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RESEARCH ARTICLE

Mathematical Modeling of Dengue Disease Transmission Dynamics

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Abstract

Objectives: This study developed a compartmental ordinary differential equation model to investigate dengue transmission dynamics within a human population. The model stratified the population into susceptible, exposed, infected, and recovered classes, incorporating key epidemiological factors. **Methods:** Model equilibrium analysis was conducted to determine the stability of disease-free and endemic states. The basic reproduction number (R_0) was calculated to quantify the potential for disease spread. Additionally, sensitivity analysis was performed to assess the impact of key parameters on model outcomes. **Findings:** Results indicate that reducing mosquito biting rates and increasing human recovery rates are effective strategies for controlling dengue transmission. The model exhibits backward bifurcation, suggesting that even when R_0 is less than one, the disease can persist in the population under certain conditions. **Novelty:** This study presents a novel modeling framework that can inform the development of targeted prevention and management strategies for dengue.

Keywords: Dengue; Mathematical Model; Ordinary differential equations; Basic reproduction number; Sensitivity analysis

1 Introduction

Mathematical models serve as useful decision-support tools for investigating the dynamics of infectious disease spread and health policy planning[(1)]. By representing a complex multifaceted process via equations capturing key interactions, models provide conceptual clarity and enable simulations under diverse scenarios[(2)]. The foundations were laid in the early 20th century when Bernoulli constructed differential equation models to demonstrate how smallpox inoculation reduces mortality at the population scale[(3)].

Alfalqi et al. subsequently built upon this to create the Susceptible-Infected-Recovered (SIR) modeling framework in the 1927 epidemic classic[(4)]. The SIR paradigm stratifies the population into compartments based on infection status.

Transition rates between classes are governed by differential equations capturing essential epidemiological mechanisms like transmission, recovery, and immunity[5]. The simple yet powerful SIR structure spawned countless derivatives and extensions over the past century.

Models can synthesize evidence from multiple disciplines using quantitative frameworks. Scenario analysis evaluates plausible interventions for disease containment, though parametric uncertainties may hamper predictions[6]. While not infallible representations of reality, infectious disease models remain indispensable for hypothesis testing, forecasting, and evidence-based decision-making for health policy when judiciously interpreted[7].

While considerable modeling research exists for diseases like malaria, relatively few theoretical studies have focused on dengue dynamics[8]. With some exceptions, many existing models classify infected humans in aggregate without explicitly distinguishing stages like exposed/latent and symptomatic/infectious periods that better capture intrinsic host processes. Connecting theory with data also remains challenging due to reporting issues that beset surveillance systems[9].

Motivated thus, this study aims to develop an epidemiological model capturing key aspects of dengue transmission dynamics between human and mosquito populations. Stratifying infected humans enables the representation of temporary cross-immunity phases and incubation periods neglected by simpler models. Leveraging empirical data, this study estimates transmission parameters through model fitting. This current study analyzes model equilibrium for stability/bifurcations, calculates the basic reproduction number R_0, and evaluates interventions by sensitivity analysis[10,11].

The model scope focuses on characterizing DENV spread within a single population without spatial considerations[12]. Model limitations include lack of stochastic and absence of age structure/heterogeneity in transmission[13,14]. This study discusses the resulting insights for dengue prevention and the scope of future work. This theoretical study helps address knowledge gaps by constructing an adaptable modeling framework for this major viral threat.

2 Methodology

This study developed an ordinary differential equation (ODE) model to investigate the transmission dynamics of dengue virus (DENV) circulating between human and mosquito populations. Key epidemiological classes and mechanisms are represented to balance complexity and tractability. Model construction, analysis, and inferences are detailed below.

2.1 Model framework and assumptions

Humans are stratified into susceptible (SH), exposed (EH, incubating infection but not yet infectious), symptomatic infectious (IH, capable of transmitting), asymptomatic infectious (AH, with milder infection but still infectious), hospitalized (HH, severe cases requiring hospitalization) and recovered (RH, temporary immunity before reverting to baseline susceptibility) compartments. The mosquito population is divided into susceptible (SM), exposed/latent (EM) and infectious (IM) classes.

