

STABILITY ANALYSIS OF A DENGUE MODEL WITH AWARENESS AND REINFECTION

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Received April 30, 2026, Received in revised form July 17, 2026
 Accepted August 8, 2026 Available online October 22, 2026

Abstract. *A mathematical model is developed to investigate dengue transmission by incorporating behavioral awareness, treatment, and reinfection within a coupled human–mosquito system. The human population is structured into unaware susceptible, aware susceptible, exposed, infectious, treated, and recovered compartments, while the mosquito population is divided into susceptible and infectious classes, with the dynamics represented by a nonlinear system of ordinary differential equations. The study characterizes the disease-free and endemic equilibrium states, using the basic reproduction number R_0 as the threshold indicator, and analyzes local stability via the Jacobian matrix and eigenvalue criteria. The disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, implying disease elimination, while for $R_0 > 1$ the system approaches an endemic equilibrium, reflecting sustained transmission. The results further show that awareness and treatment reduce transmission intensity, whereas reinfection can sustain disease persistence, indicating that the combined effects of these three mechanisms jointly determine whether dengue transmission is controlled or remains endemic.*

Keywords: dengue model, reproduction number, stability analysis

1. Introduction

Dengue fever, transmitted primarily by *Aedes aegypti* mosquitoes, remains one of the most significant mosquito-borne diseases in tropical and subtropical regions, with its persistence shaped by environmental conditions, population density, and vector abundance ([1],[2]). Mathematical modeling, particularly host–vector frameworks, has become an important tool for understanding dengue transmission and evaluating intervention strategies ([3],[4]), with the basic reproduction number R_0 and equilibrium stability serving as fundamental quantities for characterizing long-term disease behavior ([5],[6]).

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Beyond biological and environmental factors, dengue transmission is increasingly recognized as being shaped by human behavior and public health interventions: awareness programs reduce infection risk through preventive behavior, treatment shortens the infectious period, and waning immunity with reinfection sustains transmission in endemic regions. Although these mechanisms have been studied individually, models that simultaneously incorporate awareness, treatment, and reinfection within a unified host–vector framework remain relatively limited ([3],[4]). Motivated by this gap, this study develops such a model and analyzes it through the determination of equilibrium points, derivation of R_0 , and local stability analysis, supported by numerical simulations of key parameter effects.

2. The Dengue Model

2.1. Formulation of the Model

The proposed model extends the classical SEIR framework by integrating awareness dynamics and treatment processes within a host–vector interaction system ([5],[7]). The human population is divided into six compartments: unaware susceptible (S_u), aware susceptible (S_a), exposed (E), infectious (I), treatment (P_h), and recovered (R), while the mosquito population consists of susceptible (S_v) and infected (I_v) classes.

Transmission occurs through host–vector interactions ([7],[8]), with awareness modeled as a transition from S_u to S_a that reduces effective infection risk ([2],[9],[10]), treatment reducing the infectious population [11], and reinfection allowing recovered individuals to re-enter the susceptible class ([4],[12]).

2.2. Model Diagram

The diagram in Figure 1 illustrates the flow of individuals between compartments in the coupled human–mosquito system, including the awareness, treatment, and reinfection mechanisms.

As shown in Figure 1 ([7],[8]), humans move from S_u to S_a at rate α via awareness (reducing infection risk by η ([9],[10]), become exposed upon mosquito contact, progress to I at rate γ , and are then treated (rate τ , via P_h at rate σ) or recover naturally (rate δ_h); waning immunity returns recovered individuals to S_u at rate ω , closing the reinfection loop. On the vector side, susceptible mosquitoes become infected after biting infectious humans and remain infectious for life ([7],[8]), completing the transmission cycle.

