DOI: 10.5281/zenodo.21400013 | Volume: 1 | Issue: 1 | ISSN: 3141-643X

Ktrend – International Journal of Computational Mathematics and Scientific Computing

Mathematical Modelling of the Impact of Dengue Fever Using Nonlinear Differential Equations

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Abstract

Dengue fever is a mosquito-borne viral disease that remains a major public-health concern in tropical and subtropical regions. This paper studies dengue transmission through a seven-compartment nonlinear differential-equation model consisting of four human classes—susceptible, exposed, infectious and recovered—and three mosquito classes—susceptible, exposed and infectious. The analysis establishes non-negativity of solutions, discusses boundedness and the biologically feasible region, determines the disease-free equilibrium, derives the basic reproduction number using the next-generation matrix method, and examines the threshold behaviour of the disease-free and endemic states. A wider review of dengue modelling literature is included to position the model within classical host–vector theory, SEIR–SEI formulations, reproduction-number analysis, vector control, climate-sensitive transmission and recent African outbreaks. The model indicates that dengue transmission can be interrupted when the reproduction threshold is reduced below unity, while persistence is expected when it exceeds unity. The results reinforce the importance of integrated mosquito control, environmental sanitation, personal protection, surveillance, early diagnosis and appropriate clinical management.

Keywords: dengue fever; SEIR–SEI model; nonlinear differential equations; positivity; boundedness; basic reproduction number; stability.

1. Introduction

Dengue is an arboviral infection transmitted mainly by infected female *Aedes aegypti* mosquitoes and, to a lesser extent, *Aedes albopictus*. Clinical outcomes range from asymptomatic infection and self-limiting febrile illness to severe dengue, which may involve plasma leakage, haemorrhage, shock and organ impairment. The disease burden is shaped by rapid urbanisation, human mobility, unreliable water supply, household water storage, weak vector control, climatic suitability and limitations in disease surveillance [3, 15, 4].

Dengue transmission follows a host–vector cycle. A susceptible mosquito may acquire the virus when it feeds on an infectious person. After the extrinsic incubation period, the mosquito becomes

infectious and may transmit the virus to susceptible humans during subsequent blood meals. Newly infected humans also pass through an intrinsic incubation period before becoming infectious. These biological delays motivate the use of exposed compartments for both humans and mosquitoes in SEIR–SEI models [7, 11, 23].

The African burden of dengue has often been underestimated because symptoms overlap with malaria and other febrile illnesses, while laboratory confirmation and routine surveillance remain limited. Nevertheless, recent outbreaks in West Africa, especially Burkina Faso, have demonstrated that dengue can cause substantial morbidity and mortality. Regional reports have emphasised the importance of improved surveillance, laboratory capacity, risk communication, case management and vector-control programmes [26, 27].

Mathematical modelling provides a systematic means of translating biological assumptions into equations that can be analysed for disease invasion, persistence and control. The classical epidemic theory of Kermack and McKendrick established the importance of threshold quantities in infectious-disease dynamics [14]. For mosquito-borne infections, Ross–Macdonald theory highlighted the roles of mosquito density, biting frequency, vector competence and mosquito survival [21]. Modern dengue models build on these foundations and may incorporate latent infection, demographic turnover, vaccination, multiple serotypes, seasonality, spatial movement, climate, vertical transmission and biological control [1, 17].

The purpose of this paper is to formulate and analyse a seven-class dengue transmission model. The analysis focuses on positivity and boundedness of solutions, the biologically feasible region, the basic reproduction number, disease-free and endemic equilibria, and the stability properties of the system.

2. Literature Review

2.1. Classical host–vector models

Early mathematical work on mosquito-transmitted infection established that pathogen persistence depends on the interaction between human infectiousness and vector survival. Ross introduced a quantitative framework for malaria transmission, and Macdonald subsequently formalised the influence of mosquito density, biting and longevity. Smith et al. reviewed this theoretical tradition and showed its continuing relevance to contemporary vector-borne disease modelling [21].

