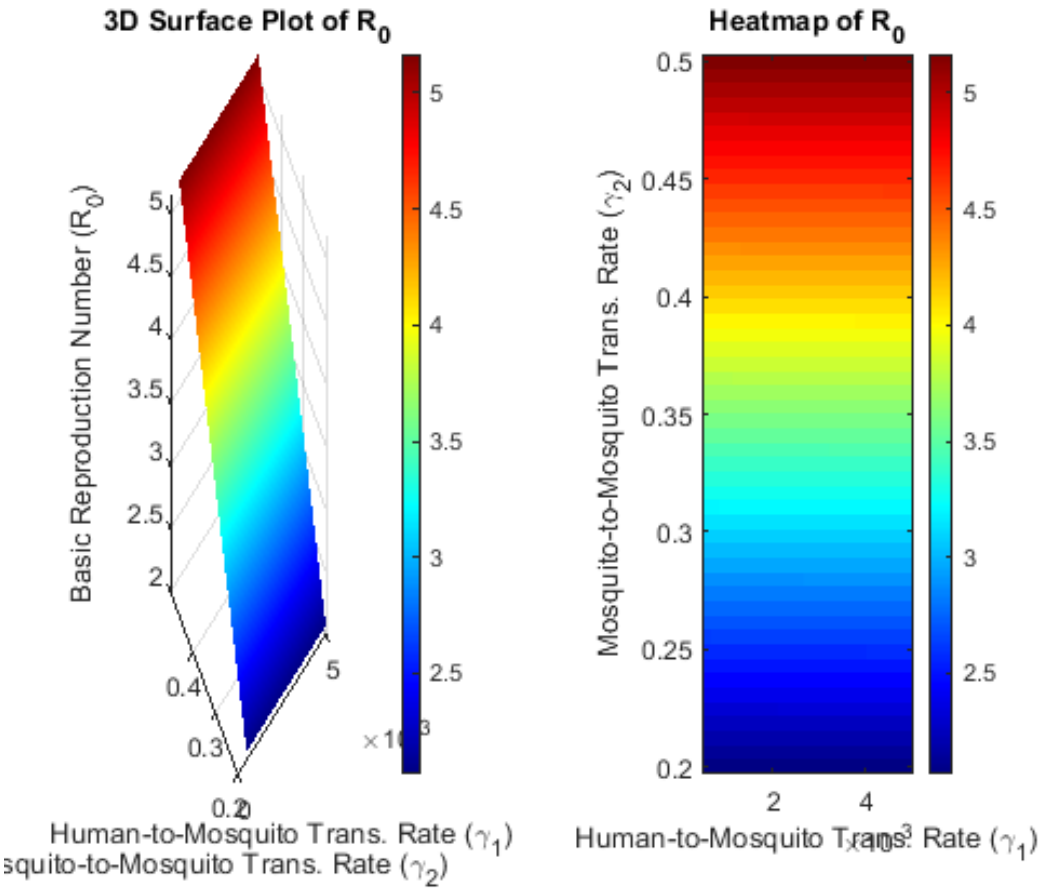

Figure 5: **Compares of 3D Surface Plot and Heatmap of the model R_0 with Human-to-mosquito transmission rate (γ_1) and Mosquito-to-mosquito transmission rate (γ_2)**

Figure 5 explores how R_0 changes based on the probabilities of human-to-mosquito (γ_1) and mosquito-to-mosquito (γ_2) transmission. The surface plot shows that R_0 increases steeply with higher γ_1, indicating that human-to-mosquito transmission is a critical driver. The heatmap reflects areas of high R_0 when both transmission rates are elevated. Thus, targeting γ_1 through measures like treating infectious humans or reducing mosquito contact with infected individuals will significantly lower R_0.