2.3. Mathematical Model

Based on the described transmission mechanisms and previous host–vector modeling approaches ([7],[8],[12]), the dynamics of the model are described by a nonlinear system of ordinary differential equations given as follows:

$$\begin{aligned}\frac{dS_u}{dt} &= \Lambda_h - \beta_h S_u I_v - \alpha S_u - \mu_h S_u + \omega R, \\ \frac{dS_a}{dt} &= \Lambda_h - \beta_h S_a I_v - \alpha S_a - \mu_h S_a + \omega R,\end{aligned}$$

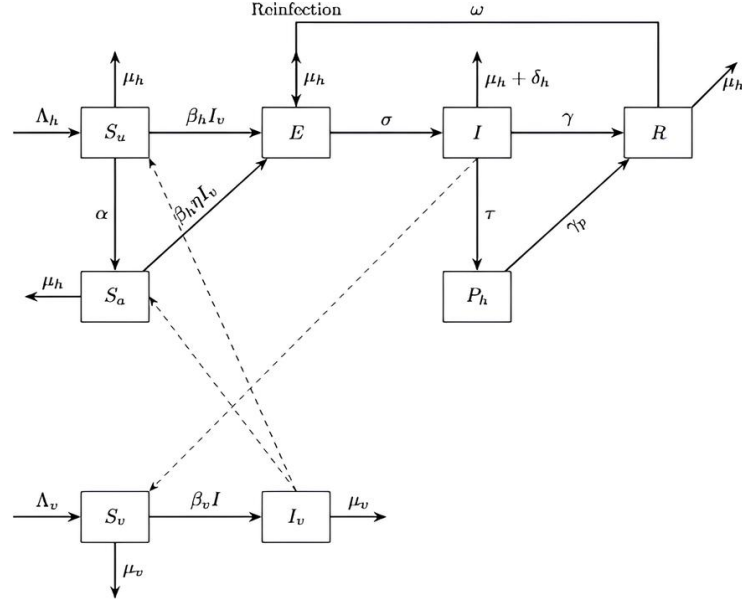


Figure. 1: Dengue transmission model diagram with awareness, treatment, and reinfection

$$\begin{aligned}
 \frac{dS_a}{dt} &= \alpha S_u - \eta \beta_h S_a I_v - \mu_h S_a, \\
 \frac{dE}{dt} &= \beta_h S_u I_v + \eta \beta_h S_a I_v - (\gamma + \mu_h) E, \\
 \frac{dI}{dt} &= \gamma E - (\delta_h + \tau + \mu_h) I, \\
 \frac{dP_h}{dt} &= \tau I - (\sigma + \mu_h) P_h, \\
 \frac{dR}{dt} &= \delta_h I + \sigma P_h - (\omega + \mu_h) R, \\
 \frac{dS_v}{dt} &= \Lambda_v - \beta_v S_v I - \mu_v S_v, \\
 \frac{dI_v}{dt} &= \beta_v S_v I - \mu_v I_v.
 \end{aligned} \tag{2.1}$$

2.4. Model Assumptions

- (1) The population is homogeneous, with constant recruitment (Λ_h , Λ_v) and constant natural mortality rates (μ_h , μ_v); spatial heterogeneity and migration are not considered.
- (2) Exposed individuals (E) are infected but not yet infectious, progressing to the infectious class (I) at rate γ ; infectious individuals (I) either undergo treatment at rate τ or recover naturally at rate δ_h , while treated individuals (P_h) recover into class R at rate σ .

- (3) Recovered individuals lose immunity at rate ω and return to S_u .
- (4) Awareness transitions individuals from S_u to S_a at rate α , reducing infection risk by a factor $0 < \eta < 1$; transmission itself is governed by the host–vector interaction rates β_h and β_v .
- (5) Infected mosquitoes (I_v) remain infectious for life and do not recover.

