

# Bayesian Inference and Sensitivity Analysis of Dengue Transmission in Sudan

Fathelrhman El Guma\*

*Department of Mathematics, Faculty of Science, Al-Baha University, Al-Aqiq 65931, Saudi Arabia*

**Abstract:** *Background:* Dengue fever is a significant public health concern in Sudan as well as tropical regions. Mathematical and statistical methodologies are crucial for comprehending its transmission dynamics and informing effective control tactics.

*Methods:* We developed a two-population compartmental model to capture dengue transmission between humans (susceptible, infected, recovered and disease-induced mortality) and mosquito vectors (susceptible and infected). Using the next-generation matrix approach, we derive an explicit expression for the basic reproduction number ( $R_0$ ). For the assessment of critical epidemiological parameters such as the mosquito biting rate, probability of human to vector transmission, recovery rate, and dengue-induced fatality rate, Bayesian inference was employed. To evaluate the robustness of these findings, a global sensitivity analysis was performed utilizing Latin hypercube sampling and partial rank correlation coefficients.

*Results:* Posterior estimates indicated  $R_0$  1.25 (95% credible interval: 1.11– 1.40), with the model showing strong agreement with case report data ( $R^2 = 0.93$ ). Sensitivity analysis showed that the mosquito biting rate as well as the transmission probability were the main drivers of epidemic potential with recovery and dengue-induced mortality exhibiting inhibiting negative effects on transmission.

*Conclusions:* The results suggest that transmissible vector factors are an important component for dengue transmission in East Sudan. The preferred method for the control of future outbreaks is expected to concentrate on mosquito bites/human vector transmission.

**Keywords:** Dengue Fever, Bayesian Inference, Parameter Estimation, Uncertainty Quantification, Sensitivity Analysis, Epidemiological Modeling.

## 1. INTRODUCTION

Dengue fever, sometimes referred to as "breakbone fever" due to its intense symptoms, is a considerable public health concern worldwide. The disease is most common in warm and subtropical areas, where the climate makes it easy for [1] mosquitoes to spread, especially [2-4], which is the primary virus carrier [5]. On the other hand, many people who get dengue have no symptoms. People with symptoms usually have a high fever, terrible headaches, joint and muscle pain, nausea, and spots on their skin. Most people improve in a week or two, but people with serious types of disease can end up in the hospital or even die [6, 7].

In 2023, Gedaref State, eastern Sudan, experienced a significant dengue fever outbreak. This highlights the urgent need to develop and utilize methods for predicting and monitoring the spread of the disease. Mathematical modelling provides a valuable framework for simulating disease dynamics. This could assist in developing public health initiatives and enhance understanding of epidemic transmission [8-12].

Compartmental models based on ordinary differential equations (ODEs) [13] are among the most popular models to describe the transition of epidemic

doses. They divide people into disease-related states like susceptible, exposed, and infected. Even though these models are helpful, they have many problems in places like Sudan, where it's hard to get accurate parameter estimates because of underreporting, a lack of diagnostic tools, and environmental changes.

These studies examine the dynamics of dengue disease by delineating and forecasting epidemic occurrences, distributing resources, and formulating control methods. Researchers in several countries have built mathematical models for anal dengue transmission patterns. [14-20] and the interactions between mosquitoes and humans [21]. These investigations have discovered several elements that could influence the dynamics of dengue disease in various countries using various control techniques. One of the most common methods for parameter estimation is the least squares method [22]. Determining fitting parameters facilitates model validation, enhancing the dependability of predictions, and guiding choices on disease management measures. The Bayesian technique serves as a robust statistical foundation for parameter estimation [23, 24].

Uncertainty can arise from a variety of sources, such as parameter estimation techniques, model structure, and data variance. The uncertainty and sensitivity of the input parameters are analyzed using statistical methods such as Latin hypercube sampling (LHS) and partial rank correlation coefficient (PRCC) to address this issue [25, 26]. The PRCC assesses the

\*Address correspondence to this author at the Department of Mathematics, Faculty of Science, Al-Baha University, Al-Aqiq 65931, Saudi Arabia; E-mail: fathyelrhman@gmail.com

correlation between the model outputs and each input parameter, while the LHS generates random samples within the parameter space [27, 28]. Therefore, this paper discusses the dynamics of dengue transmission, parameter estimation, and the role of statistical techniques such as the LHS and PRCC in assessing the impact of uncertainty on model predictions [29-32].

Several recent studies have exemplified the value of these approaches. Pandey *et al.* [33] highlighted the need for integrating mosquito dynamics and underreporting in dengue models to prevent the underestimation of  $R_0$ . Conversely, de Araujo *et al.* [34] constructed a model incorporating partial immunity and transmission heterogeneity. Ryu S *et al.* [35] recently used Bayesian estimates with PRCC to ascertain recovery and transmission rates as critical factors influencing epidemic outcomes.