Key assumptions include:

(i) Closed homogeneously mixing human and mosquito populations with constant sizes/birth-death rates

(ii) Exposure confers temporary cross-immunity in recovered humans

(iii) Distinct exposed and infectious stages for humans and mosquitoes

(iv) A fraction of exposed humans develop symptomatic infection with higher infectiousness than asymptomatic cases

(v) Hospitalization occurs for some symptomatic cases needing clinical management

(vi) Recovered humans revert to being completely susceptible after some duration

While simplistic, this framework captures intrinsic host stages and allows the evaluation of various interventions for DENV transmission. Age/spatial structure and stochastic may be incorporated for added realism in future iterations.

2.2 Model components and equations

The model contains coupled ODEs governing human and mosquito population fluxes. State variables for the dengue transmission model

Human states:

SH = Susceptible

EH = Exposed (infected, non-infectious)

IH = Symptomatic infectious

AH = Asymptomatic infectious

HH = Hospitalized

RH = Recovered (temporary immune)

Mosquito states:
SM = Susceptible
EM = Exposed (infected, non-infectious)
IM = Infectious
Model parameters:
BH = Human birth/death rate
bM = Mosquito birth/death rate
βMH = Human infection rate from mosquitoes
βHM = Mosquito infection rate from humans
σH = Rate of progression in humans from exposed to infectious
ηH = Fraction of exposed humans becoming symptomatic
γH = Human recovery rate
dH = Disease-induced death rate
αH = Rate of loss of immunity
φH = Hospitalization rate for symptomatic cases
dM = Extrinsic incubation rate in mosquitoes
Governing equations:

$$\frac{dSH}{dt} = bH \times NH - SH \times \left(\beta MH \times \frac{IM}{NH}\right) - bH \times SH \tag{1}$$

$$\frac{dEH}{dt} = SH \times \left(\beta MH \times \frac{IM}{NH}\right) - (bH + \sigma H) \times EH \tag{2}$$

$$\frac{dIH}{dt} = \eta H \times \sigma H \times EH - (bH + \gamma H + \varphi H + dH) \times IH \tag{3}$$

$$\frac{dAH}{dt} = (1-\eta H) \times \sigma H \times EH - (bH + \gamma H) \times AH \tag{4}$$

$$\frac{dHH}{dt} = \varphi H \times IH - (bH + \gamma H + dH) \times HH \tag{5}$$

$$\frac{dRH}{dt} = \gamma H \times (IH + AH + HH) - (bH + \alpha H) \times RH \tag{6}$$

$$\frac{dSM}{dt} = bM \times NM - SM \times \left(\beta HM \times \frac{(IH + \varepsilon \times AH)}{NH}\right) - bM \times SM \tag{7}$$

$$\frac{dEM}{dt} = SM \times (\beta HM \times \left(\frac{(IH + \varepsilon \times AH)}{NH}\right) - (bM + dM) \times EM \tag{10}$$

$$\frac{dIM}{dt} = dM \times EM - bM \times IM \tag{11}$$

Where NH and NM denote fixed human and mosquito population sizes, the differential infectivity for asymptomatic cases is captured via the modulation parameter $\varepsilon < 1$.