For dengue, Esteva and Vargas developed influential deterministic host–vector models and derived threshold conditions for disease invasion and persistence [7, 8]. Their studies demonstrated that vector mortality, recruitment and transmission efficiency can strongly influence the basic reproduction number. Garba et al. later showed that nonlinear dengue models may exhibit backward bifurcation under particular epidemiological assumptions, indicating that reducing the reproduction number below one may not always be sufficient in more complex models [11].

2.2. SEIR–SEI dengue formulations

The SEIR structure for humans represents susceptibility, latent infection, infectiousness and recovery. The corresponding SEI structure for mosquitoes reflects the fact that mosquitoes do not recover and normally remain infectious until death. This formulation is widely used because it captures

the intrinsic incubation period in humans and the extrinsic incubation period in mosquitoes while remaining analytically manageable [23, 18].

Systematic reviews have documented substantial growth in dengue modelling and a wide range of model extensions. Aguiar et al. reviewed developments in dengue epidemiology models, including immune interactions and multi-serotype behaviour [1]. Ogunlade et al. examined models of dengue transmission and vector control and highlighted the importance of clearly stated assumptions, parameter interpretation and intervention objectives [17].

2.3. The basic reproduction number and stability

The basic reproduction number, $\mathcal{R}_0$, is a central threshold quantity in epidemic modelling. It represents the expected number of secondary infections generated by a typical infectious individual introduced into a wholly susceptible population. Diekmann et al. provided a general theoretical definition, while van den Driessche and Watmough developed the next-generation matrix procedure now widely used for compartmental systems [6, 25].

In a host–vector system, transmission requires two linked stages: infection passes from an infectious human to a mosquito and then from an infectious mosquito to a human. For this reason, the reproduction number often takes a square-root form. When $\mathcal{R}_0 < 1$, infection introduced near the disease-free state is expected to decline; when $\mathcal{R}_0 > 1$, invasion and persistence become possible. The precise global conclusion, however, depends on the nonlinear structure of the model.

2.4. Vector control, vaccination and biological interventions

Dengue control has traditionally relied on reducing mosquito breeding sites, applying larvicides, using adulticides during outbreaks, improving household protection and promoting community participation. Mathematical comparisons suggest that integrated strategies are more effective than isolated short-term interventions [2, 17]. Biological methods have also received increasing attention. Models predicted that the deployment of *Wolbachia*-infected mosquitoes could reduce dengue transmission [9], and a cluster-randomised trial later demonstrated a substantial reduction in virologically confirmed dengue and hospitalisation [24].

Vaccination models show that population-level effects depend on vaccine characteristics, age, serostatus and local transmission intensity [13, 10]. Vaccination and biological-control compartments are not included in the present baseline formulation.

2.5. Climate, mobility and changing dengue risk

Temperature affects mosquito development, biting, survival and the extrinsic incubation period. Mechanistic studies show that the relationship between temperature and transmission is nonlinear [16, 5]. Climate-change projections suggest that the geographical distribution and seasonality of *Aedes*-borne virus risk may shift over time [20]. Human movement also connects local transmission foci and can spread infection between households, communities and regions [22]. These processes are not explicit in the present autonomous model, but they provide important directions for future extensions.

2.6. Global burden and the African context

Bhatt et al. estimated that the true global number of dengue infections greatly exceeded routinely reported cases [3]. Guo et al. documented the worldwide expansion of outbreaks between 1990 and 2015 [12], while Messina et al. mapped current and future populations at risk [15]. In Africa, limited diagnostic capacity and under-reporting continue to create uncertainty, although recent regional situation reports confirm major outbreaks and a need for integrated arbovirus preparedness [26, 27].

A useful contribution in settings with sparse data is a transparent baseline model whose assumptions and thresholds can be clearly interpreted. The present model serves that purpose by retaining seven epidemiologically meaningful compartments and a direct nonlinear host–vector transmission mechanism.

