

Mathematical Model for Dengue Transmission: A Comparative Study from the Perspective of Urban and Rural Areas in Bangladesh

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Abstract—Dengue, spread by *Aedes* mosquitoes (vector) is one of the major public health concerns in Bangladesh, with transmission patterns differing notably between urban and rural areas. This study develops a SEIR-SEI compartmental model to capture dengue dynamics, integrating human (Susceptible, Exposed, Infectious, Recovered) and mosquito (Susceptible, Exposed, Infectious) populations. Using daily case of infected and death data from Directorate General of Health Services (DGHS) within the time interval from January 2024 to March 2025, we estimate key parameters, including transmission (β_1, β_2) and recovery (r) rates, via least squares optimization to fit observed trends in urban (city-corporation) and rural (outside-city-corporation) regions. A novel contribution is the comparative analysis of urban-rural transmission dynamics, revealing disparities that guide region specific interventions. Sensitivity analysis of the basic reproduction number (R_0) identifies critical drivers of outbreaks, while stability analysis of the disease-free equilibrium informs control efficacy. Our findings advocate for targeted strategies, such as intensified urban vector control and rural awareness campaigns, to mitigate dengue's spread. This model enhances data-driven public health planning, supporting effective outbreak mitigation in Bangladesh.

Index Terms—Dengue transmission, parameter estimation, sensitivity analysis, urban-rural epidemiology.

I. INTRODUCTION

Dengue has become the fastest-spreading mosquito-borne viral disease worldwide: in 2024 alone, the World Health Organization received reports of more than 14.4 million cases and 11,201 deaths from over 100 countries – the highest global dengue burden ever recorded [1] – against an estimated underlying burden of about 390 million infections annually, of which roughly 96 million manifest clinically, with Asia bearing approximately 70% of the global total [2], [3]. Within the South-East Asia and Western Pacific regions, the 2024 cases were reported primarily from Bangladesh, India, Indonesia, Malaysia, and Viet Nam [1], underscoring that the urban-rural transmission patterns examined here are part of a broader regional and global public health challenge, not an isolated national concern. The *Aedes aegypti* mosquito's life cycle, including egg, larva, pupa, and adult stages, relies on water-filled breeding sites to produce adult mosquitoes that can transmit the dengue virus through bites, with adults remaining infectious for their 2–4 week lifespan [4], [5]. This study employs a normalized SEIR-SEI model, estimating parameters like transmission probabilities (β_1, β_2), recovery rate (r) and other parameters via least squares optimization. Key

contributions include model normalization, urban-rural comparative analysis, and sensitivity analysis of the basic reproduction number (R_0) to identify outbreak drivers. These efforts aim to inform data-driven, region-specific strategies to mitigate dengue's impact.

This dengue remains an acute public health problem in the tropics like Bangladesh, its rapid spread threatening communities and healthcare systems [3]. The disease of four different serotypes (DEN-1 to DEN-4) manifests with symptoms varying from simple fever to severe hemorrhagic ones with recent outbreaks in Bangladesh being attributed to the DEN-3 serotype [6]. Bangladesh's dengue burden has escalated dramatically in recent years: the 2023 epidemic alone recorded more deaths than the cumulative total of the previous two decades, with epidemiological surveillance additionally documenting a marked geographic expansion of transmission beyond Dhaka into southern and rural districts that were previously considered non-endemic [7]. According to Directorate General of Health Services (DGHS) data, Bangladesh reported 103,082 dengue cases and 588 deaths between January 1, 2024, and March 31, 2025, with urban areas registering 38,878 cases and 352 deaths and rural areas 64,204 cases and 236 deaths [8]. This calls for different interventions against different epidemiological scenarios prevailing in urban and rural areas.

As stated by different sources [9], [10], in 2024, urban areas with city-corporation regions, consisting of Dhaka North, Dhaka South, Gazipur, Barishal, Comilla, Rajshahi, Narayanganj, Rangpur, Mymensingh, Sylhet, Chattogram, and Khulna City-Corporation, had a population of 72 million or approximately 41.23% of 174.7 million. Dense urban areas with poor infrastructure become breeding grounds for mosquitoes [11]. Rural Bangladesh, representing 58.77% of the population (approximately 102.7 million), grapples with mosquito breeding grounds dispersed due to farming practices and constrained healthcare resources [12]. These unique regional traits necessitate the development of bespoke mathematical models to guide effective dengue control measures.

Mathematical modeling of dengue has seen important developments since its establishment. Higher-level developments, adapted for dengue from the early models led by Ross for malaria [13], mainly comprised models based on the Susceptible-Infected-Recovered (SIR) framework [14]. A significant step finally occurred when the SEIR model for

humans and the SEI model for mosquitoes were introduced by Esteva and Vargas, who considered the virus's incubation period [15]. Thereafter, models have evolved to incorporate such factors as environment (rain, temperature) [16], spatial dynamics [17], and stochasticity to represent habitat variability [18]. Newton and Reiter [19] further examined vector control, whereas Ferguson et al. [20] studied how multiple serotypes influenced the intensity of outbreaks. However, existing dengue models are largely generic or fitted to other countries' surveillance systems, and application to Bangladesh specifically – let alone a direct urban-rural comparison grounded in Bangladesh's own DGHS case data – remains limited. This gap motivates the present model: rather than relying on transmission parameters borrowed from other settings, we estimate them directly from 456 days of Bangladesh-specific daily case data, allowing the model to reflect the country's actual, distinct urban and rural epidemiological regimes rather than a generic dengue-endemic setting. Building on Pongsumpun's model [21], this study develops a normalized SEIR-SEI framework to compare transmission dynamics in urban and rural Bangladesh, using DGHS data from January 2024 to March 2025.

The paper's organization is as follows: Modeling and assumptions are discussed in Section II. Basic Traits of the Model, including positivity and the invariant region, are studied in Section III. Equilibria and reproduction R_0 follow in Section IV. Stability and parameter estimation are discussed in Section V. Section VI contains sensitivity analysis and numerical simulations. Finally, conclusions and public health directions are followed on in Section VII.

II. BASIC ASSUMPTION AND MODEL FORMULATION

We extend the model by Pongsumpun [21] with normalization and urban-rural parameter estimation to investigate dengue transmission in Bangladesh using a compartmental epidemiological model based on the Susceptible-Exposed-Infected-Recovered (SEIR) framework for humans and the Susceptible-Exposed-Infected (SEI) framework for mosquitoes.

The model assumes the population to be divided into four categories with S_h denoting susceptible individuals, E_h denoting exposed individuals, I_h denoting infected individuals, and R_h denoting individuals who have recovered. The total human population is assumed to remain constant, and can be expressed as the sum $N_h = S_h + E_h + I_h + R_h$. Newly born humans enter the susceptible compartment at rate $\Lambda = \mu_h N_h$. Susceptible humans become exposed when bitten by infected mosquitoes (I_m) and the rate is $\frac{a_h \beta_2}{N_h} S_h I_m$, where a_h is the biting rate of mosquitoes and β_2 is the transmission probability, keeping in view the daytime biting behavior of *Aedes aegypti* [4], [22]. Exposed humans turn infected at the rate γ_h after the latent period of one to eleven days, generally from four to seven days, where they are without symptom [23], [24]. Then, infected humans recover at rate r to join the class of recovery who have a lifetime immunity for that specific dengue serotype, or die at rate θ , whereas the infectious period being 3-7 days

[4], [24]. As for the rate of natural mortality, μ_h , it remains constant for all human classes.

The mosquito population, denoted by $N_m = S_m + E_m + I_m$, is partitioned into three categories: susceptible (S_m), exposed (E_m), and infected (I_m). The recruitment rate of mosquitoes into the vulnerable compartment is $C = \mu_m N_m$. Biting infectious people exposes or infects susceptible mosquitoes at a rate of $\frac{a_h \beta_2}{N_h} S_m I_h$, where β_2 is the likelihood of human-to-mosquito transmission [25]; the rate depends on N_h rather than N_m because a mosquito's bites are distributed across the human population, so the probability that a given bite lands on an infectious human, I_h/N_h , does not depend on the size of the mosquito population itself. After 8–12 days of extrinsic incubation, exposed mosquitoes transition to the condition of infected mosquitoes at a rate γ_m [4]. With no recovery compartment, the infected mosquito dies at a rate of μ_m and stays infectious for around two to four weeks [5]. The natural death rate for each mosquito compartment—susceptible, exposed, and infected—is μ_m .

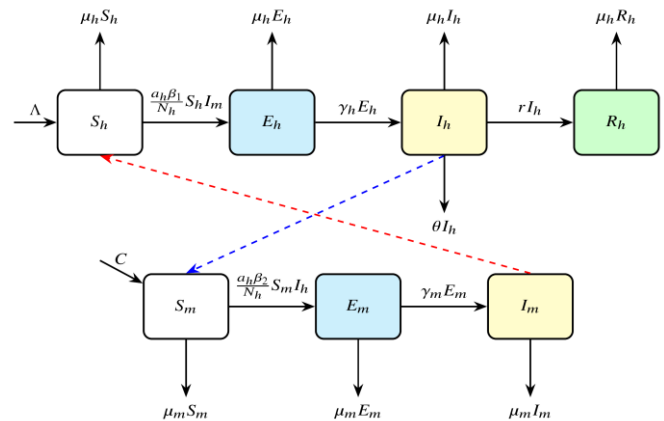


Fig. 1. Compartmental diagram of the SEIR-SEI dengue transmission model, showing human and mosquito compartments. Red dashed arrows indicate mosquito-to-human infection, and blue dashed arrows indicate human-to-mosquito infection.