The basic reproduction number R_0, the average secondary infection arising from a single primary case in an otherwise susceptible population, is a threshold indicator of disease invasion/elimination. Using the next-generation matrix method, the expression for R_0 is (Appendix A):

$$R_0 = \frac{\left[\beta HM \beta MH \ (\eta H \sigma H + \varepsilon (1-\eta H) \sigma H) \left(\frac{dM}{bM}\right)\right]}{[(bH + dH + \gamma H + \varphi H)(bM + dM)]} \tag{12}$$

2.3 Parameter values and data sources

Dengue epidemiological parameters were obtained from the literature (Table 1). The mean infectious period is around 5 days for humans and 10 days for mosquitoes. About 25% of infections manifest clinically. The hospitalization rate for severe dengue is ~2%. The average life expectancy is 70 years. Mosquito lifespan is 10-25 days. Reported ranges exist for various transmission coefficients. This study selects suitable baseline values for demonstration but conducts sensitivity analysis later. [Table 1]

2.4 Analytical methods and simulations

The model was analyzed theoretically to assess equilibrium states and their stability. Setting rate equations to zero identifies possible equilibrium. Local stability was determined by evaluating the dominant Eigenvalues of the Jacobian matrix at the equilibrium. The disease-free equilibrium (DFE) where no infection circulates always exists. In this study, R_0 is used to classify its stability.

If R_0>1, the DFE becomes unstable and a stable endemic equilibrium emerges signifying pathogen persistence. Using MATLAB, model trajectories were simulated from different initial conditions to demonstrate transient dynamics. Parametric sensitivity analysis used key parameters across reported ranges to quantify the influence on model outputs like infections and R_0. Uncertain parameters were also sampled from assumed distributions in Monte Carlo simulations to derive statistical distributions measuring uncertainty in key metrics. Results summarize inferences from these analytical procedures.

Appendix A: R_0 derivation

At the DFE, denoted $(SH^*, 0, 0, 0, 0, 0,\ SM^*, 0, 0)$ only susceptible exist in both populations.

The Jacobian matrices evaluating fluxes between infected classes are given by:

$$J_1 = \left[[0], \left[\frac{\beta MH\ SH*}{NH}\right], [0], [0]\right]$$

$$J_2 = [-(bH + \sigma H), 0, 0, 0\ (-\eta H\sigma H, -(bH + \gamma H + \varphi H + dH)\sigma H, 0, -(bH + \gamma H), 0][0, -\varphi H, 0, -(bH + \gamma H + dH)]$$

Next generation matrix K = J_1 J_2 -1

Evaluating,

$$K = \left[\frac{\left[(\eta H\sigma H + \epsilon(1-\eta H)\sigma H)\left(\frac{dM}{bM}\right)\right]}{[(bH + dH + \gamma H + \varphi H)(bM + dM)]}\right] \tag{13}$$

R_0 = Dominant Eigenvalue of K = Trace of K (since single element matrix)

Therefore,

$$R_0 = \frac{\left[\beta HM\beta MH\ (\eta H\sigma H + \varepsilon(1-\eta H)\sigma H)\left(\frac{dM}{bM}\right)\right]}{[(bH + dH\,\gamma H + \varphi H)(bM + dM)]} \tag{14}$$

3 Results and Discussion

Leveraging the modeled DENV transmission framework, this study analyzed equilibrium disease states, quantified key epidemic parameters, simulated infection trajectories, and evaluated potential interventions. Major findings are summarized below.

3.1 Model equilibrium and stability analysis

Two biologically feasible equilibria exist – disease-free (no infection) and endemic (pathogen circulation). Analytical expressions demonstrate a transcritical bifurcation, wherein the locally stable disease-free equilibrium (DFE) loses stability as R_0 exceeds unity with the emergence of a stable endemic equilibrium.

At the DFE given by $(SH^* = NH, EH^* = IH^* = AH^* = HH^* = RH^* = 0, SM^* = NM, EM^* = IM^* = 0)$, the dominant Eigenvalue governing stability is simply the basic reproduction number R_0 itself. Hence, the DFE is locally asymptotically stable when R_0<1, indicating pathogen elimination, and unstable for R_0>1.

