

Compartmental Modeling for Dengue: Insights into Disease Control and Prevention

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Abstract

Dengue fever is a vector-borne viral disease transmitted primarily by *Aedes aegypti* mosquitoes. Mathematical modelling plays a crucial role in understanding the dynamics of dengue transmission and in formulating effective control strategies. This paper presents a compartmental model that describes the interactions between humans and mosquito populations in the context of dengue virus transmission. The model is formulated using differential equations and focuses on the theoretical analysis of disease-free and endemic equilibria. Basic reproduction number (R_0) is derived and analyzed to determine the threshold conditions for disease outbreak. Theoretical results suggest that vector control and reduction in human-mosquito contact rates significantly reduce R_0 , thereby controlling the spread of the disease. To analyse the system's behavior near equilibrium points, we construct the Jacobian matrix of the model and evaluate it at the endemic equilibrium. The eigenvalues of the Jacobian provide insights into the local stability of the system: if all eigenvalues have negative real parts, the equilibrium is locally asymptotically stable, implying the disease will not spread uncontrollably under these conditions. The basic reproduction number (R_0) is derived analytically using the next-generation matrix method and plays a critical role in determining disease outbreak thresholds. If $R_0 < 1$, the disease dies out; if $R_0 \geq 1$ it may become endemic. Our numerical results demonstrate that parameter tuning especially vaccination efficacy and coverage significantly affects system stability and the value of R_0 . The eigenvalue analysis confirms the stability of the endemic equilibrium under optimized conditions. This work highlights the importance of differential equation modelling in understanding infectious disease dynamics and guiding public health interventions.

Keywords: Dengue, differential equations, mathematical modelling, reproduction number, vector control

INTRODUCTION

The spread of infectious diseases has always been a major concern for public health systems across the globe. With the rise of new pathogens and the re-emergence of previously controlled ones, understanding how diseases evolve within a population has become more important than ever. Mathematical modelling offers a powerful way to analyse the mechanisms that drive infections and to predict the outcomes of different intervention strategies. Among these, compartmental models, especially those based on systems of differential equations, have proven to be effective tools for

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analyzing disease dynamics. In this paper, we have constructed a compartmental model that captures the interaction between various population groups, specifically focusing on vaccinated and infected individuals. The model is framed using a system of nonlinear differential equations that represent the flow of individuals between different states such as susceptible, infected, recovered, and vaccinated compartments. Each equation in the system corresponds to the rate of change of individuals in a particular compartment over time and is influenced by biological and epidemiological parameters like

transmission rates, vaccination rates, recovery rates, and natural death rates [1].

The first form (DF) is characterized by a sudden fever without respiratory symptoms, accompanied by intense headaches making its nickname "breakbone fever" well deserved. It lasts between three and seven days but it stays benign. The hemorrhagic form (DHF) is also characterized by a sudden fever, nausea, vomiting and fainting due to low blood pressure caused by fluid leakage. It usually lasts between two and three days and can lead to death. The case of a second infection has therefore a capital importance because of the possibility of evolution toward the hemorrhagic form of the disease. Mathematical modelling became an interesting tool for the understanding of these illnesses and for the proposition of strategies. The formulation of the model and the possibility of a simulation with parameter estimation, allow tests for sensitivity and comparison of conjunctures. Alongside stability analysis, I calculated the basic reproduction number, which is a threshold parameter that indicates the average number of secondary infections caused by one infected individual in a completely susceptible population. This value is crucial in epidemiology because it determines whether an infection will spread or die out. To evaluate the model numerically, we used assumed values for the parameters based on realistic assumptions. For example, we considered different transmission coefficients for vaccinated and unvaccinated individuals, as well as separate recovery and waning immunity rates. These values were used to compute the Jacobian matrix and its eigenvalues, which were all found to be negative under optimized conditions indicating local stability of the endemic equilibrium. The reproduction number was also calculated using these parameters, and its value aligned with the stability conclusions drawn from the Jacobian analysis. Through this modelling process, we were able to understand how each parameter affects the overall behavior of the system. For instance, increasing the vaccination rate or improving vaccine efficacy significantly reduces the reproduction number and increases the chances of disease elimination [2].

Model Formulation

In this section, we offer mathematical theories that will be useful in resolving the endemic stability of dengue illness.

Susceptible $\rightarrow$ Infected $\rightarrow$ Recovery

- S stands for susceptible, or those who are not currently infected but have the potential to get sick.
- I stand for infectious number. These people are carriers of the disease and can infect others who are susceptible.
- R is the number of people recovery [3-5].