The parameters of the model, detailed in Table I, represent various biological and epidemiological processes. The rate of human mosquito bites (a_h) and the transmission probabilities (β_1, β_2) control the spread of the infection between species. The rates of progression to the infected state for humans (γ_h) and mosquitoes (γ_m) define the transition from exposed to infected individuals. The human recovery rate (r) and disease-related death rate (θ) describe the consequences of infection, while the natural mortality rates for both humans (μ_h) and mosquitoes (μ_m) represent the inherent death rates in the population.

The model relies on the following key assumptions:

- (i) Constant human and mosquito populations, balancing births and deaths – exact for mosquitoes, and an approximation for humans, whose true population declines slightly during an active outbreak due to disease-induced death (see Lemma 2).
- (ii) Homogeneous mixing within populations, assuming equal contact rates.
- (iii) No vertical transmission in mosquitoes, as it is negligible

for dengue [26].

- (iv) Lifelong serotype-specific immunity for recovered humans [24].
- (v) No seasonal variation in mosquito recruitment, simplifying the model, as in [15].

These assumptions, aligned with [21], ensure a tractable model while capturing essential transmission dynamics.

The model's structure is illustrated in Fig. 1, which shows the human and mosquito compartments and their interactions, with red dashed arrows indicating mosquito-to-human infection and blue dashed arrows indicating human-to-mosquito infection. Following Pongsumpun [21] and Esteva and Vargas [15], we formulate the model with incubation periods for both populations. The system of ordinary differential equations is:

$$\frac{dS_h}{dt} = \Lambda - \frac{a_h \beta_1}{N_h} S_h I_m - \mu_h S_h, \quad (1)$$

$$\frac{dE_h}{dt} = \frac{a_h \beta_1}{N_h} S_h I_m - (\mu_h + \gamma_h) E_h, \quad (2)$$

$$\frac{dI_h}{dt} = \gamma_h E_h - (\mu_h + r + \theta) I_h, \quad (3)$$

$$\frac{dR_h}{dt} = r I_h - \mu_h R_h, \quad (4)$$

$$\frac{dS_m}{dt} = C - \frac{a_h \beta_2}{N_h} S_m I_h - \mu_m S_m, \quad (5)$$

$$\frac{dE_m}{dt} = \frac{a_h \beta_2}{N_h} S_m I_h - (\mu_m + \gamma_m) E_m, \quad (6)$$

$$\frac{dI_m}{dt} = \gamma_m E_m - \mu_m I_m, \quad (7)$$

where Table I provides the relevant parameter definitions.

TABLE I
MODEL PARAMETER DEFINITIONS

Parameter	Description
Λ	Rate of human births in the population.
C	Rate of mosquito recruitment into the environment.
μ_h	Natural mortality rate of humans.
μ_m	Natural mortality rate of mosquitoes.
a_h	Rate of mosquito bites on humans.
β_1	Transmission probability from mosquito to human.
β_2	Transmission probability from human to mosquito.
γ_h	Rate at which humans progress from exposed to infected.
γ_m	Rate at which mosquitoes progress from exposed to infected.
r	Rate of human recovery from infection.
θ	Disease-induced death rate for humans.
ρ	Case-ascertainment fraction: proportion of true new infections captured in DGHS case reports.

To simplify analysis, we normalize the model by expressing compartments as proportions of their respective populations, following [15], [27]. Define the normalized variables:

$$s_h = \frac{S_h}{N_h}, \quad e_h = \frac{E_h}{N_h}, \quad i_h = \frac{I_h}{N_h}, \quad r_h = \frac{R_h}{N_h}, \quad (8)$$

for humans, where N_h is a fixed reference population (assumption (i)), so that $s_h + e_h + i_h + r_h \leq 1$ (with equality only when $\theta = 0$; see Lemma 2), and

$$s_m = \frac{S_m}{N_m}, \quad e_m = \frac{E_m}{N_m}, \quad i_m = \frac{I_m}{N_m}, \quad (9)$$

for mosquitoes, satisfying $s_m + e_m + i_m = 1$. Using $\Lambda = \mu_h N_h$ and $C = \mu_m N_m$, and defining adjusted transmission parameters:

$$\beta_1' = a_h \beta_1 \frac{N_m}{N_h}, \quad \beta_2' = a_h \beta_2, \quad (10)$$

Note that β_1' carries a population-ratio factor N_m/N_h because a mosquito's bites are distributed across the human population, whereas β_2' requires no such factor: a mosquito's own probability of biting an infectious human, I_h/N_h , is already independent of the mosquito population size N_m . The normalized system becomes:

$$\frac{ds_h}{dt} = \mu_h(1 - s_h) - \beta_1' s_h i_m, \quad (11)$$

$$\frac{de_h}{dt} = \beta_1' s_h i_m - (\mu_h + \gamma_h) e_h, \quad (12)$$

$$\frac{di_h}{dt} = \gamma_h e_h - (\mu_h + r + \theta) i_h, \quad (13)$$

$$\frac{dr_h}{dt} = r i_h - \mu_h r_h, \quad (14)$$

$$\frac{ds_m}{dt} = \mu_m(1 - s_m) - \beta_2' s_m i_h, \quad (15)$$

$$\frac{de_m}{dt} = \beta_2' s_m i_h - (\mu_m + \gamma_m) e_m, \quad (16)$$

$$\frac{di_m}{dt} = \gamma_m e_m - \mu_m i_m. \quad (17)$$

This normalized model facilitates analysis of urban and rural transmission dynamics, as explored in Section V.

III. BASIC TRAITS OF THE MODEL

In the process of validating the model mathematically, an analysis is performed to verify its biological consistency, based on the normalized equations (11) to (17). It is shown that within the designated invariant regions, the state variables do not become negative, and the populations remain confined within their respective boundaries, as discussed in Section II.

A. Positivity of the State Variables

Lemma 1. Given the initial conditions $\mathbf{S}_h = \{s_h, e_h, i_h, r_h\} \in \mathbb{R}_{\geq 0}^4$ for the human population, and $\mathbf{S}_m = \{s_m, e_m, i_m\} \in \mathbb{R}_{\geq 0}^3$ for the mosquito population, which satisfy the constraints defined by the model equations (11)–(17), the solution sets $\mathbf{S}_h$ and $\mathbf{S}_m$ will remain in the non-negative domain $\mathbb{R}_{\geq 0}^4$ and $\mathbb{R}_{\geq 0}^3$, respectively, for all $t \geq 0$ within the region Ω .

Proof. Consider the differential equation governing s_h :

$$\frac{ds_h}{dt} = \mu_h(1 - s_h) - \beta_1' s_h i_m.$$

At $s_h = 0$, it follows that $\frac{ds_h}{dt} = \mu_h > 0$, ensuring that $s_h(t)$ cannot decrease below zero. For any initial value $s_h(0) \geq 0$, the recruitment term $\mu_h(1 - s_h)$ guarantees that $s_h(t) \geq 0$. A similar reasoning applies to the other variables. The equations for e_h, i_h, r_h, s_m, e_m , and i_m each contain positive inflows (such as $\beta_1' s_h i_m, \gamma_h e_h, r i_h, \mu_m(1 - s_m), \beta_2' s_m i_h$, and $\gamma_m e_m$) that

ensure their derivatives do not result in negative values. Therefore, the values for all state variables related to humans and mosquitoes will stay non-negative throughout the time span $t \geq 0$, provided the initial conditions satisfy the model's constraints. This behavior is ensured within the region Ω , as explained in Section III-B.

B. Invariant Regions

Lemma 2. The normalized human population satisfies $s_h + e_h + i_h + r_h \leq 1$ for all $t \geq 0$ whenever this holds at $t = 0$, and the normalized mosquito population satisfies $s_m + e_m + i_m = 1$ for all $t \geq 0$. Consequently $\Omega = \Omega_h \times \Omega_m$ is positively invariant, where $\Omega_h = \{(s_h, e_h, i_h, r_h) \in \mathbb{R}_{\geq 0}^4 \mid s_h + e_h + i_h + r_h \leq 1\}$ and $\Omega_m = \{(s_m, e_m, i_m) \in \mathbb{R}_{\geq 0}^3 \mid s_m + e_m + i_m = 1\}$.

Proof. Summing (11)–(14) and writing $H = s_h + e_h + i_h + r_h$:

$$\begin{aligned} \frac{dH}{dt} &= \mu_h(1 - s_h) - \beta_1' s_h i_m + \beta_1' s_h i_m \\ &\quad - (\mu_h + \gamma_h) e_h + \gamma_h e_h - (\mu_h + r + \theta) i_h + r i_h - \mu_h r_h \\ &= \mu_h(1 - H) - \theta i_h. \end{aligned} \quad (18)$$

Unlike the mosquito population (below), this does *not* vanish identically: the disease-induced death term $-\theta i_h$ is a genuine additional outflow, since human births enter at the constant rate $\Lambda = \mu_h N_h$ (assumption (i)) while disease deaths remove individuals beyond the natural death rate μ_h already accounted for in each compartment. Consequently $H \equiv 1$ does *not* hold once $\theta > 0$ and $i_h > 0$. However, $H \leq 1$ is still positively invariant: at the boundary $H = 1$, $\frac{dH}{dt} = -\theta i_h \leq 0$ for all $i_h \geq 0$, so H cannot increase past 1, and $H(0) \leq 1$ implies $H(t) \leq 1$ for all $t \geq 0$. Since $e_h, i_h, r_h \geq 0$ (Lemma 1), this gives $s_h = H - e_h - i_h - r_h \leq H \leq 1$ throughout Ω – the property used in Theorem 1 below. (Equivalently, $s_h \leq 1$ follows directly from (11) alone: at $s_h = 1$, $\dot{s}_h = -\beta_1' s_h i_m \leq 0$.)