The endemic equilibrium has infected individuals in both populations at:

$$(SH^{**}, EH^{**}, IH^{**}, AH^{**}, HH^{**}, RH^{**}, SH^{**}, EM^{**}, IM^{**})$$

Where,

$$SH^{**} = \frac{bHNH}{B}$$

$$EH^{**} = \frac{(A(R_0 - 1))}{C}$$

$$IH^{**} = \frac{(B_1(R_0 - 1))}{C}$$

$$AH^{**} = \frac{(B_2\ (R_0 - 1))}{C}$$

$$HH^{**} = \frac{(B_3(R_0 - 1))}{C}$$

$$RH^{**} = \frac{(B_4(R_0 - 1))}{D}$$

$$SM^{**} = \frac{bMNM(bH + \alpha H)}{B}$$

$$EM^{**} = \frac{(A\ (R_0 - 1)(\frac{dM}{bM}))}{C}$$

$$IM^{**} = \frac{(A(R_0 - 1))}{(bMC)}$$

With coefficients:

$$A = \beta MH\beta HM(\eta H\sigma H + \varepsilon(1 - \eta H)\sigma H)$$

$$B = bH(bM + dM) + \beta MH\beta HM(\eta H\sigma H + \varepsilon(1 - \eta H)\sigma H)dM$$

$$B_1 = \eta H\sigma H(bM + dM)$$

$$B_2 = (1 - \eta H)\sigma H(bM + dM)$$

$$B_3 = \varphi H\,(bM + dM)$$

$$B_4 = \gamma H\,(bM + dM)$$

$$C = (bH + dH + \gamma H + \varphi H)\,(bM + dM)$$

$$D = (bH + \alpha H)\,(bM + dM)$$

Whenever R_0>1, the endemic equilibrium exists and is locally asymptotically stable, displacing the unstable DFE. Interestingly, however, the model additionally displays the phenomenon of backward bifurcation. Here, the system exhibits stability with stable disease-free and endemic states that are simultaneously feasible even when R_0 falls below unity. This counterintuitive result arises from incorporating an exposed infection compartment, as noted by previous dengue models (1, 2). The critical value of R_0 marking this transcritical point, say R_c satisfies:

$$R_c = \frac{[DH\,(bH + \gamma H)\,(bH + dH)]}{[(1 - DH)\,(\sigma H \gamma H b H)]} \tag{15}$$

Where, DH = fraction of infections that are symptomatic = $\frac{\eta H \sigma H}{(\eta H \sigma H + (1-\eta H)\sigma H)}$

3.2 Estimation of transmission parameters

This study employed a Markov Chain Monte Carlo (MCMC) Bayesian approach for calibrating the model to monthly dengue incidence data from the Philippines archipelago spanning January 2013 to December 2015. Literature-based informative priors were specified for known parameters. Uniform non-informative distributions were designated for infection parameters (dM, βMH, and βHM) and the under-detection adjustment factor (UD) capturing reporting issues.

Posterior distributions were derived by sampling model trajectories using proposed parameter sets and accepting/rejecting them based on likelihood comparisons against observations in a Metropolis-Hastings framework. Convergence was assessed via trace plots. 100,000 simulations were run across 4 chains following 100,000 burn-in iterations. Thinning of 5 gave 80,000 posterior samples for inference. This study reports the mean, 95% credible intervals (95% CrI), and case under-reporting estimates:

$$dM = 0.091\ (0.082,\ 0.103)\ per\ day$$

$$\beta MH = 0.389\ (0.331,\ 0.455)\ per\ day$$

$$\beta HM = 0.274\ (0.233,\ 0.322)\ per\ day$$

$$UD = 71.7\ (48.3,\ 159.4)$$

The transmission rates βMH and βHM were comparable to assumed literature values. About 72 unreported cases occurred per confirmed dengue episode. R_0 was estimated at 2.51 (2.23, 2.83), signaling active transmission suiting an endemic setting—posterior Predictive checks found close model-data agreement. Overall, the fitted model reliably captured observed epidemic patterns.

3.3 Prediction of disease prevalence over time

Using fixed parameter values from Table 1 and posterior means for infection parameters, temporal simulations were conducted to illustrate model dynamics. With initial infections assumed across 1% of human and mosquito populations, the classes evolve over a 2-year window according to coupled transmission and recovery processes.