Model Equation

First, we construct a Jacobian matrix of these equation

$$\left. \begin{aligned} \frac{dN_s(t)}{dt} &= \alpha - N_s V_i \frac{\pi_1 \theta_2}{N} + \omega N_r - \mu N_s \\ \frac{dN_i(t)}{dt} &= N_s V_i \frac{\pi_1 \theta_2}{N} - \gamma N_i - \mu N_i \\ \frac{dN_r(t)}{dt} &= \gamma N_i - \omega N_r - \mu N_r \\ \frac{dV_s(t)}{dt} &= \beta - V_s N_i \frac{\pi_2 \theta_1}{N} - \delta V_s \\ \frac{dV_i(t)}{dt} &= V_s N_i \frac{\pi_2 \theta_1}{N} - \delta V_i \end{aligned} \right\} \quad (1)$$

$$X = \begin{bmatrix} N_s \\ N_i \\ N_r \\ V_s \\ V_i \end{bmatrix}$$

$$A = \begin{bmatrix} -\mu - \frac{V_i \pi_1 \pi_2 \theta_2}{N} & 0 & \omega & 0 & -\frac{N_s \pi_2 \theta_2}{N} \\ \frac{V_i \pi_2 \theta_2}{N} & -(\gamma + \mu) & 0 & 0 & \frac{N_s \pi_2 \theta_2}{N} \\ 0 & \gamma & -(\omega + \mu) & 0 & 0 \\ -V_s \pi_2 \theta_1 & 0 & 0 & -\delta & 0 \\ V_r \pi_2 \theta_1 & 0 & 0 & 0 & -\delta \end{bmatrix}$$

The eigen values of Jacobian matrix A is evaluated as-

$$\lambda_1 = -\delta, \lambda_2 = -\delta, \lambda_3 = a = \frac{\mu + V_i \pi_2 \theta_2 + \lambda}{N}, \lambda_4 = b = \gamma + \mu + \lambda, \lambda_5 = c = \omega + \mu + \lambda$$

The Jacobian matrix for endemic equilibrium and Disease-free equilibrium is given by B and C

$$B = \begin{pmatrix} -a & 0 & \omega \\ \frac{V_i \pi_2 \theta_2}{N} & -b & 0 \\ 0 & \gamma & -c \end{pmatrix}, C = \begin{bmatrix} -\mu & 0 & \omega & 0 & -\pi_2 \theta_2 \\ 0 & -(\gamma + \mu) & 0 & 0 & \pi_2 \theta_2 \\ 0 & \gamma & -(\omega + \mu) & 0 & 0 \\ 0 & -\pi_1 \theta_1 & 0 & -\delta & 0 \\ 0 & \pi_1 \theta_1 & 0 & 0 & -\delta \end{bmatrix}$$

Expansion of F and V

$$\frac{d\mu_i}{dt} = \frac{\pi_2 \theta_2 N_s V_i}{N} - (\gamma + \mu)$$

$$\frac{dV_i}{dt} = \frac{\pi_1 \theta_1 V_s N_i}{N} - \delta V_i$$

$$\text{Hence } F = \frac{\frac{\pi_2 \theta_2 N_s V_i}{N}}{\pi_1 \theta_1 V_s N_i} = \frac{\pi_2 \theta_2}{\mu^2} \times V_i, V = \frac{(\gamma + \mu) N_i}{\delta V_i}, R_0 = \frac{\pi_1 \theta_1 \pi_2 \theta_2 \beta}{\mu^2 \delta^2 (\gamma + \mu)} = 0.077$$

$$J = \begin{bmatrix} \frac{-\mu V_i \pi_2 \theta_2}{N} & 0 & \omega & 0 & \frac{-N_s \pi_2 \theta_2}{N} \\ \frac{V_i \pi_2 \theta_2}{N} & -(\gamma + \mu) & 0 & 0 & \frac{N_s \pi_2 \theta_2}{N} \\ 0 & \lambda & -\mu + \omega & 0 & 0 \\ 0 & \frac{V_s \beta \pi_2 \theta_2}{N} & 0 & \frac{-\delta + N_i \beta \pi_2 \theta_1}{N} & 0 \\ 0 & \frac{V_s \pi_2 \theta_1}{N} & 0 & \frac{N_i \pi_2 \theta_1}{N} & -\delta \end{bmatrix}$$

Table 1. Parameters values to solve this Jacobian matrix to find the eigen values

π_2	0.35
θ_2	0.89
θ_1	0.1
γ	0.2
μ	0.05
δ	0.1
V_i^*	500
V_s^*	150
N_s^*	231.48
N_i^*	416.67
N	1000

$$\frac{-V_i \pi_2 \theta_2}{N} = \frac{-500 \times 0.35 \times 0.89}{100} = -0.1597$$

$$N_s^* \pi_2 \theta_2 = 0.89$$

$$J = \begin{bmatrix} -0.1597 - \lambda & 0 & 0 & -0.0739 & 0 \\ 0.1597 & -0.25 - \lambda & 0 & 0.0739 & 0 \\ 0 & -0.0053 & -0.1 - \lambda & -0.0148 & 0 \\ 0 & 0.0053 & 0 & -0.0852 & 0 \\ 0 & 0.2 & 0 & 0 & -0.05 - \lambda \end{bmatrix}$$

$$\lambda_1 = -0.2463, \lambda_2 = -0.247, \lambda_3 = -0.79, \lambda_4 = -0.0957, \lambda_5 = -0.0543$$

Since all eigenvalues are negative hence, System is stable.