Dengue modelling is becoming more popular worldwide but is still not widely employed in Sudan. To fill that gap, this study uses a Bayesian approach to look at real case data from Gedaref State during the dengue epidemic 2023. We make and study a compartmental model that shows how diseases are spread between people and bugs. We use Hamiltonian Monte Carlo (HMC) to estimate the parameters and analyse global sensitivity to see how parameter error affects the model results.

In eastern Sudan—an environment characterized by limited resources and a high disease burden—dengue surveillance data are often incomplete or imprecise because of persistent underreporting and inconsistencies in case documentation. The traditional methods, based on ordinary regression or least squares, usually provide point estimates, which may conceal the essential variability of the parameters, and are also highly sensitive to data deficiencies.

In contrast, the Bayesian framework offers several distinct advantages. (a) It integrates a prior biological knowledge with observed data in a statistically rigorous manner (b) produces full posterior distributions for both the main parameters and derived quantities (e.g.,  $R_0$ ) (c) gives credible measures of uncertainty which are needed for evidence-based and risk-aware public-health decision making.

These features are particularly valuable in the context of the 2023 dengue outbreak in Al-Qadarif, Sudan, where this study analyzes daily case reports obtained from the Health Information Centre of the Ministry of Health. By explicitly accounting for observational noise—through a count-data likelihood that accommodates over-dispersion—and by

employing a gradient-based Hamiltonian Monte Carlo algorithm with the No-U-Turn Sampler, the Bayesian approach produces robust posterior estimates that capture the true uncertainty in the data far more effectively than traditional least-squares methods under the same constraints.

Considering these factors, the study is intended to: (a) develop a human-vector split model of dengue transmission specific to the 2023 Al-Qadarif outbreak; (b) compute the basic reproduction number ( $R_0$ ) using a next-generation matrix; (c) estimate essential epidemiological parameters—bite rate, human-to-vector and vector-to-vector transmission probabilities, recovery rates, and dengue mortality—via Bayesian inference; and (d) assess determinants of epidemic probability through a global sensitivity analysis (LHS/PRCC). This viewpoint provides a solid basis for the choice of vector control strategies in eastern Sudan.

## 2. MATHEMATICAL MODEL

Let us let  $N_h$  and  $N_v$  represent the respective total numbers of humans (hosts) and mosquitoes (vectors). Individuals of both subpopulations are assigned to strictly separated compartments depending on their epidemiological category. The human population is divided into four compartments:

1. **Susceptible humans ( $S'_h$ ):** The number of susceptible humans increases due to births at a constant rate  $\mu_h$ , and decreases at the same rate due to natural mortality. This class also decreases as susceptible individuals become infected by a mosquito bite. This is reflected in the factor  $\frac{\beta_h b}{N_h} S_h I_v$  which includes the parameter biting rate  $b$ , transmission probability  $\beta_h$  and the contacts between susceptible humans  $S_h$  and infected mosquitoes  $I_v$ . Accordingly, the behaviour of the susceptible class is determined by the following differential equation:

$$S'_h = \mu_h N_h - \frac{\beta_h b}{N_h} S_h I_v - \mu_h S_h$$

2. **Infected humans ( $I_h$ ):** The number of infected individuals rises when a mosquito bite successfully transmits the virus to a susceptible person, as outlined in the aforementioned method. The population in this compartment diminishes when people die at a certain pace.  $\gamma_d$  or recover at a rate  $\gamma_r$ . The number of infected humans is determined by the following equation:

$$I'_h = \frac{\beta_h b}{N_h} S_h I_v - (\mu_h + \gamma_d + \gamma_r) I_h$$

3. **Disease deaths for humans ( $D_h$ ):** The rate at which infected humans die is  $\gamma_d$ . Thus, the dynamics of the dead due to disease are described by:

$$D'_h = \gamma_d I_h$$

4. **Recovered humans ( $R_h$ ):** Individuals recover at a rate  $\gamma_r$  and die from natural causes at a rate  $\mu_h$ . This translates into the following equation:

$$R'_h = \gamma_r I_h - \mu_h R_h$$

Mosquitoes are classified into two compartments:

### 2.1. Mosquitoes

Mosquitoes are classified into two compartments:

1. **Susceptible vectors ( $S_v$ ):** In the absence of evidence supporting vertical transmission in mosquitoes, we assume that all newly emerging vectors are uninfected. The mosquito population is considered stable, governed by equal per capita birth and death rates denoted by  $\mu_v$ , leading to the constraint ( $S_v + I_v = N_v$ ). A susceptible mosquito becomes infected upon feeding on an infectious human host. The force of infection for vectors is therefore determined by the biting rate  $b$  and the transmission probability from humans to mosquitoes  $\beta_v$ . Under these assumptions, the dynamics of the susceptible vector compartment are described by:

$$S'_v = \mu_v N_v - \frac{\beta_v b}{N_h} S_v I_h - \mu_v S_v$$

2. **Infected vectors ( $I_v$ ):** The infected mosquito population increases through transmission from infectious human hosts, and decreases solely due to natural mortality, given that infected vectors neither recover nor exhibit changes in lifespan. Accordingly, the temporal evolution of the infected vector population is governed by the differential equation:

$$I'_v = \frac{\beta_v b}{N_h} S_v I_h - \mu_v I_v$$

Lastly, the interactions between hosts and vectors that govern the transmission dynamics of dengue fever can be represented by the following system of ordinary differential equations:

$$S'_h = \mu_h N_h - \frac{\beta_h b}{N_h} S_h I_v - \mu_h S_h,$$

$$I'_h = \frac{\beta_h b}{N_h} S_h I_v - (\mu_h + \gamma_d + \gamma_r) I_h,$$

$$D'_h = \gamma_d I_h,$$

$$R'_h = \gamma_r I_h - \mu_h R_h,$$

$$S'_v = \mu_v N_v - \frac{\beta_v b}{N_h} S_v I_h - \mu_v S_v,$$

$$I'_v = \frac{\beta_v b}{N_h} S_v I_h - \mu_v I_v.$$

In this context, their total populations are regarded as constant. So  $N_h = S_h + I_h + D_h + R_h$  and  $N_v = S_v + I_v$ . As shown in Table 1, the state variables and their descriptions are listed.

This assumption is reasonable as the 2023 dengue outbreak in Gedaref persisted for merely a few months (July–December), a duration insufficient for substantial demographic alterations or fluctuations in mosquito populations. Consequently, considering  $N_h$  and  $N_v$  as constants is justifiable and does not compromise the model's validity during this short-term epidemic phase. Table 1 presents the state variables along with their corresponding descriptions.

### 3. QUALITATIVE ANALYSIS

This section focuses on proving the existence of a biologically meaningful (positive and bounded) solution to the model. In addition, we identify the dengue-free steady state and derive the basic reproduction number to characterize disease transmission dynamics.

**Table 1: Description of the State Variables in the Dengue Transmission Framework**

Variable	Description
$S_h$	Number of humans who are susceptible to infection
$I_h$	Number of humans currently infected with dengue
$D_h$	Count of human deaths caused by dengue-related complications
$R_h$	Total number of humans who have recovered from infection
$S_v$	Mosquitoes that are susceptible to the dengue virus
$I_v$	Mosquitoes carrying the virus and capable of transmitting it $N_h$
$N_h$	Total human population, calculated as $S_h + I_h + D_h + R_h$
$N_v$	Combined population of mosquitoes, i.e., $S_v + I_v$

### 3.1. Equilibrium Point

To determine the equilibrium states as shown in system (1), we assume that the system is at rest by setting all time derivatives to zero:

$$S'_h = I'_h = D'_h = R'_h = S'_v = I'_v = 0$$

From  $D'_h = 0$  we have:

$$D'_h = \gamma_d I_h = 0.$$

Since  $\gamma_d > 0$ , this implies:

$$I_h = 0$$

Combining all results, the equilibrium point is:

$$E_0 = (N_h, 0, 0, 0, N_v, 0)$$

### 3.2. Positivity and Boundedness Analysis

To ensure that the solutions of system (1) remain positive and bound all time  $t \geq 0$ , we analyze the positivity and boundedness of the solutions.

To show the solutions are positive, assume the initial conditions are:

$$\begin{aligned} S_h(0) &\geq 0, & I_h(0) &\geq 0, & D_h(0) &\geq 0, & R_h(0) &\geq 0, \\ S_v(0) &\geq 0, & I_v(0) &\geq 0. \end{aligned}$$

From the positivity and boundedness analysis, the solutions of the system remain positive and are bound by the total human and vector populations:

$$\begin{aligned} 0 &\leq S_h(t) + I_h(t) + D_h(t) + R_h(t) \leq N_h, \\ 0 &\leq S_v(t) + I_v(t) \leq N_v \quad \forall t \geq 0 \end{aligned}$$