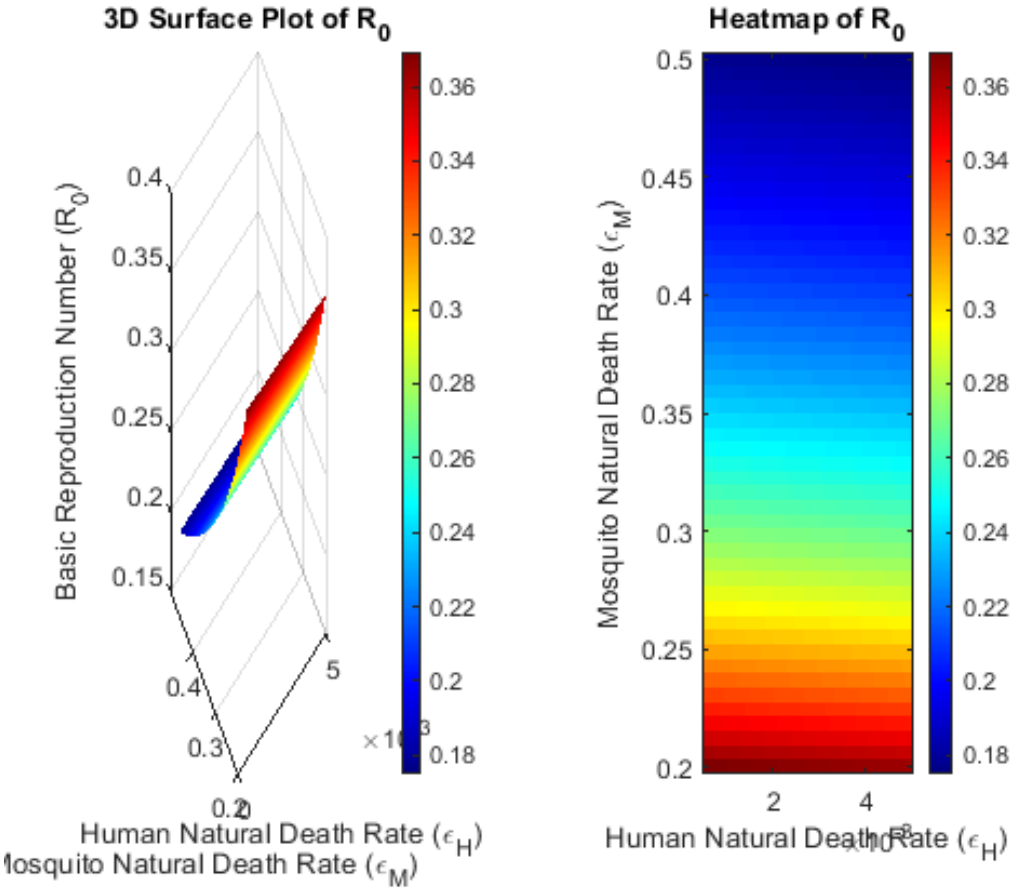

Figure 6: **Compares of 3D Surface Plot and Heatmap of the model R_0 with Human natural rate (ε_H) and Mosquito natural rate (ε_M)**

Figure 6 examines the influence of natural mortality rates on R_0. The 3D plot and heatmap show an **inverse relationship**: R_0 decreases as ε_H or ε_M increases. Higher natural death rates in mosquitoes (ε_M) have a stronger impact on reducing R_0 compared to humans (ε_H). Thus, mosquito control strategies that increase mosquito mortality, such as insecticide spraying or habitat disruption, can significantly reduce dengue transmission.