3. Mathematical Model

3.1. State variables

The total human population is divided into susceptible humans S_h, exposed humans E_h, infectious humans I_h and recovered humans R_h. The mosquito population is divided into susceptible mosquitoes S_v, exposed mosquitoes E_v and infectious mosquitoes I_v. The total populations are

$$N_h = S_h + E_h + I_h + R_h, \qquad N_v = S_v + E_v + I_v.$$

3.2. Governing equations

The dengue transmission dynamics are governed by the following system of seven nonlinear differential equations:

$$\frac{dS_h}{dt} = \mu_h N_h - \beta_{hv} S_h I_v - \mu_h S_h, \tag{1}$$

$$\frac{dE_h}{dt} = \beta_{hv} S_h I_v - (\sigma_h + \mu_h) E_h, \tag{2}$$

$$\frac{dI_h}{dt} = \sigma_h E_h - (\gamma + \mu_h) I_h, \tag{3}$$

$$\frac{dR_h}{dt} = \gamma I_h - \mu_h R_h, \tag{4}$$

$$\frac{dS_v}{dt} = \mu_h N_v - \beta_{vh} S_v I_h - \mu_v S_v, \tag{5}$$

$$\frac{dE_v}{dt} = \beta_{vh} S_v I_h - (\sigma_v + \mu_v) E_v, \tag{6}$$

$$\frac{dI_v}{dt} = \sigma_v E_v - \mu_v I_v. \tag{7}$$

4. Existence, Positivity and Boundedness

Let

$$X(t) = (S_h, E_h, I_h, R_h, S_v, E_v, I_v)^T.$$

Table 1: State variables and parameters of the model.

Symbol	Definition
S_h	Susceptible human population.
E_h	Exposed human population.
I_h	Infectious human population.
R_h	Recovered human population.
S_v	Susceptible mosquito population.
E_v	Exposed mosquito population.
I_v	Infectious mosquito population.
N_h, N_v	Total human and mosquito populations.
μ_h, μ_v	Human and mosquito demographic and mortality parameters.
β_{hv}	Transmission coefficient from infectious mosquitoes to susceptible humans.
β_{vh}	Transmission coefficient from infectious humans to susceptible mosquitoes.
σ_h, σ_v	Progression rates from exposed to infectious classes in humans and mosquitoes.
γ	Human recovery rate.

The right-hand side of equations (1)–(7) is polynomial in the state variables and is therefore locally Lipschitz continuous on $\mathbb{R}^7$. Hence, for every initial condition, a unique local solution exists.

4.1. Positivity of solutions

Theorem 1 (Positivity)**.** *Assume that all model parameters are nonnegative and that*

$$S_h(0), E_h(0), I_h(0), R_h(0), S_v(0), E_v(0), I_v(0) \geq 0.$$

Then every solution of equations (1)–(7) *remains in* $\mathbb{R}^7_+$ *for all* $t \geq 0$ *for which the solution exists.*

Proof. The proof follows directly from the governing equations. From (1),

$$\frac{dS_h}{dt} + \big(\beta_{hv} I_v + \mu_h\big) S_h = \mu_h N_h \geq 0.$$

Using the integrating-factor method gives

$$S_h(t) = S_h(0) \exp\left(-\int_0^t [\beta_{hv} I_v(s) + \mu_h]\, ds\right) + \int_0^t \mu_h N_h(\tau) \exp\left(-\int_\tau^t [\beta_{hv} I_v(s) + \mu_h]\, ds\right) d\tau \geq 0.$$

The remaining human compartments satisfy

$$E_h(t) = E_h(0)e^{-(\sigma_h+\mu_h)t} + \int_0^t \beta_{hv} S_h(\tau) I_v(\tau) e^{-(\sigma_h+\mu_h)(t-\tau)} d\tau \geq 0,$$
$$I_h(t) = I_h(0)e^{-(\gamma+\mu_h)t} + \int_0^t \sigma_h E_h(\tau) e^{-(\gamma+\mu_h)(t-\tau)} d\tau \geq 0,$$
$$R_h(t) = R_h(0)e^{-\mu_h t} + \int_0^t \gamma I_h(\tau) e^{-\mu_h(t-\tau)} d\tau \geq 0.$$