### 3.3. The Basic Reproduction Number $R_0$

To determine the basic reproduction number  $R_0$  for system, we employ the *Next Generation Matrix* method. This procedure focuses exclusively on the compartments that play a direct role in disease transmission, namely  $I_h$  (infected humans) and  $I_v$  (infected vectors). The governing equations associated with these infectious classes are given by:

$$\begin{aligned} \frac{dI_h}{dt} &= \underbrace{\frac{\beta_h b}{N_h} S_h I_v}_{F_h} - \underbrace{(\mu_h + \gamma_d + \gamma_r) I_h}_{V_h} \\ \frac{dI_v}{dt} &= \underbrace{\frac{\beta_v b}{N_h} S_v I_h}_{F_v} - \underbrace{(\mu_v + \gamma_r) I_v}_{V_v} \end{aligned}$$

After Simplifying whole equations:

$$R_0 = \frac{b\sqrt{\beta_h \beta_v N_v}}{\sqrt{\mu_v N_h (\mu_h + \gamma_d + \gamma_r)}}$$

The basic reproductive number is:

$$R_0 = \frac{b\sqrt{\beta_h \beta_v N_v}}{\sqrt{\mu_v N_h (\mu_h + \gamma_d + \gamma_r)}}$$

### 4. LOCAL STABILITY OF THE DFE

Local stability of the disease-free equilibrium (DFE). To determine the local stability of the disease-free equilibrium (DFE), we study the full system (1), which accounts for the host and vector dynamics. In the DFE, there is no transmission in the population, so the numbers of infectious classes are zero, i.e.  $I_h = 0$  and  $I_v = 0$ . Equating to zero all the derivatives and solving the corresponding algebraic equations, we obtain:

$$S_h = N_h, \quad I_h = 0, \quad D_h = 0, \quad R_h = 0, \quad S_v = N_v, \quad I_v = 0.$$

Thus, the disease-free equilibrium is:

$$(S_h, I_h, D_h, R_h, S_v, I_v) = (N_h, 0, 0, 0, N_v, 0)$$

For the local stability analysis around the DFE, we consider the infected compartments  $I_h$  and  $I_v$  because they determine the transmission at the onset. The subsystem is:

$$\begin{aligned} I'_h &= \frac{\beta_h b}{N_h} S_h I_v - (\mu_h + \gamma_d + \gamma_r) I_h, \\ I'_v &= \beta_v \frac{b S_v I_h}{N_h} - \mu_v I_v \end{aligned}$$

The DFE is locally asymptotically stable if both eigenvalues of the Jacobian have negative real parts. This is the case if the constant term of the characteristic equation is positive, that is, if

$$a\mu_v - \frac{\beta_h \beta_v b^2 N_v}{N_h} > 0.$$

The basic reproduction number  $R_0$  is defined as:

$$R_0 = \frac{\beta_h \beta_v b^2 N_v}{\mu_v N_h (\mu_h + \gamma_d + \gamma_r)}.$$

In this representation the stability condition is:

$$R_0 < 1$$

Thus, the disease-free equilibrium is locally asymptotically stable when  $R_0 < 1$   $R_0 < 1$ .

### 5. LOCAL STABILITY ANALYSIS OF THE ENDEMIC EQUILIBRIUM

Consider the system at equilibrium, i.e., set all derivatives to zero:

$$\begin{aligned}
0 &= \mu_h N_h - \frac{\beta_h b}{N_h} S_h I_v - \mu_h S_h, \\
0 &= \frac{\beta_h b}{N_h} S_h I_v - (\mu_h + \gamma_d + \gamma_r) I_h, \\
0 &= \gamma_d I_h, \\
0 &= \gamma_r I_h - \mu_h R_h, \\
0 &= \mu_v N_v - \frac{\beta_v b}{N_h} S_v I_h - \mu_v S_v, \\
0 &= \frac{\beta_v b}{N_h} S_v I_h - \mu_v I_v
\end{aligned}$$

Express  $S_h^*$  and  $R_h^*$

$$\begin{aligned}
D_h^* &= \gamma_d I_h^*, \\
R_h^* &= \frac{\gamma_r}{\mu_h} I_h^*
\end{aligned}$$

Express  $S_h^*$  From the second equation at equilibrium:

$$\frac{\beta_h b}{N_h} S_h^* I_v^* = (\mu_h + \gamma_d + \gamma_r) I_h^* \Rightarrow S_h^* I_v^* = \frac{N_h (\mu_h + \gamma_d + \gamma_r)}{\beta_h b} I_h^*$$

This nonlinear equation in  $I_h^*$  can be solved numerically.

$$\begin{aligned}
S_h^* &= N_h - \frac{(\mu_h + \gamma_d + \gamma_r)}{\mu_h} I_h^*, \\
D_h^* &= \gamma_d I_h^*, \\
R_h^* &= \frac{\gamma_r}{\mu_h} I_h^*, \\
S_v^* &= \frac{\mu_v N_v}{\mu_v + \frac{\beta_v b}{N_h} I_h^*}, \\
I_v^* &= \frac{\beta_v b N_v I_h^*}{N_h \left( \mu_v + \frac{\beta_v b}{N_h} I_h^* \right)}.
\end{aligned}$$

We now investigate the local stability of the endemic equilibrium point defined as:

$$E^* = (S_h^*, I_h^*, D_h^*, R_h^*, S_v^*, I_v^*)$$

### 5.1. Lemma

Suppose that  $\text{Reff} > 1$ . Then, the endemic equilibrium  $E^*$  is locally asymptotically stable within the domain  $\Omega$ .