For the non-normalized human population, we have:

$$\frac{dN_h}{dt} = \Lambda - \mu_h N_h - \theta I_h \leq \Lambda - \mu_h N_h, \quad (19)$$

implying $N_h \leq \frac{\Lambda}{\mu_h}$ with $\Lambda = \mu_h N_h(0)$ (assumption (i)); the population is bounded but, as just shown, only approximately constant, declining slightly during an active outbreak due to disease mortality. Summing (15)–(17), we get $\frac{d}{dt}(s_m + e_m + i_m) = \mu_m(1 - (s_m + e_m + i_m))$, since mosquitoes have no disease-induced death term in this model; with $s_m + e_m + i_m = 1$ at $t = 0$, uniqueness of solutions gives $s_m + e_m + i_m = 1$ for all $t \geq 0$.

IV. MODEL ANALYSIS

This section investigates the equilibrium dynamics and transmission potential of the normalized dengue model (11)–(17), providing insights into disease persistence and control strategies.

A. Infection-Free Equilibrium State

To determine the disease-free equilibrium, the system in its

normalized form, as described by equations (11)–(17), is set to zero, under the assumption of no infection ($e_h = i_h = e_m = i_m = 0$). Solving $\mu_h(1 - s_h) = 0$ and $\mu_m(1 - s_m) = 0$ yields $s_h = 1$ and $s_m = 1$; meanwhile, $r_h = 0$ follows from $r i_h - \mu_h r_h = 0$ with $i_h = 0$, resulting in $E_0 = (s_h, e_h, i_h, r_h, s_m, e_m, i_m) = (1, 0, 0, 0, 1, 0, 0)$. This equilibrium points to a population without dengue, in which all humans and mosquitoes are susceptible. Therefore, the other equilibrium stands as our starting point in terms of eradicating the disease, and the reproduction number is computed from it in Section IV-B.

B. Reproduction Number

Based on the linear stability analysis of the disease-free equilibrium (DFE), denoted as $E_0 = (1, 0, 0, 0, 1, 0, 0)$, the R_0 (The Basic Reproduction Number) acts as a threshold, determining whether the DFE is stable or unstable. It can be thought of as the average number of secondary infections caused by an infectious individual in a population that is completely susceptible [28]. For the infected compartments (e_h, i_h, e_m, i_m), the matrices F and V are created; the DFE evaluates F for new infections and V for transitions like progression, recovery, and mortality-recovery. These are provided by:

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_1' \\ 0 & 0 & 0 & 0 \\ 0 & \beta_2' & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \gamma_h + \mu_h & 0 & 0 & 0 \\ -\gamma_h & r + \theta + \mu_h & 0 & 0 \\ 0 & 0 & \gamma_m + \mu_m & 0 \\ 0 & 0 & -\gamma_m & \mu_m \end{pmatrix}, \quad (20)$$

evaluated at $s_h = 1, s_m = 1$. The inverse V^{-1} is:

$$V^{-1} = \begin{pmatrix} \frac{1}{\gamma_h + \mu_h} & 0 & 0 & 0 \\ \frac{\gamma_h}{(\gamma_h + \mu_h)(r + \theta + \mu_h)} & \frac{1}{r + \theta + \mu_h} & 0 & 0 \\ 0 & 0 & \frac{1}{\gamma_m + \mu_m} & 0 \\ 0 & 0 & \frac{\gamma_m}{(\gamma_m + \mu_m)\mu_m} & \frac{1}{\mu_m} \end{pmatrix}. \quad (21)$$

The next-generation matrix FV^{-1} is:

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_1' \gamma_m}{(\gamma_m + \mu_m)\mu_m} & \frac{\beta_1'}{\mu_m} \\ 0 & 0 & 0 & 0 \\ \frac{\beta_2' \gamma_h}{(\gamma_h + \mu_h)(r + \theta + \mu_h)} & \frac{\beta_2'}{r + \theta + \mu_h} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}. \quad (22)$$

The spectral radius of FV^{-1} yields:

$$R_0 = \rho(FV^{-1}) = \sqrt{\frac{\beta_1' \beta_2' \gamma_h \gamma_m}{(\gamma_h + \mu_h)(r + \theta + \mu_h)(\gamma_m + \mu_m)\mu_m}}, \quad \mathcal{R}_0 = R_0^2. \quad (23)$$

R_0 measures the basic reproduction, which is the average number of secondary infections that serve either an infected human or mosquito in the completely susceptible group [28]. It takes into account mosquito biting rates and transmission probabilities from humans to mosquitoes and vice versa.

C. Endemic Steady State

The steady state where the disease continues to circulate

within both the human and mosquito compartments is referred to as the endemic steady state, represented as $E^* = (s_h^*, e_h^*, i_h^*, r_h^*, s_m^*, e_m^*, i_m^*)$. The following results are derived by solving the system (11)–(17) for zero derivatives using the Maple program:

$$s_h^* = \frac{(\gamma_h + \mu_h)(r + \theta + \mu_h)\mu_m + \gamma_h\mu_h\beta_2'}{\mathcal{R}_0(\gamma_h + \mu_h)(r + \theta + \mu_h)\mu_m + \gamma_h\mu_h\beta_2'} \quad (24)$$

$$e_h^* = \frac{\mu_m(r + \theta + \mu_h)(\mathcal{R}_0 - 1)\mu_h}{\mathcal{R}_0(\gamma_h + \mu_h)(r + \theta + \mu_h)\mu_m + \gamma_h\mu_h\beta_2'} \quad (25)$$

$$i_h^* = \frac{\gamma_h(\mathcal{R}_0 - 1)\mu_h\mu_m}{\mathcal{R}_0(\gamma_h + \mu_h)(r + \theta + \mu_h)\mu_m + \gamma_h\mu_h\beta_2'} \quad (26)$$

$$r_h^* = \frac{\gamma_h(\mathcal{R}_0 - 1)\mu_m r}{\mathcal{R}_0(\gamma_h + \mu_h)(r + \theta + \mu_h)\mu_m + \gamma_h\mu_h\beta_2'} \quad (27)$$

$$s_m^* = \frac{\mathcal{R}_0(\gamma_h + \mu_h)(r + \theta + \mu_h)\mu_m + \gamma_h\mu_h\beta_2'}{[(\gamma_h + \mu_h)(r + \theta + \mu_h)\mu_m + \gamma_h\mu_h\beta_2']\mathcal{R}_0} \quad (28)$$

$$e_m^* = \frac{\gamma_h\beta_2'(\mathcal{R}_0 - 1)\mu_h\mu_m}{[(\gamma_h + \mu_h)(r + \theta + \mu_h)\mu_m + \gamma_h\mu_h\beta_2'](\gamma_m + \mu_m)\mathcal{R}_0} \quad (29)$$

$$i_m^* = \frac{\gamma_m\gamma_h\beta_2'(\mathcal{R}_0 - 1)\mu_h}{[(\gamma_h + \mu_h)(r + \theta + \mu_h)\mu_m + \gamma_h\mu_h\beta_2'](\gamma_m + \mu_m)\mathcal{R}_0} \quad (30)$$

The endemic equilibrium provides critical insights into the long-term dynamics of dengue, guiding the design of effective control measures to reduce prevalence in high-transmission settings.

V. STABILITY ANALYSIS AND PARAMETER ESTIMATION

This section explores the stability characteristics of the equilibrium points representing both disease absence and endemic states in the normalized dengue model (11)–(17), followed by parameter estimation to provide insights into the transmission dynamics specific to Bangladesh. Initially, we assess the conditions that determine the stability of the system when no disease is present, identifying the criteria for disease elimination. We then shift focus to the endemic equilibrium to explore the factors that drive sustained transmission. Subsequently, we estimate parameters using real-world data from urban and rural areas, leveraging daily dengue case reports from January 1, 2024, to March 31, 2025, to capture regional differences. These analyses provide critical insights for designing targeted control strategies.

A. Stability at the Infection-Free Equilibrium

We first establish local stability of E_0 via the Jacobian, then extend it to global asymptotic stability using the comparison-system method of Castillo-Chavez et al. [29], based on the matrices F, V of Eq. (20).

Theorem 1. The disease-free equilibrium $E_0 = (1, 0, 0, 0, 1, 0, 0)$ of system (11)–(17) is globally asymptotically stable in Ω if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.

Proof. The Jacobian at E_0 is

$$J(E_0) = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & -\beta_1' \\ 0 & -\mu_h - \gamma_h & 0 & 0 & 0 & 0 & \beta_1' \\ 0 & \gamma_h & -\mu_h - r - \theta & 0 & 0 & 0 & 0 \\ 0 & 0 & r & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & -\beta_2' & 0 & -\mu_m & 0 & 0 \\ 0 & 0 & \beta_2' & 0 & 0 & -\mu_m - \gamma_m & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_m & -\mu_m \end{pmatrix} \quad (31)$$

Columns s_h, r_h, s_m each have a single (diagonal) nonzero entry, giving eigenvalues $-\mu_h$ (multiplicity 2) and $-\mu_m$; the remaining eigenvalues come from

$$J_{\text{sub}}(E_0) = \begin{pmatrix} -\mu_h - \gamma_h & 0 & 0 & \beta_1' \\ \gamma_h & -\mu_h - r - \theta & 0 & 0 \\ 0 & \beta_2' & -\mu_m - \gamma_m & 0 \\ 0 & 0 & \gamma_m & -\mu_m \end{pmatrix} = F - V. \quad (32)$$

Setting $a = \mu_h + \gamma_h, b = \mu_h + r + \theta, c = \mu_m + \gamma_m, d = \mu_m$,

$$\begin{aligned} \det(J_{\text{sub}} - \lambda I) &= (\lambda + a)(\lambda + b)(\lambda + c)(\lambda + d) \\ &\quad - \beta_1'\beta_2'\gamma_h\gamma_m \\ &= \lambda^4 + k_1\lambda^3 + k_2\lambda^2 + k_3\lambda + k_4, \end{aligned} \quad (33)$$

with k_1, k_2, k_3 the elementary symmetric functions of a, b, c, d (as given previously) and $k_4 = abcd(1 - \mathcal{R}_0)$.