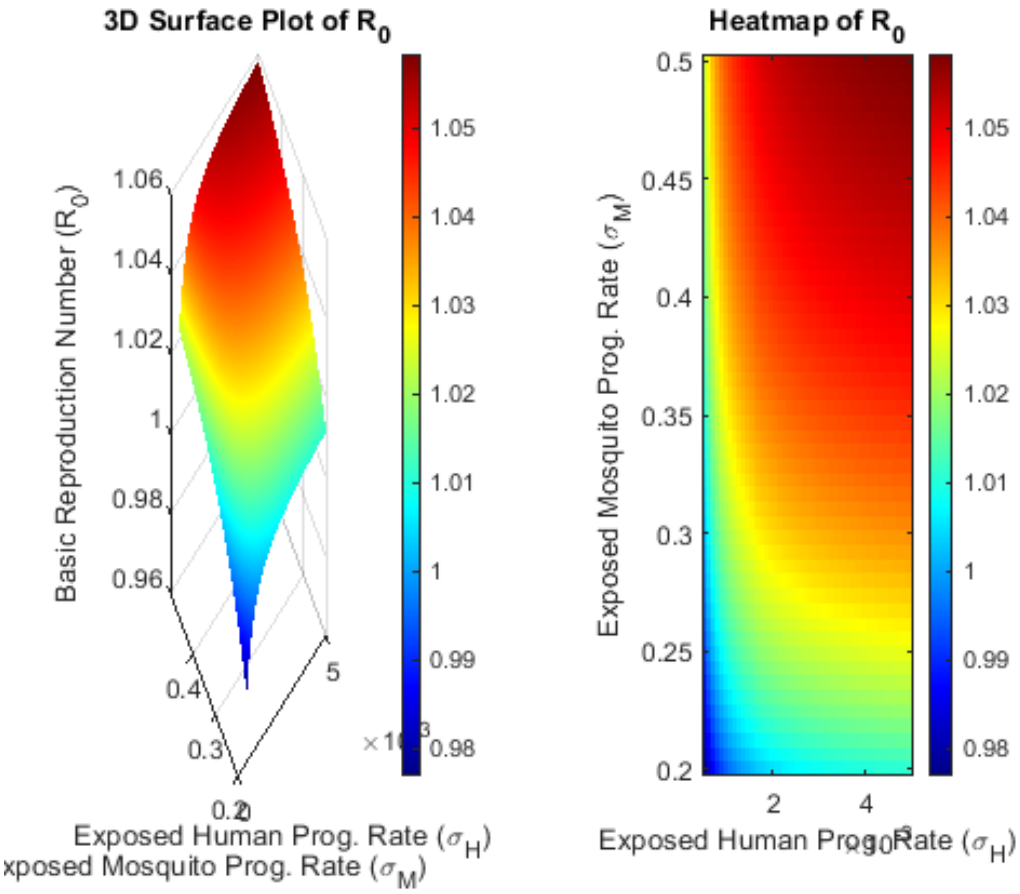

Figure 7: **Compares of 3D Surface Plot and Heatmap of the model R_0 with Exposed Human progression to infectious rate (σ_H) and Exposed Mosquito progression to infectious rate (σ_M)**

Figure 7 evaluates how progression rates from the exposed to infectious states impact R_0. The surface plot shows that R_0 increases with higher progression rates (σ_H and σ_M), as these accelerate the infectious phase. The heatmap highlights areas of elevated R_0 when both progression rates are high. Therefore, slowing progression to the infectious stage through interventions like antiviral treatments or immune-modulating drugs (for humans) can help reduce R_0.