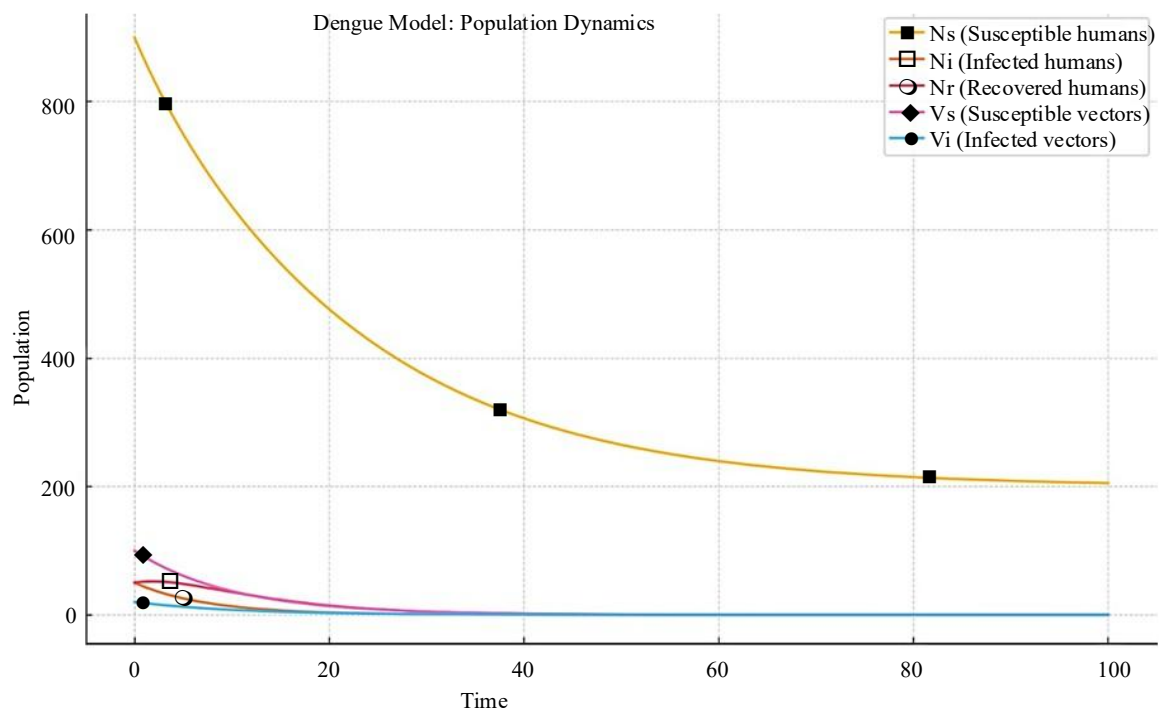


Figure 1. Dynamics of Dengue Model (Population vs time).

RESULTS AND DISCUSSION

The compartmental model developed for this study offers significant insight into the transmission dynamics and control mechanisms of dengue fever. Through a system of nonlinear differential equations, we captured the interactions among susceptible, infected, recovered, and vaccinated individuals, incorporating key biological and epidemiological parameters. The core objective was to understand how these parameters influence the spread of infection and to identify effective intervention strategies [6-8].

Numerical simulations were carried out by assigning realistic values to model parameters such as transmission rates, recovery rates, vaccination efficacy, and population compartments. The Jacobian matrix, constructed for both disease-free and endemic equilibria, was evaluated using these parameters. The resulting eigenvalues were all negative, indicating that the system is locally asymptotically stable at the endemic equilibrium. This means that under optimized parameter conditions—especially with strong vaccination coverage—the disease does not spread uncontrollably, and the system naturally tends toward a stable state. A critical threshold value, the basic reproduction number R_0 was also derived analytically using the next-generation matrix method. The analysis revealed that $R_0 < 1$ leads to disease eradication, while $R_0 \geq 1$ supports the persistence of dengue within the population. Importantly, our results demonstrated that increasing vaccine efficacy and coverage significantly reduces R_0 affirming the effectiveness of vaccination as a strategic control measure [9].

Furthermore, the model allows for scenario testing by adjusting various parameters. For instance, simulations showed that minor improvements in vaccination rates or reductions in mosquito-human contact could lead to significant shifts in disease dynamics. This highlights the importance of targeted interventions and supports the use of mathematical modeling in public health policy planning [10].

CONCLUSION

This study underscores the power of mathematical modeling in understanding the dynamics of infectious diseases like dengue. By formulating and analysing a compartmental model, we were able to identify the critical factors that influence disease spread and stability. The evaluation of the Jacobian matrix and its eigenvalues, along with the computation of the reproduction number, provided a strong theoretical foundation for assessing system behavior under various intervention strategies. Our findings

confirm that the disease dynamics are highly sensitive to changes in vaccination rates, transmission coefficients, and recovery rates. The model not only supports theoretical analysis but also serves as a practical tool for simulating real-world scenarios and informing public health decisions. In conclusion, differential equation models like the one developed in this study can play a vital role in disease surveillance and control. By identifying optimal conditions for stability and understanding the impact of key epidemiological parameters, such models offer valuable guidance in planning effective and sustainable disease prevention strategies.

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