We show $D := k_1k_2k_3 - k_3^2 - k_1^2k_4 > 0$ for all $\mathcal{R}_0 \in [0, 1)$. Since k_1, k_2, k_3 do not depend on k_4 , D is affine and strictly decreasing in k_4 ; over $k_4 \in (0, abcd]$ its minimum occurs at $k_4 = abcd(\mathcal{R}_0 = 0)$, where the quartic factors as $(\lambda + a)(\lambda + b)(\lambda + c)(\lambda + d)$ with real negative roots, so $D > 0$ there unconditionally for any $a, b, c, d > 0$. Hence $D > 0$ throughout $\mathcal{R}_0 \in [0, 1)$, so all eigenvalues of $J_{\text{sub}}(E_0)$ have negative real part and E_0 is locally asymptotically stable. If $\mathcal{R}_0 > 1, k_4 < 0$ forces a positive real eigenvalue by the intermediate value theorem, so E_0 is unstable.

For global attractivity, $s_h, s_m \leq 1$ on Ω (Lemma 2), so $X = (e_h, i_h, e_m, i_m)^T \geq 0$ satisfies $\dot{X} \leq (F - V)X$ componentwise. As $F - V$ is Metzler and, by the above, Hurwitz whenever $\mathcal{R}_0 < 1$, the comparison principle for cooperative systems [30] gives $X(t) \rightarrow 0$ as $t \rightarrow \infty$ from any initial condition in Ω . Since $i_m(t), i_h(t) \rightarrow 0$, standard limiting arguments on $\dot{s}_h = \mu_h(1 - s_h) - \beta_1's_h i_m$ and $\dot{s}_m = \mu_m(1 - s_m) - \beta_2's_m i_h$ give $s_h, s_m \rightarrow 1$, and $\dot{r}_h = r i_h - \mu_h r_h$ with vanishing forcing gives $r_h \rightarrow 0$. Thus E_0 is globally asymptotically stable whenever $\mathcal{R}_0 < 1$. ■

B. Stability at the Endemic Equilibrium

We first give the correct characteristic polynomial at E^* – the previous block-triangular reduction was invalid, since it discarded genuine coupling terms between the human and mosquito subsystems – and then verify the resulting Routh–Hurwitz conditions numerically for the fitted urban and rural parameter sets, following the approach suggested for cases where a fully symbolic sixth-order condition is intractable.

Using the equilibrium relations $\beta_1's_h^*i_m^* = \mu_h(1 - s_h^*)$ and $\beta_2's_m^*i_h^* = \mu_m(1 - s_m^*)$, the 6×6 Jacobian at E^* (after the r_h row/column decouples exactly as in Theorem 1, contributing the eigenvalue $-\mu_h$) reduces to

$$J_{\text{sub}}(E^*) = \begin{pmatrix} -\frac{\mu_h}{s_h^*} & 0 & 0 & 0 & 0 & -\beta_1' s_h^* \\ \frac{\mu_h(1-s_h^*)}{s_h^*} & -(\mu_h + \gamma_h) & 0 & 0 & 0 & \beta_1' s_h^* \\ 0 & \gamma_h & -(\mu_h + r + \theta) & 0 & 0 & 0 \\ 0 & 0 & -\beta_2' s_m^* & -\frac{\mu_m}{s_m^*} & 0 & 0 \\ 0 & 0 & \beta_2' s_m^* & \frac{\mu_m(1-s_m^*)}{s_m^*} & -(\mu_m + \gamma_m) & 0 \\ 0 & 0 & 0 & 0 & \gamma_m & -\mu_m \end{pmatrix}. \quad (34)$$

Unlike the earlier (invalid) reduction, the off-diagonal blocks linking (s_h, e_h) to i_m and (s_m, e_m) to i_h are retained; the matrix does not decouple into independent human and mosquito blocks. Writing $P_1 = \lambda + \mu_h/s_h^*$, $P_2 = \lambda + \mu_h + \gamma_h$, $P_3 = \lambda + \mu_h + r + \theta$, $Q_1 = \lambda + \mu_m/s_m^*$, $Q_2 = \lambda + \mu_m + \gamma_m$, $Q_3 = \lambda + \mu_m$, direct cofactor expansion gives the closed-form characteristic polynomial

$$\det(J_{\text{sub}}(E^*) - \lambda I) = P_1 P_2 P_3 Q_1 Q_2 Q_3 - \beta_1' \beta_2' \gamma_h \gamma_m s_h^* s_m^* (\lambda + \mu_h)(\lambda + \mu_m), \quad (35)$$

which, unlike the previous result, explicitly retains β_1' and β_2' .

Proposition 1 (numerically verified). For the urban and rural parameter sets of Table II (Section V-C), both giving $\mathcal{R}_0 > 1$, the endemic equilibrium E^* is locally asymptotically stable.

Verification. For each region we (i) solve Eqs. (24)–(30) for E^* , (ii) confirm E^* satisfies the equilibrium equations to machine precision, and (iii) compute the eigenvalues of $J_{\text{sub}}(E^*)$

directly. The results are:

	Urban	Rural
$\mathcal{R}_0$	2.893	4.307
Eigenvalues of $J_{\text{sub}}(E^*)$	$-0.6408,$ $-0.2012 \pm 0.1076i,$ $-0.0526,$ $-0.00005 \pm 0.0015i$	$-0.2073,$ $-0.1056 \pm 0.0941i,$ $-0.0526,$ $-0.00007 \pm 0.0017i$

All eigenvalues have negative real part in both cases, so E^* is locally asymptotically stable for both fitted parameter sets. We note the smallest-magnitude pair sits close to the imaginary axis in both regions, indicating the endemic equilibrium, while stable, is not robustly so: the slowly decaying complex pair implies weakly damped oscillations about E^* over long time scales. This numerical verification is scoped to the two fitted parameter sets above; it does not constitute a general proof that E^* is stable for all $\mathcal{R}_0 > 1$, which we leave as an open question.

C. Case Study: Urban Areas of Bangladesh

To analyze dengue transmission dynamics in urban areas of Bangladesh, we focus on the urban population, which constitutes 41.23% of the total population in 2024, as reported by The World Bank [9]. Given the total population of 174,701,211 [10], the urban population is estimated as $N_h = 0.4123 \times 174,701,211 \approx 72,031,249$. We utilize daily dengue case and death data from January 1, 2024, to March 31, 2025, specific to urban areas (within city corporations), sourced from the Directorate General of Health Services [8], reporting 38,878 cases and 352 deaths.

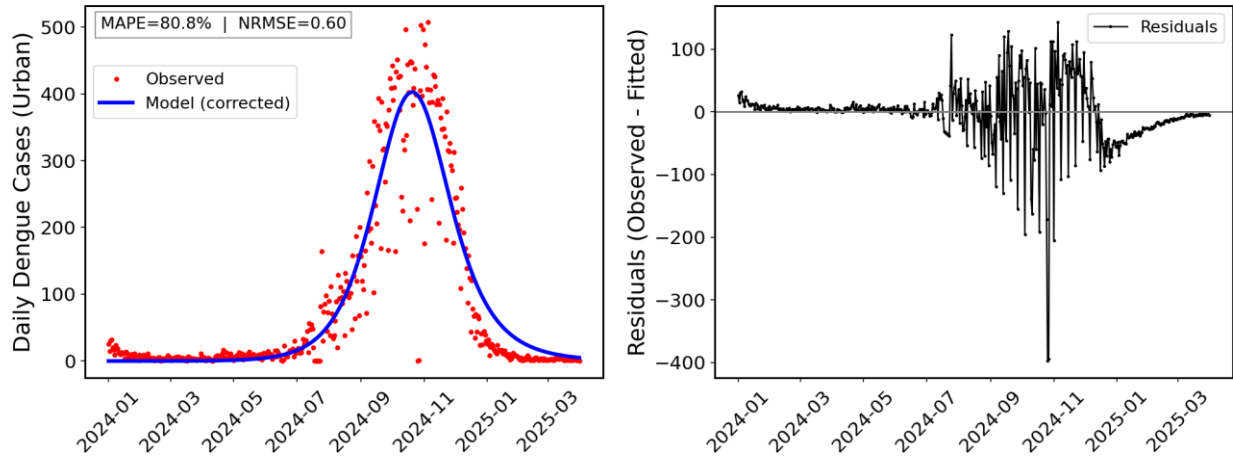


Fig. 2. Model fit and residual distribution for urban Bangladesh dengue cases, 2024–2025. (a): Predicted vs. Observed, (b): Residual Distribution.