Likewise, the mosquito compartments satisfy

$$S_v(t) = S_v(0) \exp\left(-\int_0^t [\beta_{vh} I_h(s) + \mu_v]\, ds\right) + \int_0^t \mu_h N_v(\tau) \exp\left(-\int_\tau^t [\beta_{vh} I_h(s) + \mu_v]\, ds\right) d\tau \geq 0,$$
$$E_v(t) = E_v(0)e^{-(\sigma_v+\mu_v)t} + \int_0^t \beta_{vh} S_v(\tau) I_h(\tau) e^{-(\sigma_v+\mu_v)(t-\tau)} d\tau \geq 0,$$
$$I_v(t) = I_v(0)e^{-\mu_v t} + \int_0^t \sigma_v E_v(\tau) e^{-\mu_v(t-\tau)} d\tau \geq 0.$$

Each expression is a sum of nonnegative terms. Therefore all seven state variables remain nonnegative. Equivalently, the vector field points inward or is tangent on every coordinate boundary, so the nonnegative orthant is positively invariant. □

Remark 1. *This result establishes the biological admissibility of the model: population variables that start nonnegative cannot become negative.*

4.2. Human population bound

Adding equations (1)–(4) gives

$$\frac{dN_h}{dt} = 0.$$

Therefore,

$$N_h(t) = N_h(0)$$

for all $t \geq 0$. The total human population is constant and consequently each human compartment is bounded above by $N_h(0)$.

4.3. Mosquito population bound

Adding equations (5)–(7) gives

$$\frac{dN_v}{dt} = (\mu_h - \mu_v)N_v.$$

Hence

$$N_v(t) = N_v(0)e^{(\mu_h-\mu_v)t}.$$

Under the biologically meaningful boundedness condition $\mu_h \leq \mu_v$, the mosquito population is bounded for all $t \geq 0$. In particular, if $\mu_h = \mu_v$, then $N_v(t) = N_v(0)$; if $\mu_h < \mu_v$, then $N_v(t)$ decreases

exponentially. Thus a feasible region is

$$\Omega = \left\{X \in \mathbb{R}_+^7 : S_h + E_h + I_h + R_h = N_h(0),\ S_v + E_v + I_v \le N_v(0)\right\}$$

when $\mu_h \le \mu_v$.

5. Disease-Free Equilibrium

At the disease-free equilibrium, all exposed and infectious populations vanish:

$$E_h^0 = I_h^0 = R_h^0 = E_v^0 = I_v^0 = 0.$$

The susceptible populations are equal to their respective total populations. Therefore, the disease-free equilibrium is

$$\boxed{\mathcal{E}_0 = (N_h, 0, 0, 0, N_v, 0, 0).}$$

This is the disease-free state of the model.

6. Basic Reproduction Number

Let the infected-state vector be

$$x = (E_h, I_h, E_v, I_v)^T.$$

At the disease-free equilibrium, the new-infection matrix is

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_{hv} N_h \\ 0 & 0 & 0 & 0 \\ 0 & \beta_{vh} N_v & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

and the transition matrix is

$$V = \begin{pmatrix} \sigma_h + \mu_h & 0 & 0 & 0 \\ -\sigma_h & \gamma + \mu_h & 0 & 0 \\ 0 & 0 & \sigma_v + \mu_v & 0 \\ 0 & 0 & -\sigma_v & \mu_v \end{pmatrix}.$$

The next-generation matrix is FV^{-1}, and its spectral radius gives

$$\boxed{\mathcal{R}_0 = \sqrt{\frac{\beta_{hv}\beta_{vh} N_h N_v \sigma_h \sigma_v}{(\sigma_h + \mu_h)(\gamma + \mu_h)(\sigma_v + \mu_v)\mu_v}}.}$$

The square root reflects the two-stage host–vector transmission cycle. One generation of human infection requires transmission from human to mosquito and subsequently from mosquito to human.