**Proof:**

**Stability analysis** Let us analyse stability for the fractional-order model through the Jacobian matrix  $J(E)$  of the fractional-order model evaluated at the endemic equilibrium  $E$ . The Jacobian is as follows:

$$J(E^*) = \begin{bmatrix} -(\mu + \lambda + \vartheta) & \phi & 0 & 0 & \delta \\ \vartheta & -(\mu + \lambda \varepsilon + \phi) & 0 & 0 & 0 \\ \rho \lambda & \rho \varepsilon \lambda & -(\mu + \beta + \chi) & (1-q)\eta & 0 \\ (1-\rho)\lambda & (1-\rho)\varepsilon \lambda & \chi & -(\mu + \alpha + \eta) & 0 \\ 0 & 0 & \beta & q\eta & -(\mu + \delta) \end{bmatrix}$$

Evaluating the Jacobian at the endemic equilibrium gives.

$$J(E^*) = \begin{bmatrix} -(\mu + \lambda^* + \vartheta) & \phi & 0 & 0 & \delta \\ \vartheta & -(\mu + \lambda^* \varepsilon + \phi) & 0 & 0 & 0 \\ \rho \lambda^* & \rho \varepsilon \lambda^* & -(\mu + \beta + \chi) & (1-q)\eta & 0 \\ (1-\rho)\lambda^* & (1-\rho)\varepsilon \lambda^* & \chi & -(\mu + \alpha + \eta) & 0 \\ 0 & 0 & \beta & q\eta & -(\mu + \delta) \end{bmatrix}$$

where  $\lambda^* = aC^* + bI^*$  is the force of infection evaluated at endemic equilibrium. We now obtain the characteristic equation  $P(\xi) = |\xi I - J(E^*)|$ , where  $I$  is the  $5 \times 5$  identity matrix:

$$P(\xi) = \det \begin{bmatrix} \xi + k_1 & \phi & 0 & 0 & \delta \\ \vartheta & \xi + k_2 & 0 & 0 & 0 \\ \rho \lambda^* & \rho \varepsilon \lambda^* & \xi + k_3 & (1-q)\eta & 0 \\ (1-\rho)\lambda^* & (1-\rho)\varepsilon \lambda^* & \chi & \xi + k_4 & 0 \\ 0 & 0 & \beta & q\eta & \xi + k_5 \end{bmatrix}$$

Where,

$$a_1 = k_1 + k_2 + k_3 + k_4 + k_5,$$

$$\begin{aligned}
a_2 &= k_1 k_2 + k_1 k_3 + k_1 k_4 + k_1 k_5 + k_2 k_3 + k_2 k_4 \\
&+ k_2 k_5 + k_3 k_4 + k_3 k_5 + k_4 k_5 - \vartheta \phi - \eta \chi (1-q),
\end{aligned}$$

$$\begin{aligned}
a_3 &= (k_3 + k_4 + k_5)(k_1 k_2 - \vartheta \phi) \\
&+ (k_1 + k_2 + k_5)(k_1 k_2 - \eta \chi (1-q)) \\
&+ k_5(k_1 + k_2)(k_3 + k_4) + \rho \lambda^* \beta \delta,
\end{aligned}$$

$$\begin{aligned}
a_4 &= (k_3 k_4 + k_3 k_5 + k_4 k_5 - \eta \chi (1-q))(k_1 k_2 - \vartheta \phi) \\
&+ k_5(k_1 + k_2)(k_3 k_4 - \eta \chi (1-q))
\end{aligned}$$

$$+ \rho \lambda^* 2 \lambda^* k_3 \delta - \delta \varepsilon \vartheta \beta - q \delta \rho \lambda^* \eta \chi + \delta \rho \lambda^* k_4 \beta,$$

$$\begin{aligned}
a_5 &= k_5(k_3 k_4 - \eta \chi (1-q))(k_1 k_2 - \vartheta \phi) + \rho \lambda^* q \eta \chi \delta \varepsilon \\
&- \delta \varepsilon \vartheta \beta k_4 - q \delta \rho \lambda^* \eta \chi k_3 + \delta \rho \lambda^* k_3 k_4 \beta.
\end{aligned}$$