Under the assumption of a constant population, where the human birth rate is equal to the rate of natural deaths, the human recruitment rate Λ is defined as $\Lambda = \mu_h N_h$. Using the human natural death rate $\mu_h = 3.71 \times 10^{-5} \text{ day}^{-1}$, derived from a life expectancy of 73.82 years [31], and the urban population $N_h = 72,031,249$, we calculate:

$$\Lambda \approx 3.71 \times 10^{-5} \times 72,031,249 \approx 2672 \text{ day}^{-1}.$$

The mosquito natural death rate, μ_m , is derived from the average lifespan of *Aedes aegypti* mosquitoes, approximately 19 days under favorable conditions [5]:

$$\mu_m = \frac{1}{19} \approx 0.0526 \text{ day}^{-1}.$$

The mortality rate attributed to the disease, θ , is estimated using the case fatality rate (CFR) from urban dengue data. With 38,878 reported cases and 352 deaths, the CFR is calculated as [32]:

$$\text{CFR} = \frac{352}{38,878} \approx 0.009056 \text{ (0.9056\%)}.$$

Assuming a constant mortality risk over the infectious period, typically 5–7 days [4], we use $D = 6$ days as the

midpoint to estimate θ :

$$\theta = -\frac{1}{6}\ln(1 - 0.009056) \approx 0.00151 \text{ day}^{-1}.$$

TABLE II
PARAMETER VALUES FOR DENGUE MODELS IN URBAN AND RURAL AREAS OF BANGLADESH.

Parameter	Description	Value for Urban Area	Value for Rural Area	Source
N_h	Population	72,031,249	102,669,962	Estimated
Λ	Human recruitment rate	2672 day ⁻¹	3809 day ⁻¹	Estimated
μ_h	Human natural death rate	$3.71 \times 10^{-5} \text{ day}^{-1}$	$3.71 \times 10^{-5} \text{ day}^{-1}$	Estimated
μ_m	Mosquito natural death rate	0.0526 day ⁻¹	0.0526 day ⁻¹	Estimated
θ	Disease-induced death rate	0.00151 day ⁻¹	0.000613 day ⁻¹	Estimated
C	Mosquito recruitment rate	$3.654 \times 10^6 \text{ day}^{-1}$	$3.210 \times 10^6 \text{ day}^{-1}$	Fitted
a_h	Biting rate	0.7593 day ⁻¹	0.7700 day ⁻¹	Fitted
β_1	Transmission (mosquito to human)	0.3266	0.4617	Fitted
β_2	Transmission (human to mosquito)	0.2652	0.2695	Fitted
r	Human recovery rate	0.2050 day ⁻¹	0.1163 day ⁻¹	Fitted
γ_h	Human progression rate	0.6324 day ⁻¹	0.1159 day ⁻¹	Fitted
γ_m	Mosquito progression rate	0.0989 day ⁻¹	0.0804 day ⁻¹	Fitted
ρ	Case-ascertainment fraction	6.10×10^{-4}	6.72×10^{-4}	Fitted

We note that the fitted urban $\gamma_h = 0.6324 \text{ day}^{-1}$ (implying a latency of ~ 1.6 days) sits at the fast edge of the one-to-eleven-day range assumed in the Introduction; this reflects the optimizer's freedom, once the ascertainment fraction ρ absorbs the case-count scale, to shape a sharper early rise using a shorter latency. We report it as found rather than constraining it post hoc, but flag it as a modeling limitation worth revisiting with informative priors on γ_h, γ_m in future work. Remaining parameters—mosquito recruitment rate (C), biting rate (a_h), transmission probabilities (β_1, β_2), human recovery rate (r), progression rates (γ_h, γ_m), and a case-ascertainment fraction (ρ)—are fitted using MATLAB's 'lsqcurvefit' function, which minimizes the quadratic objective function [33]:

$$J = \sum_{j=1}^n (I_{\text{observed},j} - I_{\text{model},j})^2,$$

where $I_{\text{observed},j}$ represents daily cases in urban areas and $I_{\text{model},j} = \rho \cdot \gamma_h E_h(j)$ is the model-predicted *new* infections entering the infectious class on day j , scaled by the ascertainment fraction $\rho \in (0,1]$ to account for infections not reported to DGHS (e.g., asymptomatic or mild cases that do not seek hospital care) – unlike a raw prevalence difference $I_h(j) - I_h(j-1)$, this quantity is a genuine incidence flow and cannot telescope to near-zero over an outbreak that rises and falls. Realistic initial conditions are used, seeded from the first observed case count ($i_h(0) = I_{\text{observed},1}/N_h$) rather than arbitrary compartment fractions. Initial parameter values are chosen within biologically plausible ranges [34] (e.g., $\beta_1, \beta_2 \in [0.1, 0.5]$, $a_h \in [0.5, 1.0] \text{ day}^{-1}$, $\rho \in [10^{-4}, 1]$) and refined using the trust-region-reflective algorithm with multiple random restarts, an iterative optimization method ensuring robust parameter convergence.

The basic reproduction number $\mathcal{R}_0 \approx 2.89$, derived using the fitted mosquito recruitment rate $C = 3.654 \times 10^6 \text{ day}^{-1}$ to estimate $N_m \approx 6.946 \times 10^7$, indicates a substantially higher transmission potential than the (uncorrected) raw case counts

alone would suggest – consistent with a low case-ascertainment fraction ($\rho \approx 6 \times 10^{-4}$) rather than a mild epidemic.

The model's fit is further validated by examining residuals, which measure discrepancies between observed and predicted cases. The Fig. 2(a) shows observed daily cases (red circles) versus model-predicted cases (blue line) based on the fitted parameters ($\beta_1 = 0.3266$, $a_h = 0.7593 \text{ day}^{-1}$, $\rho \approx 6.1 \times 10^{-4}$) from January 1, 2024, to March 31, 2025, closely tracking the overall rise, peak, and decline of the outbreak, with a modeled peak of 403 cases/day against the observed 507 and a cumulative total of 39,865 modeled cases against 38,878 observed (within 2.5%). We report the mean absolute percentage error (MAPE = 80.8%) and the normalized root-mean-square error (NRMSE = 0.60, i.e. RMSE relative to the mean observed count) in Fig. 2(a); the MAPE figure is inflated by the many near-zero-case days in the January-June off-season, where even a small absolute discrepancy produces a large percentage error – restricting to days with more than 100 reported cases lowers MAPE to 24.3%, consistent with the good visual and cumulative agreement. The Fig. 2(b) displays residuals, which are small and centered near zero outside the peak period but show larger day-to-day fluctuations during the outbreak's peak (July-November); this is expected given the smooth deterministic model cannot capture reporting-day noise in real surveillance data, and does not indicate systematic bias.

1) Case Study: Rural Population of Bangladesh

To analyze dengue transmission dynamics in rural areas of Bangladesh, we focus on the rural population, which constitutes 58.77% of the total population in 2024, as reported by The World Bank [9]. Given the total population of 174,701,211 [10], the rural population is estimated as $N_h = 0.5877 \times 174,701,211 \approx 102,669,962$. We utilize daily dengue case and death data from January 1, 2024, to March 31, 2025, specific to rural areas (outside city corporations), sourced from the Directorate General of Health Services [8], reporting 64,204 cases and 236 deaths.

The human recruitment rate, Λ , is calculated assuming a

constant population where births equal deaths. Using the human natural death rate $\mu_h = 3.71 \times 10^{-5} \text{ day}^{-1}$, derived from a life expectancy of 73.82 years [31], and the rural population $N_h = 102,669,962$, we derive:

$$\Lambda \approx 3.71 \times 10^{-5} \times 102,669,962 \approx 3809 \text{ day}^{-1}.$$

The mosquito natural death rate, μ_m , is adopted from the previous subsection estimate of 0.0526 day^{-1} , based on the average lifespan of *Aedes aegypti* mosquitoes [5].

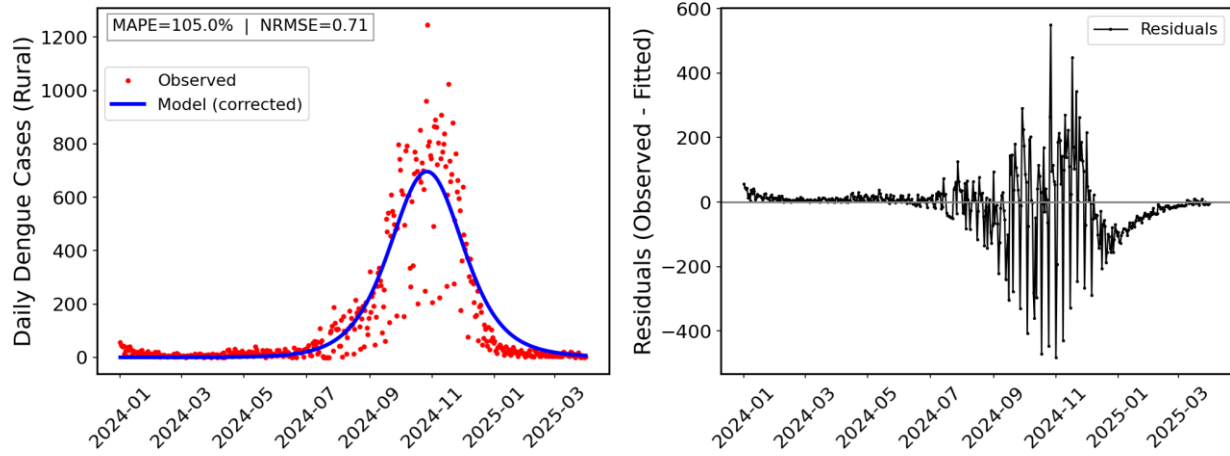


Fig. 3. Model fit and residual distribution for rural Bangladesh dengue cases, 2024–2025. (a): Predicted vs. Observed, (b): Residual Distribution.