7. Local Stability of the Disease-Free Equilibrium

Proposition 1. *The disease-free equilibrium is locally asymptotically stable when* $\mathcal{R}_0 < 1$ *and unstable when* $\mathcal{R}_0 > 1$.

Proof. The Jacobian evaluated at $\mathcal{E}_0$ separates into demographic directions and an infected subsystem. The next-generation matrix theorem states that the infected subsystem is locally stable exactly when the spectral radius $\rho(FV^{-1})$ is less than one. Since $\rho(FV^{-1}) = \mathcal{R}_0$, all infection-related eigenvalues have negative real parts when $\mathcal{R}_0 < 1$. If $\mathcal{R}_0 > 1$, at least one infection-related eigenvalue has positive real part and the disease-free equilibrium becomes unstable. □

The threshold interpretation is direct. If $\mathcal{R}_0 < 1$, a typical infectious introduction generates fewer than one secondary infection on average and the infection declines near the disease-free state. If $\mathcal{R}_0 > 1$, dengue can invade the susceptible host–vector population.

8. Endemic Equilibrium

Let

$$\mathcal{E}^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*)$$

denote an endemic equilibrium with positive infectious components. Setting the right-hand sides of the system equal to zero gives

$$S_h^* = \frac{\mu_h N_h}{\beta_{hv} I_v^* + \mu_h}, \qquad E_h^* = \frac{\beta_{hv} S_h^* I_v^*}{\sigma_h + \mu_h},$$
$$I_h^* = \frac{\sigma_h E_h^*}{\gamma + \mu_h}, \qquad R_h^* = \frac{\gamma I_h^*}{\mu_h},$$
$$S_v^* = \frac{\mu_h N_v}{\beta_{vh} I_h^* + \mu_v}, \qquad E_v^* = \frac{\beta_{vh} S_v^* I_h^*}{\sigma_v + \mu_v},$$
$$I_v^* = \frac{\sigma_v E_v^*}{\mu_v}.$$

These relations follow directly from equations (1)–(7). A biologically meaningful endemic equilibrium requires all components to be positive. Such persistence is associated with the threshold condition $\mathcal{R}_0 > 1$.

9. Discussion

The model captures the principal biological delays in dengue transmission by including exposed classes in both populations. The human exposed class represents the intrinsic incubation period, while the mosquito exposed class represents the extrinsic incubation period. The model also includes recovery in humans and lifelong infectiousness, until death, in mosquitoes.

The positivity analysis confirms that nonnegative initial populations cannot produce biologically meaningless negative solutions. The human population is exactly conserved by the four human equations. The mosquito total satisfies an exponential demographic equation, and the condition $\mu_h \leq \mu_v$ ensures boundedness under the stated demographic assumptions.

The derived reproduction number shows how transmission is promoted by the two transmission coefficients, the sizes of the susceptible human and mosquito populations, and progression through the human and mosquito incubation periods. Recovery and mosquito mortality reduce the expected completion of the transmission cycle. This agrees with classical dengue models, which identify effective contact and vector survival as important drivers [7, 21, 19].

The results support integrated prevention. Eliminating standing water can reduce mosquito recruitment; screens, repellents, protective clothing and behavioural measures can reduce effective human–mosquito contact; larval and adult control can shorten vector survival; surveillance and early diagnosis can improve outbreak response. Evidence from *Wolbachia* programmes suggests that reducing mosquito competence can also substantially decrease transmission [24].

The model is intentionally simple. It assumes homogeneous mixing, constant coefficients and a single dengue infection process. It does not include seasonal rainfall, temperature variation, human movement, multiple serotypes, temporary cross-immunity, vaccination or spatial heterogeneity. These limitations do not invalidate the baseline threshold analysis, but they define important directions for future work.