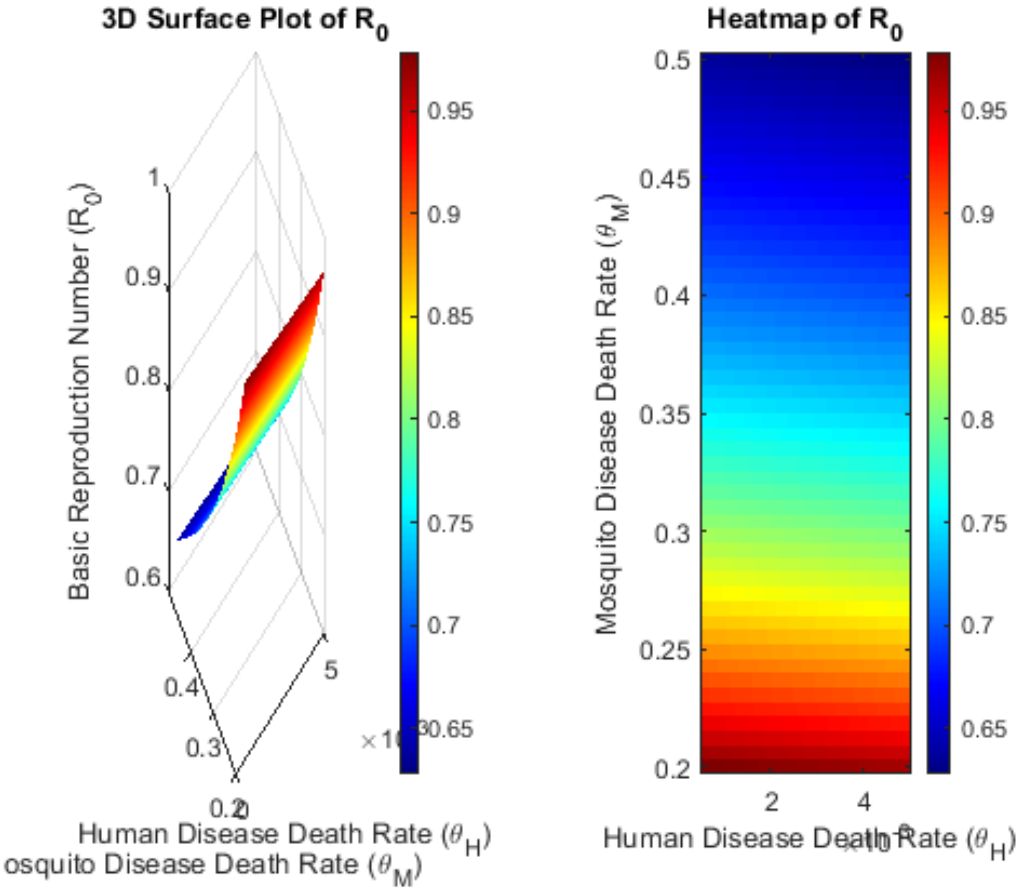

Figure 8: **Compares of 3D Surface Plot and Heatmap of the model R_0 with Human Disease Death rate (θ_H) and Mosquito Disease Death rate (θ_M)**

Figure 8 shows the impact of disease-induced death rates on R_0. The 3D surface plot indicates that R_0 decreases with higher θ_H or θ_M, reflecting the reduced infectious period due to mortality. The heatmap reinforces this negative relationship, with lower R_0 values in regions where death rates are elevated. Thus, while disease-induced death reduces R_0, relying on this alone is not a practical control strategy. Instead, efforts should focus on preventing disease spread altogether.