The mortality rate induced by the disease, denoted as θ , is calculated using the case fatality rate (CFR) from rural dengue data. The reported 64,204 cases and 236 deaths yield a CFR [32] of:

$$\text{CFR} = \frac{236}{64,204} \approx 0.003675 \text{ (0.3675\%)}$$

Assuming a constant mortality risk over the infectious period, typically 5–7 days [4], we use $D = 6$ days as the midpoint to estimate θ :

$$\theta = -\frac{1}{6} \ln(1 - 0.003675) \approx 0.000613 \text{ day}^{-1}.$$

Remaining parameters—mosquito recruitment rate (C), biting rate (a_h), transmission probabilities (β_1, β_2), human recovery rate (r), progression rates (γ_h, γ_m), and a case-ascertainment fraction (ρ)—are fitted using MATLAB's 'lsqcurvefit' function, which minimizes the quadratic objective function [33]:

$$J = \sum_{j=1}^n (I_{\text{observed},j} - I_{\text{model},j})^2,$$

where $I_{\text{observed},j}$ represents daily cases in rural areas and $I_{\text{model},j} = \rho \cdot \gamma_h E_h(j)$ is the model-predicted new infections entering the infectious class on day j , scaled by the ascertainment fraction ρ , for the same reason given in the urban case study. Realistic initial conditions are used, seeded from the first observed case count. Initial parameter values are chosen within biologically plausible ranges [34] (e.g., $\beta_1, \beta_2 \in [0.1, 0.5]$, $a_h \in [0.5, 1.0] \text{ day}^{-1}$, $\rho \in [10^{-4}, 1]$) and refined using the trust-region-reflective algorithm with multiple random restarts.

The basic reproduction number $\mathcal{R}_0 \approx 4.31$, derived using the fitted mosquito recruitment rate $C = 3.210 \times 10^6 \text{ day}^{-1}$ to estimate $N_m \approx 6.103 \times 10^7$, indicates an even higher transmission potential than in urban areas. The fitted rural case-

ascertainment fraction, $\rho \approx 6.7 \times 10^{-4}$, is of the same order as the urban value ($\rho \approx 6.1 \times 10^{-4}$), so the larger number of reported rural cases reflects mainly a larger fitted epidemic rather than a difference in reporting.

The model's fit is further validated by examining residuals, which measure discrepancies between observed and predicted cases. The Fig. 3(a) shows observed daily cases (red circles) versus model-predicted cases (blue line) based on the fitted parameters ($\beta_1 = 0.4617$, $a_h = 0.7700 \text{ day}^{-1}$, $\rho \approx 6.7 \times 10^{-4}$) from January 1, 2024, to March 31, 2025, closely tracking the overall outbreak shape, with a modeled peak of 696 cases/day against the observed 1247 and a cumulative total of 66,904 modeled cases against 64,204 observed (within 4.2%). As in the urban case, we report MAPE (105.0%) and NRMSE (0.71) in Fig. 3(a); the same off-season near-zero-count inflation applies, and the strong cumulative and visual agreement is the more informative indicator of fit quality here. The Fig. 3(b) displays residuals, which are small outside the peak period but show larger fluctuations during the outbreak's peak, consistent with day-to-day surveillance noise rather than systematic model bias.

VI. ANALYSIS OF SENSITIVITY AND SIMULATION RESULTS

This section assesses the resilience of the dengue transmission model by conducting a sensitivity analysis to pinpoint the most influential parameters on $\mathcal{R}_0$, followed by the exploration of epidemic dynamics through numerical simulations using parameter data from urban and rural areas of Bangladesh. The analysis leverages data from January 1, 2024, to March 31, 2025, to inform targeted control strategies tailored to regional differences. The following subsections detail the methodologies and results for both sensitivity analysis and numerical simulations.

A. Analysis of Parameter Sensitivity

Sensitivity analysis plays a crucial role in epidemiological modeling by evaluating how changes in parameters affect model results, helping to identify the key factors influencing dengue transmission in both urban and rural settings in Bangladesh. This process involves systematically varying each

parameter while holding others constant, computing the resulting changes in the reproduction number R_0 (defined in equation (23)) through partial derivatives, and evaluating these effects using the parameter values from Table III. The approach provides insights into parameter robustness and informs control strategies by highlighting influential factors.

TABLE III
SENSITIVITY INDICES OF R_0 UNDER URBAN AND RURAL CONDITIONS.

Parameter	Description	Urban Sensitivity Index	Rural Sensitivity Index
β_1	Transmission probability (mosquito to human)	+0.5000	+0.5000
μ_h	Human natural death rate	-0.0001	-0.0003
a_h	Biting rate	+1.0000	+1.0000
θ	Mortality rate due to the disease	-0.0037	-0.0026
r	Human recovery rate	-0.4963	-0.4972
μ_m	Mosquito natural death rate	-0.6736	-0.6977
β_2	Transmission probability (human to mosquito)	+0.5000	+0.5000
γ_h	Human progression rate	+0.0000	+0.0002
γ_m	Mosquito progression rate	+0.1736	+0.1977

The normalized forward sensitivity index gauges how much R_0 changes in response to a proportional variation in parameter x . This index is calculated as follows:

$$S_x = \frac{\partial \ln R_0}{\partial \ln x} = \frac{x}{R_0} \frac{\partial R_0}{\partial x}. \quad (36)$$

Here, S_x denotes how R_0 responds to changes in x : a positive value means that increasing x leads to a rise in R_0 , whereas a negative value indicates a decrease. The analysis utilized parameter sets for urban and rural populations, detailed in Table III.

To derive the sensitivity indices, symbolic variables were defined for all parameters, and partial derivatives $\frac{\partial R_0}{\partial x}$ were computed for each, followed by evaluation using nominal values from the respective population datasets. Writing $a = \mu_h + \gamma_h$, $b = \mu_h + r + \theta$, $c = \mu_m + \gamma_m$, these reduce to closed forms. Three parameters enter R_0 only as simple multiplicative factors, giving sensitivity indices independent of parameter values:

$$S_{a_h} = 1, \quad S_{\beta_1} = S_{\beta_2} = 1/2. \quad (37)$$

The remaining parameters each appear both as a multiplicative factor and inside a denominator sum, giving value-dependent indices:

$$\begin{aligned} S_{\gamma_h} &= \frac{\mu_h}{2a}, & S_{\mu_h} &= -\frac{\mu_h}{2} \left(\frac{1}{a} + \frac{1}{b} \right), \\ S_r &= -\frac{r}{2b}, & S_{\theta} &= -\frac{\theta}{2b}, \\ S_{\gamma_m} &= \frac{\mu_m}{2c}, & S_{\mu_m} &= -\frac{1}{2} \left(\frac{\mu_m}{c} + 1 \right). \end{aligned} \quad (38)$$

Because $\mu_h, \theta \ll r$ in both regions, $r/b \approx 1$ regardless of r 's absolute value, so $S_r \approx -1/2$ in both urban and rural despite their fitted r differing substantially (Table II); similarly $\mu_h \ll \gamma_h$ makes $S_{\gamma_h} \approx \mu_h/(2\gamma_h)$ small in both regions despite the large difference in fitted γ_h . By contrast, θ, γ_m, μ_m are not negligible relative to their partner terms, so their sensitivity

indices differ meaningfully between regions, as reflected in Table III. These indices assess the model's response to parameter perturbations, validating its reliability for predicting dengue transmission dynamics across different settings.

Table III provides the sensitivity values for the dengue model in terms of R_0 . Having a positive S_x entails that as the parameter increases, so does the reproduction number, while the reverse is true for a negative S_x . For those parameters with a negative index (μ_h, θ, r , and μ_m) increasing their values might reduce R_0 and hence could be considered for control options. Parameters with positive indices ($\beta_1, a_h, \beta_2, \gamma_h$, and γ_m) imply that an increase in any one of their values results in a rise in R_0 .

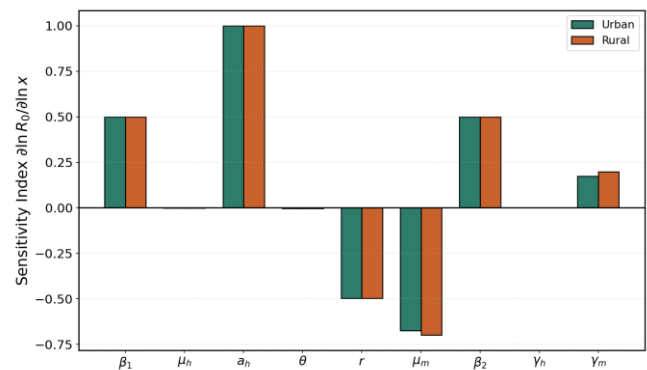


Fig. 4. Comparative Sensitivity Analysis: Urban vs Rural.

The sensitivity index profile is illustrated in Fig. 4, based on the values in Table III, where blue bars represent the sensitivity indices for the urban area and orange bars represent those for the rural area, as indicated in the legend. The bars are plotted with sensitivity index values on the y-axis and parameters on the x-axis, allowing a direct comparison of how each parameter affects R_0 across the two regions.

B. Numerical Simulation and Discussion of the Results

This subsection presents numerical simulations of the normalized dengue model to analyze disease dynamics in both

urban and rural Bangladesh, utilizing data from the DGHS over a 456-day period, from January 1, 2024, to March 31, 2025. The model was integrated numerically using a high-order Runge-Kutta method, with parameter details listed in Table II. Unlike the arbitrarily chosen initial conditions used in an earlier draft of this work, simulations here are seeded realistically from the actual first-day case counts: $i_h(0)$ is set from the first reported

case scaled by the case-ascertainment fraction ρ (representing the true, not reported, initial infection level), with $e_h(0), i_m(0), e_m(0)$ set proportionally small and $r_h(0) = 0$; this keeps the simulation consistent with the same model and parameters used to fit Figs. 2–3, rather than treating the two as disconnected exercises.