10. Conclusion

This study has presented and analysed a seven-compartment nonlinear dengue transmission model. The model divides humans into susceptible, exposed, infectious and recovered classes and mosquitoes into susceptible, exposed and infectious classes. Nonnegative initial conditions lead to nonnegative solutions. The total human population is conserved, while the mosquito population is bounded under the demographic condition stated in the analysis. The disease-free equilibrium is $\mathcal{E}_0 = (N_h, 0, 0, 0, N_v, 0, 0)$, and the basic reproduction number is obtained through the next-generation matrix method. The disease-free equilibrium is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. These findings confirm that reducing host–vector transmission below the threshold required for persistence is central to dengue control. Environmental sanitation, source reduction, personal protection, sustained vector management, surveillance, early diagnosis and appropriate clinical care should therefore be pursued together.

Acknowledgements

The author acknowledges Edo State University, Iyamho, for providing an academic environment supportive of this research.

Conflict of Interest

The author declares no conflict of interest.

Funding Statement

This research received no external funding.

References

[1] M. Aguiar, N. Stollenwerk and S. B. Halstead, "Mathematical models for dengue fever epidemiology: a 10-year systematic review," *Physics of Life Reviews*, vol. 40, pp. 65–92, 2022.

[2] M. Amaku, F. A. B. Coutinho, R. S. Azevedo, M. N. Burattini, L. F. Lopez and E. Massad, "A comparative analysis of the relative efficacy of vector-control strategies against dengue fever," *Bulletin of Mathematical Biology*, vol. 76, pp. 697–717, 2014.

[3] S. Bhatt, P. W. Gething, O. J. Brady, J. P. Messina, A. W. Farlow, C. L. Moyes, et al., "The global distribution and burden of dengue," *Nature*, vol. 496, no. 7446, pp. 504–507, 2013.

[4] O. J. Brady, P. W. Gething, S. Bhatt, J. P. Messina, J. S. Brownstein, A. G. Hoen, et al., "Refining the global spatial limits of dengue virus transmission by evidence-based consensus," *PLoS Neglected Tropical Diseases*, vol. 6, no. 8, e1760, 2012.

[5] O. J. Brady, M. A. Johansson, C. A. Guerra, S. Bhatt, N. Golding, D. M. Pigott, et al., "Modelling adult *Aedes aegypti* and *Aedes albopictus* survival at different temperatures in laboratory and field settings," *Parasites & Vectors*, vol. 6, article 351, 2013.

[6] O. Diekmann, J. A. P. Heesterbeek and J. A. J. Metz, "On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations," *Journal of Mathematical Biology*, vol. 28, pp. 365–382, 1990.

[7] L. Esteva and C. Vargas, "Analysis of a dengue disease transmission model," *Mathematical Biosciences*, vol. 150, no. 2, pp. 131–151, 1998.

[8] L. Esteva and C. Vargas, "A model for dengue disease with variable human population," *Journal of Mathematical Biology*, vol. 38, pp. 220–240, 1999.

[9] N. M. Ferguson, D. T. Kien, H. Clapham, R. Aguas, V. T. Trung, T. N. Chau, et al., "Modeling the impact on virus transmission of *Wolbachia*-mediated blocking of dengue virus infection of *Aedes aegypti*," *Science Translational Medicine*, vol. 7, no. 279, 279ra37, 2015.

[10] S. Flasche, M. Jit, I. Rodríguez-Barraquer, L. Coudeville, M. Recker, K. Koelle, et al., "The long-term safety, public health impact, and cost-effectiveness of routine vaccination with a recombinant, live-attenuated dengue vaccine," *PLoS Medicine*, vol. 13, no. 11, e1002181, 2016.

[11] S. M. Garba, A. B. Gumel and M. R. Abu Bakar, "Backward bifurcations in dengue transmission dynamics," *Mathematical Biosciences*, vol. 215, no. 1, pp. 11–25, 2008.