2.5. Equilibrium and Local Dynamics

Local stability is analyzed via the Jacobian matrix. With the state vector $X = (S_u, S_a, E, I, P_h, R, S_v, I_v)^T$, the disease-free equilibrium is:

$$E_0 = (S_u^*, S_a^*, 0, 0, 0, 0, S_v^*, 0), \quad S_u^* = \frac{\Lambda_h}{\alpha + \mu_h}, \quad S_a^* = \frac{\alpha \Lambda_h}{\mu_h(\alpha + \mu_h)}, \quad S_v^* = \frac{\Lambda_v}{\mu_v}.$$

Writing $c_1 = \beta_h(S_u^* + \eta S_a^*)$, $c_2 = \beta_v S_v^*$, the Jacobian at E_0 is:

$$J(E_0) = \begin{pmatrix} -(\alpha + \mu_h) & 0 & 0 & 0 & 0 & \omega & 0 & -\beta_h S_u^* \\ \alpha & -\mu_h & 0 & 0 & 0 & 0 & 0 & -\eta \beta_h S_a^* \\ 0 & 0 & -(\gamma + \mu_h) & 0 & 0 & 0 & 0 & c_1 \\ 0 & 0 & \gamma & -(\delta_h + \tau + \mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau & -(\sigma + \mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta_h & \sigma & -(\omega + \mu_h) & 0 & 0 \\ 0 & 0 & 0 & -c_2 & 0 & 0 & -\mu_v & 0 \\ 0 & 0 & 0 & c_2 & 0 & 0 & 0 & -\mu_v \end{pmatrix}.$$

Since (S_u, S_a) and S_v decouple from (E, I, P_h, R, I_v) at the DFE, three eigenvalues follow directly: $\lambda_1 = -(\alpha + \mu_h)$, $\lambda_2 = -\mu_h$, $\lambda_3 = -\mu_v$. The remaining five satisfy:

$$P_5(\lambda) = \lambda^5 + a_1 \lambda^4 + a_2 \lambda^3 + a_3 \lambda^2 + a_4 \lambda + a_5 = 0, \quad (2.2)$$

where, with $k_1 = \gamma + \mu_h$, $k_2 = \delta_h + \tau + \mu_h$, $k_3 = \sigma + \mu_h$, $k_4 = \omega + \mu_h$, $k_5 = \mu_v$ (corresponding to E, I, P_h, R, I_v respectively):

$$a_1 = \sum_{i=1}^5 k_i, \quad a_2 = \sum_{i < j} k_i k_j, \quad (2.3)$$

$$a_3 = \sum_{i < j < \ell} k_i k_j k_\ell - \gamma c_1 c_2, \quad a_4 = \sum_{i < j < \ell < m} k_i k_j k_\ell k_m - \gamma c_1 c_2 (k_3 + k_4), \quad (2.4)$$

$$a_5 = \prod_{i=1}^5 k_i - \gamma c_1 c_2 k_3 k_4. \quad (2.5)$$

The correction term $\gamma c_1 c_2$ in a_3, a_4, a_5 reflects the transmission cycle $E \rightarrow I \rightarrow I_v \rightarrow E$; since P_h and R do not feed back into (E, I, I_v) , a_1 and a_2 need no such correction. Using $k_1 k_2 k_5 = (\gamma + \mu_h)(\delta_h + \tau + \mu_h)\mu_v$,

$$a_5 = \left(\prod_{i=1}^5 k_i \right) (1 - R_0^2), \quad R_0^2 = \frac{\gamma c_1 c_2}{k_1 k_2 k_5} = \frac{\gamma \beta_h \beta_v S_v^* (S_u^* + \eta S_a^*)}{(\gamma + \mu_h)(\delta_h + \tau + \mu_h)\mu_v}.$$

Since $k_i > 0$ ensures $a_1, a_2, a_3, a_4 > 0$ whenever $R_0 < 1$, the Routh–Hurwitz criterion reduces stability of E_0 to the sign of a_5 : E_0 is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$, with an endemic equilibrium ($I^* > 0$) emerging in the latter case.