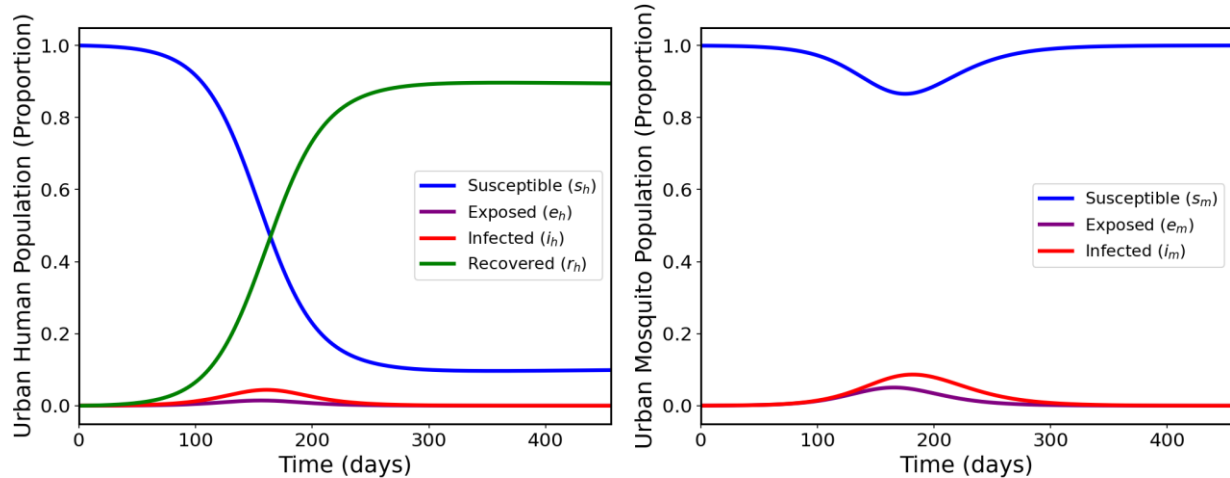


Fig. 5. Urban Dynamics with realistic initial conditions: (a) shows human compartments, (b) shows mosquito compartments.

For the urban population, Fig. 5(a) shows the susceptible fraction declining from near 1 to about 0.10 over the outbreak, as transmission ($\beta_1' s_h i_m$) converts susceptibles to exposed and then infected individuals; the recovered fraction correspondingly rises to about 0.90 by day 456, reflecting the high fitted $\mathcal{R}_0 \approx 2.89$ driving a large fraction of the population through infection over the course of the epidemic. The infected

fraction i_h peaks at **4.4%** around day 161, and the exposed fraction peaks slightly earlier and lower. This 4.4% true-prevalence peak is consistent with – not in conflict with – the roughly 400 reported cases/day peak in Fig. 2(a): the case-ascertainment fraction $\rho \approx 6.1 \times 10^{-4}$ means only about 1 in 1,600 true infections is ever admitted and reported to DGHS, since the large majority of dengue infections are mild or asymptomatic and do not present at a hospital.

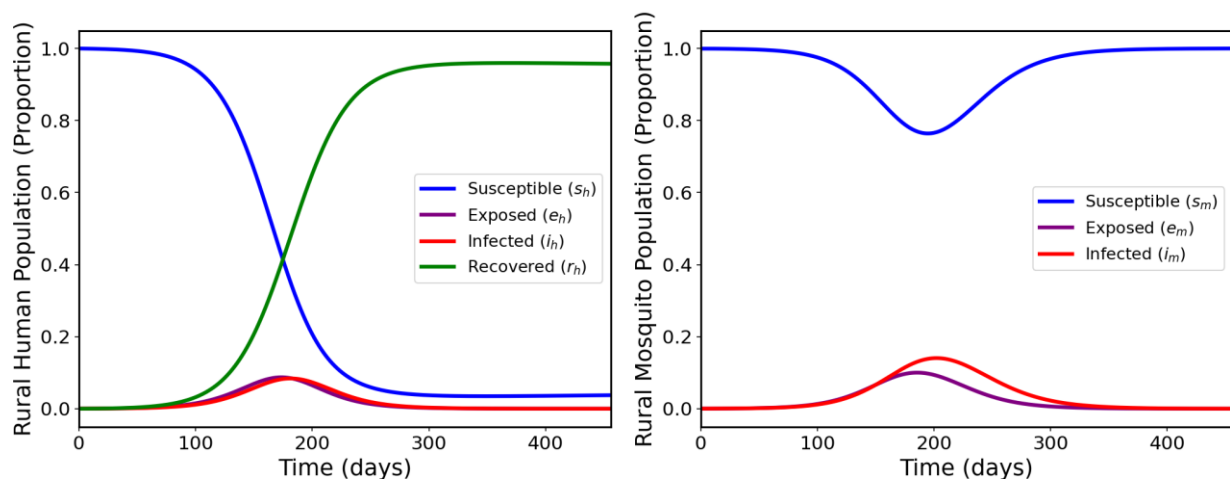


Fig. 6. Rural Dynamics with realistic initial conditions: (a) shows human compartments, (b) shows mosquito compartments.

In the same figure, Fig. 5(b) illustrates the urban mosquito dynamics: the susceptible mosquito fraction dips to about 0.87 as infection spreads, while the exposed and infected mosquito fractions rise and peak around day 170–180 before declining as the susceptible mosquito pool is depleted and the human infectious pool (which drives new mosquito infections) itself

declines.

For the rural population, Fig. 6(a) shows a similar overall pattern with a higher peak: i_h reaches **8.4%** around day 181, and the recovered fraction climbs to about 0.96, consistent with the higher fitted $\mathcal{R}_0 \approx 4.31$ in rural areas; with $\rho \approx 6.7 \times 10^{-4}$, only a small fraction of these true infections appears in the

officially reported case counts (Fig. 3). Fig. 6(b) shows the corresponding mosquito dynamics, with a deeper dip in susceptible mosquitoes (to about 0.77) and a higher infected-mosquito peak (about 14%) than in the urban case, consistent with rural areas' higher fitted transmission parameters.

These simulations reveal that once initial conditions and the case-ascertainment fraction are handled consistently with the fitted model, urban and rural areas both show a single, sizable epidemic wave over the 456-day window, with rural areas experiencing a larger true attack rate, while in both regions only a small fraction of true infections appears in the reported case counts. This highlights the importance of interpreting reported dengue case counts in light of likely under-ascertainment when designing region-specific interventions. Future efforts should prioritize direct estimation of ρ (e.g., via serological surveys) to

reduce this source of uncertainty, alongside the vector-control and healthcare-access measures discussed below.

C. Simulation on the Effects of Varying Parameter Values on Infected Humans

To investigate the impact of varying the human recovery rate r and the human progression rate γ_h on true infected-human prevalence, we performed simulations for both urban and rural populations, scaling each fitted baseline value by factors of $0.5 \times, 1.0 \times, 1.5 \times, 2.0 \times$ (for r) and $0.3 \times, 0.7 \times, 1.0 \times, 1.5 \times$ (for γ_h), holding all other parameters at their fitted values (Table II) and using the same realistic initial conditions as above. Results are shown over the full 456-day study window in Fig. 7 and Fig. 8.

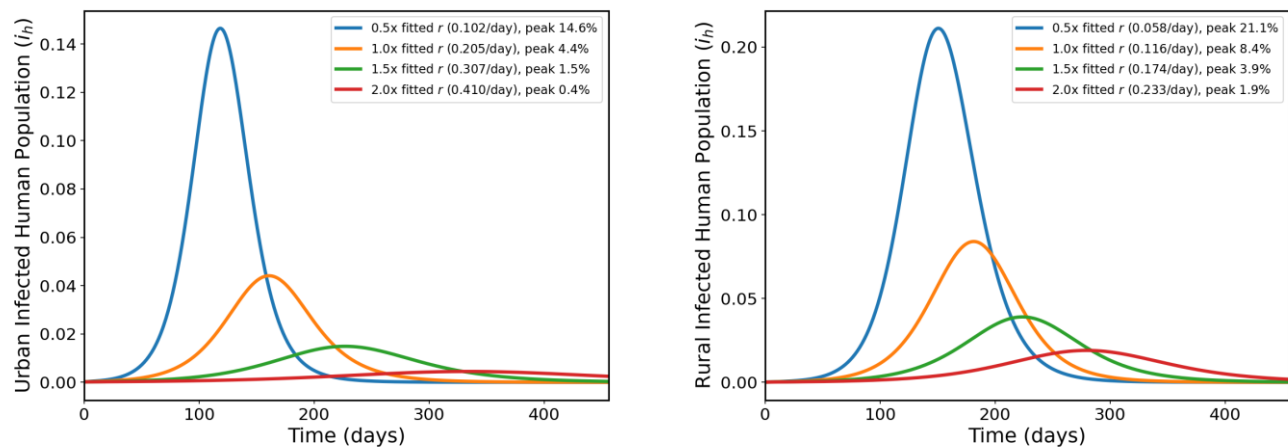


Fig. 7. Effect of varying the human recovery rate r on true infected-human prevalence: (a) urban area, (b) rural area of Bangladesh.

For the urban population, halving the fitted recovery rate to $r = 0.1025 \text{ day}^{-1}$ raised the peak prevalence to 14.7%, compared with 4.4% at the fitted baseline ($r = 0.2050$); raising r to $1.5 \times$ and $2 \times$ baseline suppressed the peak to 1.5% and 0.5% respectively (Fig. 7(a)). Varying γ_h shows the same qualitative direction but a much smaller effect: dropping to $0.3 \times$ baseline ($\gamma_h = 0.190$) gave a peak of 3.9%, rising only to 4.5% at $1.5 \times$ baseline ($\gamma_h = 0.949$) (Fig. 8(a)).