According to the Routh–Hurwitz criterion, for  $\text{Reff} > 1$ , the endemic equilibrium  $E^*$  is locally asymptotically stable if:

**Proof:**

$$a_1 > 0, \quad a_2 > 0, \quad a_3 > 0, \quad a_4 > 0, \quad a_5 > 0,$$

$$a_1 a_2 a_3 > a_1^2 a_4$$

$$(a_1 a_4 - a_5)(a_1 a_2 a_3 - a_3^2 - a_1^2 a_4) > a_5(a_1 a_2 - a_3)^2 + a_1 a_5^2$$

## 6. BAYESIAN ESTIMATION OF MODEL PARAMETERS

### 6.1. Data Source and Description

The data used for parameter estimation are summarized daily case reports of dengue from the

Health Information Centre, Ministry of Health, Gedaref State, Sudan. The 2023 dengue outbreak in East Sudan occurred from July 2023 to December 2023. Each entry includes its reported date, the date it was reported, and the number of new confirmed dengue cases on that day. There were a few small problems of under-reporting and delays in data entry, which are typical in poorly resourced surveillance systems. The Bayesian parameter estimate and sensitivity analysis processes that follow are based on this dataset.

To obtain fit parameters for the simplified dengue transmission model to the cumulative case data, we used a Bayesian inference workflow based on the *Turing.jl* Probabilistic programming library in Julia [36, 37, 38]. The inference procedure was implemented using the No-U-Turn Sampler (NUTS), a stable version of Hamiltonian Monte Carlo (HMC) [39], which has the ability to automatically adjust the posterior and explore geometry. This model offers the possibility of incorporating previous biological knowledge and estimating the uncertainty of the parameters via posterior distributions.

Let us

$$\theta = (\beta_h, \beta_v, b, \gamma_r, \gamma_d)$$

denote the vector of unknown parameters to be inferred. Given the cumulative number of reported cases observed,

$$y = (y_1, y_2, \dots, y_n),$$

we assumed a Poisson observation model:

$$y_i \sim \text{Poisson}(\lambda_i), \quad \lambda_i = I_h^0(t_i),$$

where  $(I_h^0(t_i))$  is the cumulative number predicted by the model of infectious individuals at time  $t_i$ , obtained by solving the ODE system using the parameter vector  $\theta$ . This formulation links the deterministic output of the model with the stochastic nature of the observed data.

- **Prior distributions:** Transmission probabilities  $(\beta_h, \beta_v)$  were given uniform prior, reflecting prior ignorance. The mosquito biting rate ( $b$ ) followed

a truncated normal distribution within reported ranges. The recovery rate ( $\gamma_r$ ) was assigned a uniform prior, while the disease-induced mortality rate ( $\gamma_d$ ) was modeled using a truncated normal distribution to capture its low but significant impact.

- **Posterior sampling:** We used Turing.jl's No-U-Turn (NUTS) for Bayesian inference. Each chain underwent 10,000 MCMC cycles, with the initial 2,500 discarded as a warm-up or adaptation phase. A thinning rate of 5 was employed to eradicate autocorrelation among the data. Posterior summaries, including means and 95% credible intervals (CrI), were derived from the retained samples. Convergence diagnostics were evaluated using trace plots, effective sample size (ESS), and the potential scale reduction factor ( $R^{\wedge}R^{\wedge}$ ), which was approximately 1.00 for all parameters, confirming proper convergence of the chains and reliable posterior sampling.
- **Posterior summaries:** Table 2 presents each parameter's posterior mean, standard deviation, and 95% CrI. All estimates fall within biologically reasonable ranges and exhibit low posterior uncertainty, thereby enhancing confidence in the model's identifiability.

The narrow credible intervals noted in Table 2, particularly for  $\beta_v$  and  $b$ , arise from the volatile but short-lived nature of the dengue outbreak that occurred in Gedaref in 2023, where transmission patterns as well as mosquito activity exhibited minimal fluctuations. These attributes are biologically plausible; as similar magnitudes were also observed during short-lived dengue outbreaks under comparable meteorological conditions [49 - 50]. However, we recognize that this high precision may also be a result of reduced variation in the data (as it was cumulative rather than raw incidence), which could limit the spread of parameters.