[12] C. Guo, Z. Zhou, Z. Wen, Y. Liu, C. Zeng, D. Xiao, et al., "Global epidemiology of dengue outbreaks in 1990–2015: a systematic review and meta-analysis," *Frontiers in Cellular and Infection Microbiology*, vol. 7, article 317, 2017.

[13] M. A. Johansson, J. Hombach and D. A. Cummings, "Models of the impact of dengue vaccines: a review of current research and potential approaches," *Vaccine*, vol. 29, no. 35, pp. 5860–5868, 2011.

[14] W. O. Kermack and A. G. McKendrick, "A contribution to the mathematical theory of epidemics," *Proceedings of the Royal Society A*, vol. 115, no. 772, pp. 700–721, 1927.

[15] J. P. Messina, O. J. Brady, N. Golding, M. U. G. Kraemer, G. R. W. Wint, S. E. Ray, et al., "The current and future global distribution and population at risk of dengue," *Nature Microbiology*, vol. 4, pp. 1508–1515, 2019.

[16] E. A. Mordecai, J. M. Cohen, M. V. Evans, P. Gudapati, L. R. Johnson, C. A. Lippi, et al., "Detecting the impact of temperature on transmission of Zika, dengue, and chikungunya using mechanistic models," *PLoS Neglected Tropical Diseases*, vol. 11, no. 4, e0005568, 2017.

[17] S. T. Ogunlade, M. T. Meehan, A. I. Adekunle and E. S. McBryde, "A systematic review of mathematical models of dengue transmission and vector control: 2010–2020," *Viruses*, vol. 15, no. 1, article 254, 2023.

[18] G. R. Phaijoo and D. B. Gurung, "Sensitivity analysis of an SEIR–SEI model of dengue disease," arXiv:2501.17196, 2025.

[19] H. S. Rodrigues, M. T. T. Monteiro and D. F. M. Torres, "Sensitivity analysis in a dengue epidemiological model," *Conference Papers in Mathematics*, vol. 2013, article 721406, 2013.

[20] S. J. Ryan, C. J. Carlson, E. A. Mordecai and L. R. Johnson, "Global expansion and redistribution of *Aedes*-borne virus transmission risk with climate change," *PLoS Neglected Tropical Diseases*, vol. 13, no. 3, e0007213, 2019.

[21] D. L. Smith, K. E. Battle, S. I. Hay, C. M. Barker, T. W. Scott and F. E. McKenzie, "Ross, Macdonald, and a theory for the dynamics and control of mosquito-transmitted pathogens," *PLoS Pathogens*, vol. 8, no. 4, e1002588, 2012.

[22] S. T. Stoddard, A. C. Morrison, G. M. Vazquez-Prokopec, V. Paz Soldan, T. J. Kochel, U. Kitron, J. P. Elder and T. W. Scott, "The role of human movement in the transmission of vector-borne pathogens," *PLoS Neglected Tropical Diseases*, vol. 3, no. 7, e481, 2009.

[23] R. Taghikhani and A. B. Gumel, "Mathematics of dengue transmission dynamics: roles of vector vertical transmission and temperature fluctuations," *Infectious Disease Modelling*, vol. 3, pp. 266–292, 2018.

[24] A. Utarini, C. Indriani, R. A. Ahmad, W. Tantowijoyo, E. Arguni, M. R. Ansari, et al., "Efficacy of *Wolbachia*-infected mosquito deployments for the control of dengue," *New England Journal of Medicine*, vol. 384, pp. 2177–2186, 2021.

[25] P. van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Mathematical Biosciences*, vol. 180, pp. 29–48, 2002.

[26] World Health Organization Regional Office for Africa, *Multi-country Outbreak of Dengue in the WHO African Region: Situation Report*, 2024.

[27] World Health Organization, "Dengue–global situation," Disease Outbreak News, 30 May 2024.