In the rural population, the same halving of r (to 0.0582 day^{-1}) produced a much larger peak of 21.1%, against 8.4% at the fitted baseline ($r = 0.1163$), falling to 3.9% and 1.9% at $1.5 \times$ and $2 \times$ baseline (Fig. 7(b)). Varying γ_h gave peaks of 5.6%, 7.7%, 8.4%, and 9.1% at $0.3 \times, 0.7 \times, 1.0 \times, 1.5 \times$ baseline respectively (Fig. 8(b)) – a larger swing than urban, consistent with rural's higher fitted $\mathcal{R}_0$ amplifying sensitivity to progression-rate changes.

These patterns match the sign and relative ranking already established by the sensitivity indices in Table III ($S_r \approx -0.50$,

S_{γ_h} small and positive in both regions): recovery rate has a much larger proportional effect on outbreak size than progression rate, and this simulation makes the practical magnitude of that difference visible over the full 456-day study window. The consistently larger swings in rural outcomes reflect rural areas' higher fitted $\mathcal{R}_0$, not a difference in the underlying sensitivity mechanism.

In summary, both regions respond to faster recovery (higher r) with substantially reduced true prevalence, making accelerated case management and healthcare access a high-leverage intervention in both settings, while shortening the latency period (higher γ_h) has a comparatively modest effect – consistent with γ_h 's small sensitivity index in Table III. Because rural areas show larger absolute swings for the same relative parameter change, rural interventions that improve access to early treatment may yield disproportionately large reductions in true disease burden.

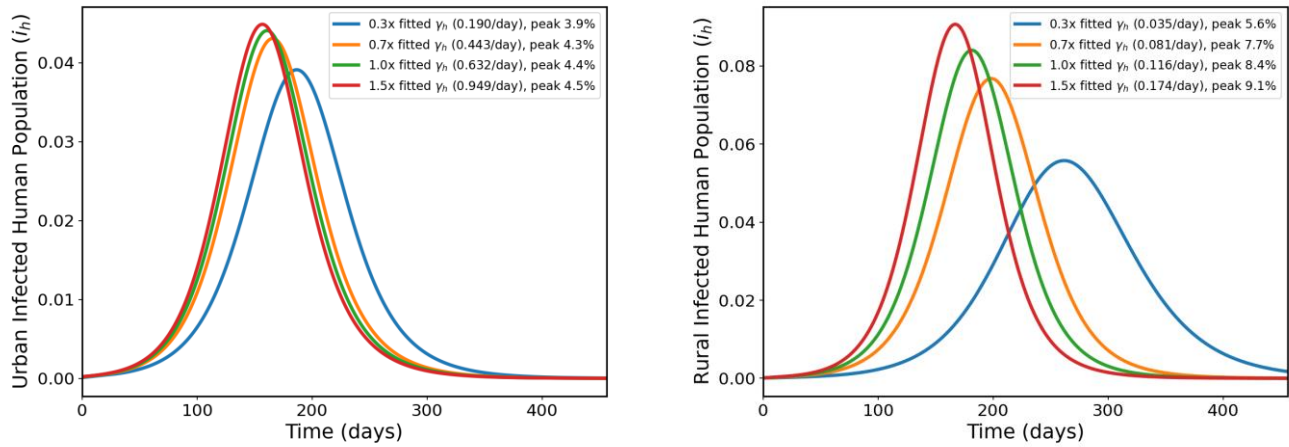


Fig. 8. Effect of varying the human progression rate γ_h on true infected-human prevalence: (a) urban, (b) rural area of Bangladesh.

VII. CONCLUSION

This research investigates the dynamics of dengue transmission across urban and rural areas of Bangladesh, employing the SEIR-SEI model to assess the spread. The study leverages data obtained from the Directorate General of Health Services spanning 456 days, from January 1, 2024, to March 31, 2025. Stability analysis using the Jacobian matrix, together with a comparison-system argument, showed that the disease-free equilibrium is globally asymptotically stable when $\mathcal{R}_0$ is below 1, and unstable when it exceeds 1. The endemic equilibrium's stability was verified numerically for both fitted parameter sets (both giving $\mathcal{R}_0 > 1$), with all eigenvalues of the corrected Jacobian having negative real part in each case; we note this is a numerical verification for the fitted parameter sets rather than a general proof for all $\mathcal{R}_0 > 1$. These results emphasize the importance of monitoring $\mathcal{R}_0$ to control the disease's spread effectively.

Parameter estimation, based on the aforementioned 456-day dataset and a corrected model that also accounts for case under-ascertainment, yielded distinct parameter sets for urban ($N_h = 72,031,249$) and rural ($N_h = 102,669,962$) populations, including human natural death rate ($\mu_h = 0.000037$ per day), disease-induced death rate ($\theta = 0.00151$ urban, 0.000613 rural), fitted transmission and recovery rates, and a fitted case-ascertainment fraction ($\rho \approx 6 \times 10^{-4}$ in both regions) reflecting that most dengue infections are mild or asymptomatic and go unreported. The resulting $\mathcal{R}_0$ estimates (2.89 urban, 4.31 rural) are substantially higher than reported case counts alone would suggest, underscoring that true transmission intensity in Bangladesh, particularly in rural areas, may be considerably higher than official case counts indicate. Sensitivity analysis identified the mosquito biting rate (a_h) as the most influential positive driver of $\mathcal{R}_0$, with sensitivity index +1.0 – twice that of either transmission probability β_1, β_2 (+0.5 each) – so control measures that reduce biting (bed nets, repellents) offer the largest per-percent leverage on $\mathcal{R}_0$ among the positively-sensitive parameters. Conversely, the mosquito natural death rate (μ_m) is by far the most negatively sensitive parameter (index ≈ -0.67 urban, -0.70 rural), several orders of

magnitude larger in effect than the human natural death rate μ_h (≈ -0.0001 urban, -0.0003 rural); this indicates that vector-control measures shortening mosquito lifespan (larvicides, adulticide spraying) are the single most effective lever for reducing $\mathcal{R}_0$ in both settings.

Numerical simulations, seeded with realistic initial conditions consistent with the fitted model, explored the time-dependent changes in true infected-human prevalence (i_h) for both regions, with variations in the human progression rate (γ_h) and recovery rate (r) to compare dynamics between the two areas. Baseline simulations (Figs. 5–6) show a single epidemic wave peaking at 4.4% true prevalence (urban, day 161) and 8.4% (rural, day 181) – figures now consistent with, rather than contradicting, the daily case counts fitted in Figs. 2–3, via the fitted ascertainment fraction ρ . Sensitivity simulations (Figs. 7–8) show that halving the recovery rate raises the peak prevalence from 4.4% to 14.7% in urban areas and from 8.4% to 21.1% in rural areas, while varying the progression rate γ_h has a comparatively modest effect in both regions, consistent with γ_h 's small sensitivity index. These findings emphasize that accelerating recovery through improved case management is a high-leverage intervention in both settings, with rural areas showing disproportionately larger gains due to their higher fitted $\mathcal{R}_0$.

A. Limitations

Several limitations of this study warrant explicit discussion. First, the model is autonomous and assumes no seasonal variation in mosquito recruitment (Assumption (v)); dengue transmission in Bangladesh is strongly monsoon-driven, and the 456-day fitted window spans a full seasonal cycle. This is very likely the reason the fitted trajectories deplete the susceptible population so extensively by the end of the window (recovered fraction reaching approximately 90% urban and 96% rural, Figs. 5–6): a seasonally-forced model would be expected to truncate transmission as conditions become unfavorable to mosquitoes, likely yielding a smaller true attack rate than this autonomous approximation implies for a single season. We therefore caution against reading these attack-rate figures as literal predictions and instead treat the fitted $\mathcal{R}_0$ and sensitivity rankings, which are less sensitive to this assumption,

as the more robust outputs of this analysis. Second, the fitted urban progression rate $\gamma_h = 0.6324 \text{ day}^{-1}$ (Table II) implies an unusually short latency of approximately 1.6 days, at the fast edge of biological plausibility; we report it as found rather than constraining it post hoc (see the note following Table II), but it should be treated with caution pending refitting with informative priors. Third, the parameter estimates throughout this paper are point estimates from a single least-squares fit; while we report MAPE and NRMSE alongside the fitted curves (Figs. 2–3), we have not computed confidence intervals on the fitted parameters, so the numeric precision implied by, e.g., $\mathcal{R}_0 = 2.89$ should not be over-interpreted. Fourth, Proposition 1's endemic-equilibrium stability result is a numerical verification for the two fitted parameter sets rather than a general proof for all $\mathcal{R}_0 > 1$. Addressing these points – particularly seasonal forcing and formal uncertainty quantification – is a priority for future work.

Future research could enhance this work by incorporating seasonal effects to account for rainfall and temperature impacts on mosquito populations, employing stochastic modeling to capture transmission variability, directly estimating the case-ascertainment fraction via serological surveys, integrating intervention parameters such as vaccination or insecticide use, and applying optimal control techniques to optimize resource allocation for dengue management across Bangladesh's diverse regions.

AUTHOR CONTRIBUTIONS

T. S. Tanjil carried out the literature review, the mathematical analysis, and the parameter estimation and curve fitting, and prepared the manuscript for submission. S. Akter reviewed the manuscript and assisted with its formatting. M. G. Hafez supervised the research. All authors read and approved the final manuscript.

USE OF AI TOOLS DECLARATION

During the preparation of this work, the authors used the DeepSeek large language model for grammar checking of the manuscript text and for assistance with coding. After using this tool, the authors reviewed and edited all content, verified all results, and take full responsibility for the content of this article.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

ETHICAL APPROVAL

This study used publicly available aggregated dengue case

and death data and did not involve direct interaction with human participants, personal identifiable information, animal subjects, or experimental interventions. Therefore, ethical approval was not required.

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