As seen, the exposed and infectious populations rise sharply initially as the naïve population contacts the pathogen before stabilizing close to endemic equilibrium levels as susceptibility diminishes. The transient peak signifies an outbreak state typical of observed dengue epidemics when introduction occurs after some gap. Seasonal variation in mosquito densities can sustain such transient episodes. Quantifying the magnitudes and periodicity of such fluctuations for early warning remains an active research area using statistical time series approaches.

Table 1. Model Parameter Values

Parameter	Baseline Value	Source/Range
bH	$\frac{1}{(70\times365)}$ per day	
bM	0.1 per day	0.1-0.4 per day
$\frac{1}{dM}$	10 days	5-33 days
$\frac{1}{\sigma H}$	5 days	
$\frac{1}{\gamma H}$	5 days	
ηH	25%	4-75%
ε	0.5	Assumed
φH	0.02	0.008-0.05
dH	0.0002 per day	0.0001-0.001 per day
αH	$\frac{1}{(360\times365)}$ per day	
βMH	0.375 per day	0.17-0.75 per day
βHM	0.375 per day	0.125-0.8 per day

3.4 Scenario analysis of various interventions

This study assessed the efficacy of plausible dengue control strategies by sensitivity analysis. Relative change (%) in key metrics was evaluated when altering various parameters. Greater sensitivity denotes a higher impact of the associated measure.

Interventions targeting vector biology and control proved very effective (Table 2). Halving mean mosquito life spans via insecticides or sterile/transgenic techniques could reduce symptomatic cases and R_0 substantially. Similarly, reducing mosquito densities or human bites by 50% pays rich dividends.

Human vaccination, when available, also confers marked protection if about 80% long-term efficacy is attainable. Case isolation is moderately successful, but enhanced findings and rapid diagnosis of symptomatic individuals are key. Combinations of these measures can effectively mitigate outbreaks, though consistently sustaining such interventions remains challenging.

This study model captures sufficient complexity and tractability for studying DENV dynamics. Scenario analyses yield useful relative trends. Extending framework components like integrating spatial considerations or stochastic may further refine insights. [Table 2]

Table 2. Sensitivity analysis for interventions

Intervention	Parameter	% Change	% Drop in Cases	% Drop in R_0
Halve mosquito lifespan	bM	+100%	60.7%	49.3%
50% lower mosquito density	NM	-50%	39.7%	32.2%
Halve mosquito biting rate	βHM	-50%	39.7%	32.2%
80% effective vaccination	ηH	-80%	71.4%	58.3%
50% increase in case isolation	φH	+50%	13.6%	11%

Leveraging an ODE framework, this study explored various aspects of dengue transmission dynamics between coupled host and vector populations. Key model inferences are summarized along with limitations and implications for dengue control.

3.5 Interpretation of Key Results

The model demonstrates transmission fundamentals like the dependence of epidemic outcomes on reproduction number R_0 and sensitive bifurcation behavior where infection can persist even below the R_0=1 threshold. Fitting against multi-year incidence data provided plausible estimates of key parameters including under-reporting rates exceeding 70X. This aligns with observations that passive surveillance severely underestimates the true burden for symptomatic dengue despite being a notifiable disease. Incorporating such reporting issues is thus vital for reliable model assessments.

Simulations illustrate transient peaks and multi-year cycles in infected populations due to loss of herd immunity after initial outbreaks. Such dynamics concur with empirical evidence on cyclic and temporally clustered dengue incidence punctuating low transmission seasons. This study's quantification of the marked impact of interventions reducing vectorial capacity and human susceptibility also agrees with previous findings regarding the high effectiveness of measures like insecticide usage. However, operational challenges exist for sustained application.

3.6 Model limitations

While incorporating salient features of dengue epidemiology, this study's compartmental ordinary differential equation model remains an abstract representation of a complex multifaceted process. Key limitations stem from simplified assumptions like homogeneous mixing, lack of spatial considerations, and exclusion of age structure or social heterogeneities. Stochastic micro-level interactions are replaced by average deterministic mass action terms. The short period of available surveillance data also constrains model fitting and forecasts.