Endemic Equilibrium

Solving system (2.1) at steady state in terms of I^* gives

$$P_h^* = \frac{\tau}{G_2} I^*, \quad E^* = \frac{G_1}{\sigma} I^*, \quad R^* = \frac{G_3}{(\omega + \mu_h) G_2} I^*,$$

where, from the balance equations $dP_h/dt = 0$, $dI/dt = 0$, and $dR/dt = 0$ respectively,

$$G_2 = \sigma + \mu_h, \quad G_1 = \frac{\sigma(\delta_h + \tau + \mu_h)}{\gamma}, \quad G_3 = \delta_h G_2 + \sigma \tau.$$

Together with

$$S_u^* = \frac{\Lambda_h}{\beta_h I_v^* + \alpha + \mu_h}, \quad S_a^* = \frac{\alpha S_u^*}{\eta \beta_h I_v^* + \mu_h}, \quad S_v^* = \frac{\Lambda_v}{\beta_v I^* + \mu_v}, \quad I_v^* = \frac{\beta_v \Lambda_v}{\mu_v (\beta_v I^* + \mu_v)} I^*,$$

the existence of $I^* > 0$ is governed by:

$$\frac{\beta_h \beta_v \Lambda_v}{\mu_v (\beta_v I^* + \mu_v)} (S_u^* + \eta S_a^*) + \frac{\omega G_3}{(\omega + \mu_h) G_2} - \frac{(\sigma + \mu_h) G_1}{\sigma} = 0.$$

2.6. Basic Reproduction Number

The value of R_0 is obtained via the Next Generation Matrix, applied to the infected compartments (E, I, I_v) . Writing $dx/dt = F(x) - V(x)$, the new-infection and transition matrices are:

$$F = \begin{pmatrix} 0 & 0 & \beta_h (S_u^* + \eta S_a^*) \\ 0 & 0 & 0 \\ 0 & \beta_v S_v^* & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \gamma + \mu_h & 0 & 0 \\ -\gamma & \delta_h + \tau + \mu_h & 0 \\ 0 & 0 & \mu_v \end{pmatrix}.$$

The $(1, 1)$ entry of V follows directly from dE/dt and is therefore $\gamma + \mu_h$, while the $E \rightarrow I$ transition γ appears as the $(2, 1)$ off-diagonal entry, representing the inflow into I .

With $K = FV^{-1}$, the nonzero entries are:

$$K_{13} = \frac{\beta_h (S_u^* + \eta S_a^*)}{\mu_v}, \quad K_{31} = \frac{\gamma \beta_v S_v^*}{(\gamma + \mu_h)(\delta_h + \tau + \mu_h)}, \quad K_{32} = \frac{\beta_v S_v^*}{\delta_h + \tau + \mu_h},$$

and $R_0 = \rho(K) = \sqrt{K_{13} K_{31}}$. Substituting the disease-free equilibrium values:

$$S_u^* + \eta S_a^* = \frac{\Lambda_h (\mu_h + \eta \alpha)}{\mu_h (\alpha + \mu_h)}, \quad S_v^* = \frac{\Lambda_v}{\mu_v},$$

yields:

$$R_0 = \sqrt{\frac{\gamma \beta_h \beta_v \Lambda_h \Lambda_v (\mu_h + \eta \alpha)}{\mu_h \mu_v^2 (\alpha + \mu_h) (\gamma + \mu_h) (\delta_h + \tau + \mu_h)}}. \quad (2.6)$$

The value of R_0 characterizes the average number of secondary infections produced by a single infectious individual in a fully susceptible population. Note that σ , which belongs to the treatment/recovery pathway ($P_h \rightarrow R$), does not participate in generating new infections and therefore does not appear in R_0 .

3. Simulation of Model Dynamics

Numerical experiments were performed using standard integration methods ([7],[12]) for two representative scenarios: $R_0 = 0.2092$ (disease-free) and $R_0 = 2.5099$ (endemic).

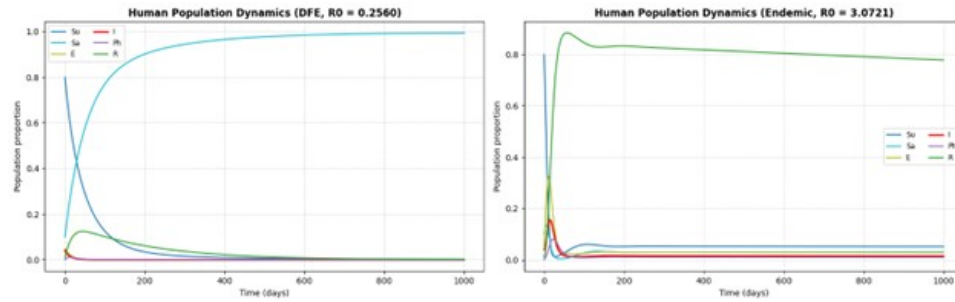


Figure. 2: Dynamics of human compartments (S_u, S_a, E, I, P_h, R) under disease-free ($R_0 < 1$) and endemic ($R_0 > 1$) scenarios.