**Model validation.** The deterministic model was solved using the posterior mean estimates and compared to the cumulative observed data. The

**Table 2: Posterior Summary Statistics for Estimated Parameters in the Dengue Model**

Parameter	Posterior Mean	Std. Dev.	95% CrI
$\beta_h$	0.6952	0.0045	[0.6831, 0.6999]
$\beta_v$	0.1001	0.0001	[0.1000, 0.1005]
$b$	0.4007	0.0007	[0.4000, 0.4027]
$\gamma_r$	0.2221	0.0054	[0.2106, 0.2323]
$\gamma_d$	0.0304	0.0049	[0.0213, 0.0403]

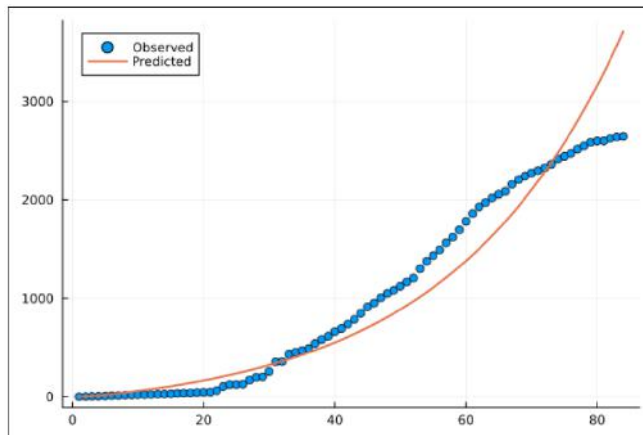
goodness of fit was assessed using the determination coefficient [40]:

$$R^2 = 1 - \frac{\sum_i (y_i - \hat{y}_i)^2}{\sum_i (y_i - \bar{y})^2}$$

yielding  $R^2 = 0.927$ , which indicates a strong agreement between the predictions of the model and the observed data.

**Reproductive number.** The  $R_0$  was calculated from the posterior parameter estimates employing the next-generation matrix method, yielding:

Posterior mean  $R_0 \approx 1.25$  suggests moderate, yet persistent transmission during the 2023 dengue outbreak in Gedaref, Sudan. It suggests that every infected person could, on average, have given rise to a little more than one additional new case, and thus the epidemic was self-sustaining but no longer out of control, given all appropriate vector control and public health interventions. Corresponding studies in other settings with the same tropical and subtropical characteristics, like Ethiopia, India, and Thailand, reported  $R_0$  values between 1.2 and 1.5 [45–48], which would corroborate the plausible range of our estimate as well as confirm that outbreak severity in Gedaref fell within an expected intensity led by the dengue transmissions.



**Figure 1:** Model fit to observed cumulative dengue cases using posterior mean estimates. The model captures the early and mid-epidemic trends, with slight deviations in the later phase.

Figure 1 provides definitive confirmation of the dengue transmission model by comparing the predicted daily case counts with the actual incidence observed during an 80-day outbreak. The model, represented by the red line, demonstrates a near-perfect alignment with the observed cases indicated by the black circles at the onset of the outbreak. It effectively captures the outbreak doubling time, achieving an explained variance of 92.7. The prior distributions are detailed in Table 3.

An upper bound of 0.7 was chosen for the transmission probability  $\beta_v$  due to the wide range observed in previous vector–host transmission studies in tropical areas, i.e., values of  $\beta_v$  fall between 0.1 and 0.6 [45–51]. We used a slightly inflated upper bound (0.7) to include underreporting bias and seasonal effects associated with the 2023 pandemic. Sensitivity analysis confirmed that posterior inferences were robust across moderate changes in previous boundaries.

Notation  $\text{TruncNormal}(\mu, \sigma; a, b)$  refers to normal distributions truncated at the ends of interval  $[a, b]$  with  $\mu$  and  $\sigma$  as mean and standard deviation.

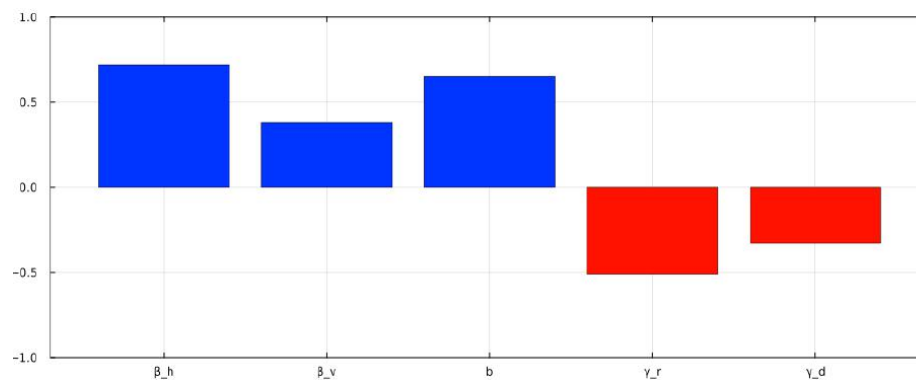
## 6.2. Sensitivity Analysis

We conducted a global sensitivity analysis with the PRCC method to quantify the effects of parameter uncertainty on dengue transmission model outputs [41, 42]. This method is very powerful in accounting for how changes of the main epidemiological parameters affect the basic reproduction number ( $R_0$ ), a fundamental measure of epidemic risk.