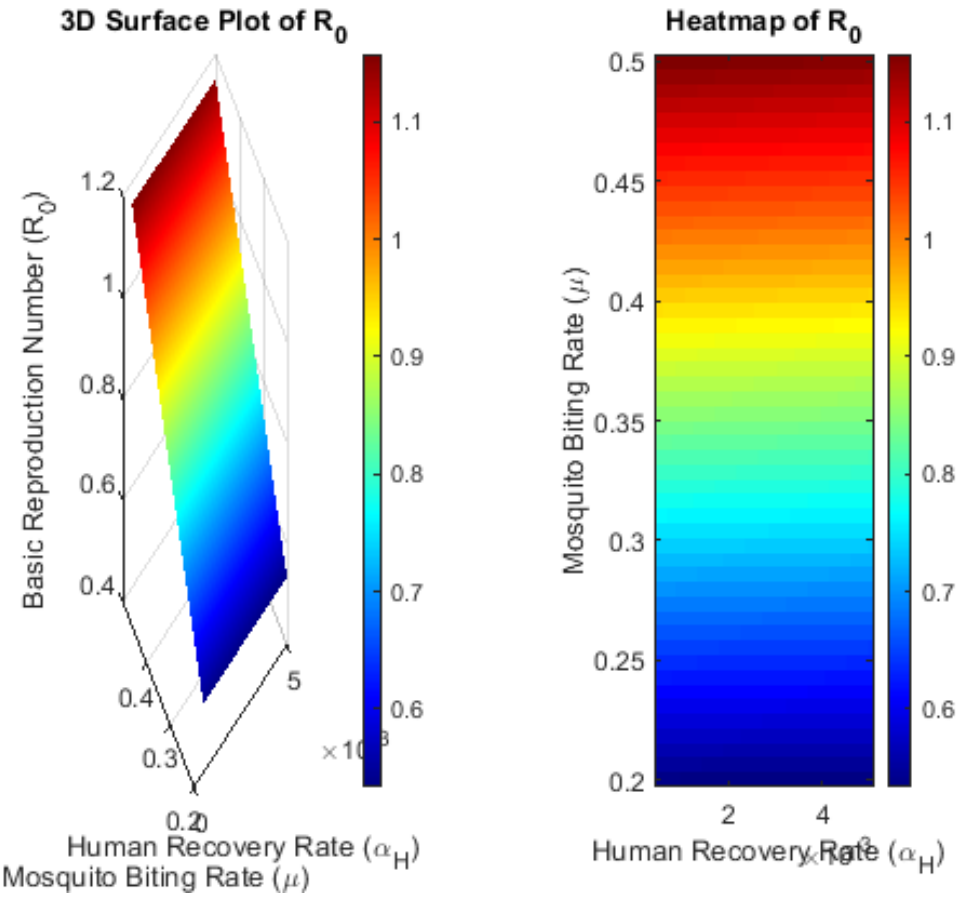

Figure 9: **Compares of 3D Surface Plot and Heatmap of the model R_0 with Human Recovery rate (α_H) and Mosquito Biting rate (μ)**

Figure 9 captures the interplay between human recovery and mosquito biting rates. The 3D surface plot shows that R_0 increases significantly with higher mosquito biting rates (μ), even when human recovery rates (α_H) are high. The heatmap emphasizes the dominance of mosquito biting behavior in driving transmission. Therefore, reducing mosquito biting rates through personal protective measures (e.g., repellents, nets) or reducing mosquito densities is crucial in controlling R_0.

4 Discussions

This study provides a comprehensive analysis of dengue transmission dynamics, focusing on the interplay between human and mosquito populations and the sensitivity of the basic reproduction number (R_0) to key model parameters. The simulation results and sensitivity analysis offer critical insights into the factors driving dengue epidemics and suggest effective intervention strategies to mitigate the disease's spread.

The human and mosquito population dynamics, illustrated in Figures 2–3, highlight the importance of the human-mosquito interaction in amplifying dengue outbreaks. Peaks in exposed and infectious compartments for both humans and mosquitoes occur almost simultaneously, reflecting the bidirectional nature of dengue transmission. These findings underscore the need for synchronized intervention measures targeting both host and vector populations to break the transmission cycle effectively.

The sensitivity analysis, illustrated in Figures 5–10, provides a deeper understanding of the model's behavior and identifies critical parameters influencing R_0. The results in Figures 5 confirm that higher transmission probabilities and biting rates significantly amplify R_0. This emphasizes the importance of minimizing human-mosquito interactions through measures such as the use of insecticide-treated