Figure 2 shows that at $R_0 = 0.2092$, the exposed, infected, and treatment classes decay toward zero ($I(t) \approx 0.000492$) as S_a grows and dominates, confirming convergence to the DFE and the effectiveness of awareness. At $R_0 = 2.5099$, these compartments instead stabilize at positive levels ($I(t) \approx 0.016220$), with reinfection via ω sustaining the endemic equilibrium (EE) despite the continued rise in S_a .

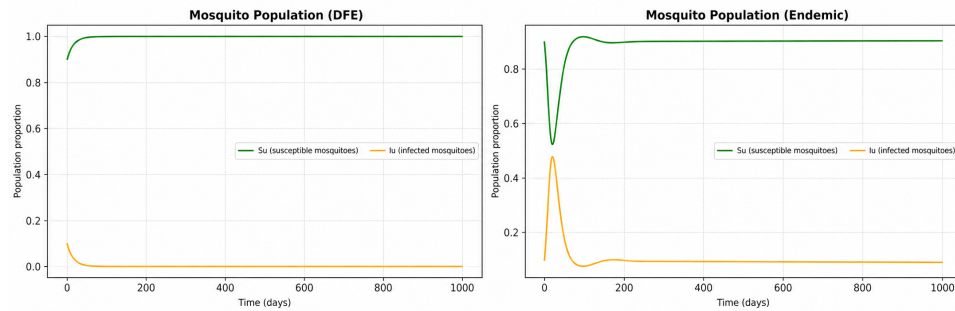


Figure. 3: Dynamics of mosquito compartments (S_v, I_v) under disease-free ($R_0 < 1$) and endemic ($R_0 > 1$) scenarios.

Figure 3 shows the corresponding mosquito dynamics: I_v decays to $I_v(t) \approx 0.000296$ under the DFE scenario, confirming an interrupted transmission cycle, but stabilizes at $I_v(t) \approx 0.104812$ under the EE scenario, underscoring the mosquito population's role in sustaining transmission.

These numerical results agree with the analytical findings: the system converges

to the DFE when $R_0 < 1$ and to the endemic equilibrium

$$X^{**} = (S_u^*, S_a^*, E^*, I^*, P_h^*, R^*, S_v^*, I_v^*),$$

when $R_0 > 1$, as derived above. Awareness and treatment reduce transmission intensity but cannot eliminate the disease under high transmission, reinforcing R_0 as the key threshold governing extinction versus persistence ([4], [6]).

These results are further supported by the sensitivity analysis in Table 2, which identifies the parameters that most strongly influence R_0 ([3],[10],[13],[14]).

Table 1: Model parameters used in the simulation

Symbol	Description	Value	Source
b	Mosquito biting rate	0.5	Modified
μ_h	Natural death rate	0.0000391	Literature
μ_v	Mosquito mortality rate	0.0833	Literature
Λ_h	Human recruitment	0.0000391	Assumption
Λ_v	Mosquito recruitment	0.0833	Assumption
β_h	Human transmission rate	0.05 / 0.6	Scenario
β_v	Mosquito transmission rate	0.05 / 0.6	Scenario
γ	Progression rate ($E \rightarrow I$)	0.2	Modified
σ	Recovery rate (treatment compartment, $P_h \rightarrow R$)	0.1429	Literature
δ_h	Recovery rate (infectious compartment, $I \rightarrow R$)	0.005	Assumption
α	Awareness rate	0.02	Model
η	Awareness effectiveness	0.4	Model
τ	Treatment rate	0.07	Model
ω	Immunity waning	0.005	Model

Beyond specifying parameter values, a sensitivity analysis of R_0 is performed to identify which parameters most strongly influence transmission. The normalized forward sensitivity index of R_0 with respect to a parameter p is defined as

$$S_p = \frac{\partial R_0}{\partial p} \frac{p}{R_0},$$

where a positive (negative) value indicates that increasing p increases (decreases) R_0 , and $|S_p|$ reflects the relative strength of that effect.