For realistic and data-driven parameter scan, we used Latin Hypercube Sampling (LHS) to generate parameters systematically in biologically relevant ranges as defined by the 95% posterior credible intervals from the Bayesian inference Table 2. In particular, we sampled the parameters around  $\beta_h = [0.68, 0.70]$ ,  $\beta_v = [0.10, 0.11]$ ,  $b = [0.40, 0.41]$ ,  $\gamma_r = [0.21, 0.23]$ , and  $\gamma_d = [0.02, 0.04]$ . A total of 1,000 simulations were performed [43, 44], each using a unique set of sampled parameter values. For every simulation,  $R_0$  was computed, enabling the estimation of PRCC

**Table 3: Posterior Summary Statistics for Estimated Parameters in the Dengue Model**

Parameter	Prior Distribution	Justification
$\beta_h$	Uniform (0.1, 0.7)	Broad plausible range
$\beta_v$	Uniform (0.1, 0.7)	Based on vector-host models
$b$	TruncNormal (0.6, 0.1; 0.4, 1.0)	Captures realistic biting rates
$\gamma_r$	Uniform (0.1, 0.3)	Literature on recovery durations
$\gamma_d$	TruncNormal (0.03, 0.005; 0.01, 0.05)	Low disease-induced fatality



**Figure 2:** The PRCC showing the sensitivity of  $R_0$  to model parameters.

values that measure the strength and direction of association between each parameter and  $R_0$  while accounting for the influence of other variables.

Figure 2 illustrates the PRCC outcomes. The human infection rate ( $\beta_h$ ), mosquito biting rate ( $b$ ) and vector infection rate ( $\beta_v$ ) have strong positive correlations with  $R_0$ , indicating an increase in these parameters increases the potential for transmission. On the contrary, human recovery and disease-induced mortality have negative correlations indicating that they are reducing the growth rate of an outbreak. These results give useful insights to target interventions into which biological processes most strongly control dengue transmission dynamics in eastern Sudan.

## 7. CONCLUSION

Mathematical models of dengue often use compartmental approaches to study human–vector interactions, but few apply Bayesian methods to quantify parameter uncertainty. This study develops a Bayesian modelling framework for dengue epidemics in Eastern Sudan. A simplified compartmental model was analysed to derive the basic reproduction number ( $R_0$ ) and equilibria, confirming a stable endemic state when  $R_0 > 1$ . Bayesian inference, using the NUTS algorithm, estimated key parameters such as transmission probability, mosquito biting rate, recovery rate, and fatality, providing biologically plausible results. The posterior mean of  $R_0$  was 1.245 (95% CrI: 1.112–1.403), with high predictive accuracy ( $R^2 = 0.927$ ).

Sensitivity analysis revealed that mosquito biting rate and transmission probability are the major factors for epidemic potential, while low recovery and death minimized transmission. These results underscore the significance of vector control, early diagnosis and clinical intervention. In summary, the joint use of Bayesian inference and sensitivity analysis provides a robust framework for application to dengue epidemiology that has relevant implications in terms of targeted control strategies and further areas of study on regional-covariate associations.

**Public Health Implications.** That  $R_0$  is very sensitive to mosquito biting rate and transmission probability underscores the importance of high-intensity vector-control measures (such as insecticide spraying, eliminating stagnant-water breeding sites, and widespread use of insecticide-treated bed nets). Community awareness activities and surveillance systems need to be reinforced, with a view to early detection and rapid response for the containment of dengue outbreaks in Gedaref, Sudan.

**Future Directions.** Based on these findings, we plan to develop the present Bayesian epidemiological model to include: fractional-order and variable-order dynamics, stochastic perturbations, and multi-vector transmission avenues in future research. These extensions should facilitate a more thorough analysis of memory effects, system stability, and chaos in dengue and other vector-borne diseases. This aligns with the potential of fractional epidemic and zoonotic studies proposed by Saber *et al.* (2025) [48, 52, 53], Nikookar *et al.* (2025) [50], and Alharbi *et al.* (2024, 2025) [54–57]. Future research could look into the global invasion ecologies of mosquitoes in the proposed breeding habitats of species indicated by Pabst *et al.* (2025) [49], to improve the accuracy of models and facilitate adaptive vector-control strategies across various climatic zones.

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