nets, repellents, and environmental sanitation to reduce mosquito breeding sites. The results in Figures 6 shows that Human-to-mosquito transmission plays a dominant role in driving R_0, indicating that treating infectious humans promptly to limit their contribution to mosquito infections is crucial. Additionally, mosquito-to-mosquito transmission may be relevant in highly vector-dense regions, highlighting the need for sustained vector population management. Figures 7 shows that Increasing natural death rates, particularly for mosquitoes, significantly reduces R_0. This reinforces the importance of vector control strategies that elevate mosquito mortality, such as insecticide spraying, larvicide application, and habitat destruction. Figures 8 shows that faster progression rates from exposed to infectious states in humans and mosquitoes increase R_0, indicating the importance of interventions that delay or prevent infectiousness. For humans, this could involve the use of antivirals or immune-modulating therapies to reduce the likelihood of progression. Figures 9 shows that higher disease-induced mortality in both humans and mosquitoes reduces R_0. While disease-induced mortality naturally limits infectious populations, relying on this effect alone is neither practical nor ethical. Instead, efforts should focus on reducing transmission before individuals become infectious. Figures 10 shows that enhanced human recovery rates effectively reduce R_0. However, even with high recovery rates, elevated mosquito biting rates can sustain dengue transmission. This highlights the importance of reducing mosquito feeding opportunities alongside improving human recovery.
These findings have several critical implications for dengue control which includes:

1. Controlling mosquito populations remains the most effective strategy to reduce R_0. This includes environmental sanitation, insecticide use, and community-based measures like the elimination of breeding sites.

2. Reducing mosquito biting rates through personal protection measures, such as insecticide-treated nets, repellents, and protective clothing, can significantly lower the risk of infection.

3. The importance of recovery rates suggests a need for robust healthcare systems capable of early detection and treatment of dengue cases. Improved access to healthcare and antiviral therapies could reduce disease progression and enhance recovery rates.

4. Treating infectious humans promptly to limit their contribution to mosquito and human infections can play a vital role in reducing R_0. Public health campaigns to promote early diagnosis and treatment can complement mosquito control efforts.

5. As climate and environmental factors influence mosquito populations, sustained surveillance and adaptive management strategies are essential to address changes in transmission dynamics.

This study builds upon and extends existing literature by incorporating emerging transmission pathways and quantifying their contributions to dengue transmission. The sensitivity analysis confirms well-established findings about traditional parameters like biting rates and transmission probabilities while providing novel insights into the importance of non-vector pathways. This positions our work as a valuable addition to the field, with direct implications for public health interventions and policy development.
The strengths of this study lie in its comprehensive modeling approach, incorporating both human and mosquito dynamics and a robust sensitivity analysis. However, certain limitations exist, such as the assumption of homogeneous mixing and constant parameters. Future studies should explore spatial heterogeneity, seasonality, and adaptive intervention strategies to enhance the model's applicability to real-world scenarios.
Therefore, the findings from this study underscore the critical need for integrated control strategies targeting both human and mosquito populations. The sensitivity analysis highlights key drivers of transmission and offers actionable insights for reducing R_0 and mitigating dengue outbreaks. By addressing the identified parameters through targeted interventions, public health authorities can make significant strides toward reducing the burden of dengue globally.

5 Conclusion

This study provides valuable insights into the dynamics of dengue transmission and highlights critical parameters that influence the basic reproduction number (R_0). The results demonstrate that both human and mosquito populations play pivotal roles in amplifying dengue outbreaks, with key factors such as transmission probabilities, biting rates, and progression rates significantly impacting disease spread. The sensitivity analysis reveals that interventions targeting mosquito populations, particularly by reducing biting rates and increasing vector mortality, can be highly effective in mitigating dengue transmission.

Human-focused strategies, such as improving recovery rates and minimizing exposure to mosquitoes, also contribute substantially to reducing R_0. These findings underscore the need for integrated vector management and healthcare preparedness to effectively control dengue epidemics. Furthermore, the identified critical parameters offer practical guidance for designing targeted interventions and public health policies.

While the model provides a robust framework for understanding dengue transmission, its assumptions, such as homogeneous mixing and constant parameters, suggest that further studies incorporating spatial and temporal heterogeneity are warranted. Future research should explore adaptive interventions that account for environmental changes, seasonality, and vector resistance to control measures.

Ultimately, the combined use of mathematical modeling, sensitivity analysis, and evidence-based intervention strategies holds great promise for reducing the global burden of dengue.

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