Differentiating $\ln R_0$ with respect to $\ln p$ in Equation (2.6) gives, for parameters entering as a simple power of R_0^2 ,

$$S_{\beta_h} = S_{\beta_v} = S_{\Lambda_h} = S_{\Lambda_v} = \frac{1}{2}, \quad S_{\mu_v} = -1,$$

and, for parameters entering through more than one factor $(\mu_h, \alpha, \eta, \gamma, \tau, \delta_h)$,

$$\begin{aligned} S_{\mu_h} &= \frac{1}{2} \left[\frac{\eta\mu_h}{\mu_h + \eta\alpha} - 1 - \frac{\mu_h}{\alpha + \mu_h} - \frac{\mu_h}{\gamma + \mu_h} \right], \\ S_{\alpha} &= \frac{1}{2} \left[\frac{\eta\alpha}{\mu_h + \eta\alpha} - \frac{\alpha}{\alpha + \mu_h} \right], \\ S_{\eta} &= \frac{1}{2} \frac{\eta\alpha}{\mu_h + \eta\alpha}, \\ S_{\gamma} &= \frac{1}{2} \left[1 - \frac{\gamma}{\gamma + \mu_h} \right], \\ S_{\tau} &= -\frac{1}{2} \frac{\tau}{\delta_h + \tau + \mu_h}, \\ S_{\delta_h} &= -\frac{1}{2} \frac{\delta_h}{\delta_h + \tau + \mu_h}. \end{aligned}$$

The parameters σ , ω , and b do not appear explicitly in R_0 and therefore have no sensitivity index with respect to it; their influence is instead on endemic prevalence rather than the transmission threshold.

Table 2 reports these indices using the baseline values from Table 1. The transmission parameters β_h , β_v , Λ_h , Λ_v , together with η , exert the strongest positive influence ($S_p = 0.50$), while μ_v exerts the strongest negative influence ($S_{\mu_v} = -1.00$), followed by μ_h and τ ; α , γ , and δ_h have comparatively small effects.

Table 2: Normalized forward sensitivity indices of R_0

Parameter	Sensitivity Index S_p	Effect	Rank
μ_v	-1.000	Strong negative	1
β_h	+0.500	Strong positive	2
β_v	+0.500	Strong positive	2
Λ_h	+0.500	Strong positive	2
Λ_v	+0.500	Strong positive	2
μ_h	-0.500	Moderate negative	2
η	+0.498	Strong positive	3
τ	-0.466	Moderate negative	4
δ_h	-0.033	Weak negative	5
α	-0.001	Negligible	6
γ	≈ 0.000	Negligible	7
σ	n/a	Does not appear in R_0	—
ω	n/a	Does not appear in R_0	—
b	n/a	Does not appear in R_0 explicitly	—

Such analysis is widely used to determine key parameters that significantly affect disease transmission and control strategies ([15],[16]).

4. Conclusion

This study formulates a dengue transmission model coupling human and mosquito populations through awareness, treatment, and reinfection, establishing two equilibria – disease-free (DFE) and endemic (EE) – and deriving R_0 in closed form via the Next Generation Matrix. The Routh–Hurwitz criterion shows that local stability of the DFE is governed entirely by the sign of $R_0 - 1$, confirming R_0 as a sharp threshold parameter, a result corroborated by numerical simulations.

The normalized forward sensitivity analysis further characterizes, in closed form, which parameter combinations govern this threshold analytically. Notably, reinfection alters the endemic equilibrium structurally without affecting R_0 itself, distinguishing the mathematical roles of transmission versus post-infection parameters. Together, these results establish the qualitative and quantitative structure of the equilibria, providing a basis for future extensions such as optimal control, global stability, or parameter estimation from epidemiological data.

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