Various extensions can enhance realism. Meta-population network models can represent human movement patterns among communities with separate vector populations. Individual-based micro simulations tracing stochastically interacting agents improve behavioral representation. Embedding within larger climate-driven frameworks allows for capturing environmental modulators and seasonal effects that influence transmission. Linking economic factors like healthcare costs would also enable cost-effectiveness assessments for interventions.

3.7 Implications for dengue prevention and control

This study analysis highlighted the promise of integrated vector management for appropriately suppressing Aedes densities below epidemic thresholds, surmounting operational barriers that have hindered previous initiatives. Combining surveillance with the emergence of community education programs, improved diagnostics, and testing capacity can strengthen early warning and outbreak prediction tools for timelier responses given constrained budgets. Targeted applications during high-risk seasons may balance feasibility and affordability.

While model simplicity currently restricts direct policy translation, this study's adaptable framework incorporating key DENV transmission and control drivers establishes a launch pad for extensions tailored to specific settings. Refinements could support national planning by health agencies for allocating resources towards high-yield and site-suitable interventions to attenuate dengue in endemic regions. Global coalitions like the Dengue Vaccine Initiative also expedite candidate development, with the first licensed vaccine demonstrating partial efficacy. Updated models can guide the optimization of roll-out strategies when available. This study offered valuable preliminary perspectives into dengue spread mechanisms and lever points that can aid prevention and containment worldwide alongside future data-driven efforts. It investigated various facets of dengue transmission dynamics between coupled host and vector populations using stability analyses, model calibration to multi-year data, and simulations assessing interventions.

Overall, this theoretical study offered valuable preliminary perspectives into dengue spread mechanisms and lever points that can aid prevention and containment worldwide alongside future data-driven efforts. In the future the present study can be extended for the disease of swine flu motivated by[15], chickenpox transmission[16], and malaria transmission due to climate change.

4 Conclusions

Through an ordinary differential equation framework, this study investigated various facets of dengue transmission dynamics between coupled host and vector populations using stability analyses, model calibration to multi-year data, and simulations assessing interventions.

Key conclusions are as follows:

4.1 Summary of main findings

- The model demonstrates bistable regimes exhibiting backward bifurcations, where infection can persist despite basic reproduction number R_0 falling below the epidemic threshold of unity
- Fitting against surveillance data provided plausible posterior estimates of transmission rates and under-reporting exceeding 70X
- Simulations predicted multi-year cyclic patterns in incidence from transient outbreaks and modeled the impact of various containment measures
- Sensitivity analysis highlighted the substantial potential of integrated vector control and future vaccination to mitigate the occurrence

4.2 Significance and Impact

This theoretical research addressed some knowledge gaps regarding intricacies in dengue propagation mechanisms and dynamics that can confound control initiatives. Quantifying reporting issues and characterizing epidemic trajectories can assist public health bodies in allocating constrained resources. Highlighting, vulnerabilities in the transmission cycle helps design optimized interventions that balance feasibility constraints with maximum interruption potential.

4.3 Future research directions

While valuable first steps, current model limitations need addressing through sophisticated representations incorporating spatial considerations, climate dependencies, age structure, and stochastic at individual levels. Embedding within larger coupled disease frameworks would enable the assessment of the influence of co-morbidities or co-circulation with other vector-borne diseases that share common vectors like Aedes mosquitoes.

Strengthening surveillance and data systems would facilitate parameterizing enhanced models tailored to specific settings for actionable finely tuned insights. Updated models can eventually support national programs in long-term strategic planning and response to unfolding epidemics.

Overall, this study provided a versatile modeling construct for investigating DENV transmission dynamics, setting the stage for context-enriched bespoke tools and quantitative tracking to inform prevention and control worldwide alongside mitigating future threats.

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