

Analysis of Vector-host SEIR-SEI Dengue Epidemiological Model

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ABSTRACT. Approximately worldwide 50 nations are still infected with the deadly dengue virus. This mosquito-borne illness spreads rapidly. Epidemiological models can provide fundamental recommendations for public health professionals, allowing them to analyze variables impacting disease prevention and control efforts. In this paper, we present a host-vector mathematical model that depicts the Dengue virus transmission dynamics utilizing a susceptible-exposed-infected-recovered (SEIR) model for the human interacting with a susceptible-exposed-infected (SEI) model for the mosquito. Using the Next Generation Technique, the basic reproduction number of the model is calculated. The local stability shows that if $R_0 < 1$ the system is asymptotically stable and the disease dies out, otherwise unstable. The Lyapunov function is also used to evaluate the global stability of disease-free and endemic equilibrium points. To analyze the effect of the crucial aspects of the disease's transmission and to validate the analytical findings, numerical simulations of a variety of compartments have been constructed using MATLAB. The sensitivity analysis of the epidemic model is performed to establish the relative significance of the model parameters to disease transmission.

1. Introduction

Dengue fever incidence has risen dramatically in the recent two decades [1]. Approximately half of the earth's population may be in danger of contracting one of the nearly 390 million new infections that are thought to arise annually. Dengue viral disease is transmitted from mosquitoes

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to humans by mosquitoes, spreading rapidly expanding over the world by its four serotypes. Prior to relatively recently, the female *Aedes aegypti* mosquito which identifies as a primary vector for dengue was mostly found in tropical and subtropical regions [2]. Due to the highly adapted ability in urban regions of the *Aedes aegypti* mosquito, dengue spread all over the world. However, the full extent of the disease's effects remains unclear, and new monitoring strategies are needed, according to issues with underreporting and case misidentification. Other arboviruses, chikungunya as well as zika have lately emerged, posing additional issues for surveillance and management, particularly in South Asia [3].

A wide range of factors (including people, mosquitoes, and the virus) interact with one another to spread the dengue virus in a diverse environment [4]. Studies on dengue transmission face several obstacles due to the space's inherent complexity. A variety of causes are related to the current epidemic. These included worldwide host and vector mobility (which accelerated viral circulation), urban congestion (which encouraged various transmissions through a single infected vector), and the loss of previously effective vector control measures. Temperature, precipitation, and humidity all affect vector development at all stages, from egg viability through adult longevity and dispersion, among other aspects of dengue transmission. Unplanned development, high inhabitants' density, and the instability of rubbish collection all of which support the growth of mosquito breeding sites, which lead to increasing dengue occurrence.

In recent years, epidemiology research and disease control have benefited greatly from the use of mathematical modeling. To understand the disease's nature as well as taking appropriate decisions regarding disease management strategies/interventions and processes, mathematical modeling has become a useful tool. Many scholars studied a deterministic model to study the influence of numerous biological parameters on disease dynamics. Prasad et al. [5] studied a systematic review of deterministic mathematical models for vector-borne viral infections. Bhuju et al. [6] described the fuzzy epidemic SEIR-SEI compartmental model with bed nets and fumigation intervention to simulate the transmission dynamics of dengue disease. Tay et al. [7] constructed a transmission model of SI-SIR dengue epidemiological characteristics model to control dengue in Malaysia. Abidemi et al. [8] analyzed the effect of single vaccine usage and its combination with treatment and adulticide measures on dengue population dynamics in Johor, Malaysia. Aleixo et al. [9] gave a clear explanation of a machine learning model that is used to predict the frequency of dengue outbreaks in Rio de Janeiro. Sow et al. [10] developed a computational Zika Dynamics model to examine the effects of vertical transmission between the

vector population and the host population. Sweilam et al. [11] introduced a unique variable-order nonlinear model of the dengue virus that minimizes intervention dosage and duration through optimum bang-bang management. Abidemi et al. [12] developed and analyzed a two-strain deterministic dengue model based on the SIR modeling framework for the spread of the disease and its management in an area with two coexisting dengue virus serotypes. Asamoah et al. [13] investigated an ideal dengue infection control model with partly immune and asymptomatic patients. Linda et al. [14] examined the discrete-time versions of the SIS and SIR models that are stochastic in nature. To assess the influence of raising awareness through the press on the spread of vector-borne illnesses, a non-linear mathematical model was suggested by Misra et al. [15]. The dynamic SIR model with climatic parameters was discussed by Nur et al. [16] for the features of dengue disease transmission in a closed community.

2. Dengue Transmission Model

The Ross-Macdonald model, which was first designed for malaria, is a classic mathematical model for vector-borne illnesses that monitors infections in humans as well as mosquitos. In this research, we present a compartmental host-vector mathematical model [17] that depicts the Dengue virus transmission dynamics utilizing a susceptible-exposed-infected-recovered (SEIR) model for the human interacting with a susceptible-exposed-infected (SEI) model for the mosquito. The host-vector mathematical model categorizes the overall human (host) population into four classes: susceptible (S_h), exposed (E_h), infectious (I_h), and recovered (R_h), whereas the mosquito (vector) population is divided into three classes: susceptible (S_m), exposed (E_m), and infectious (I_m). Thus, the total human(host) population denoted by N_h is given as

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$$

And total mosquitoes'(vector) population is given by:

$$N_m(t) = S_m(t) + E_m(t) + I_m(t)$$

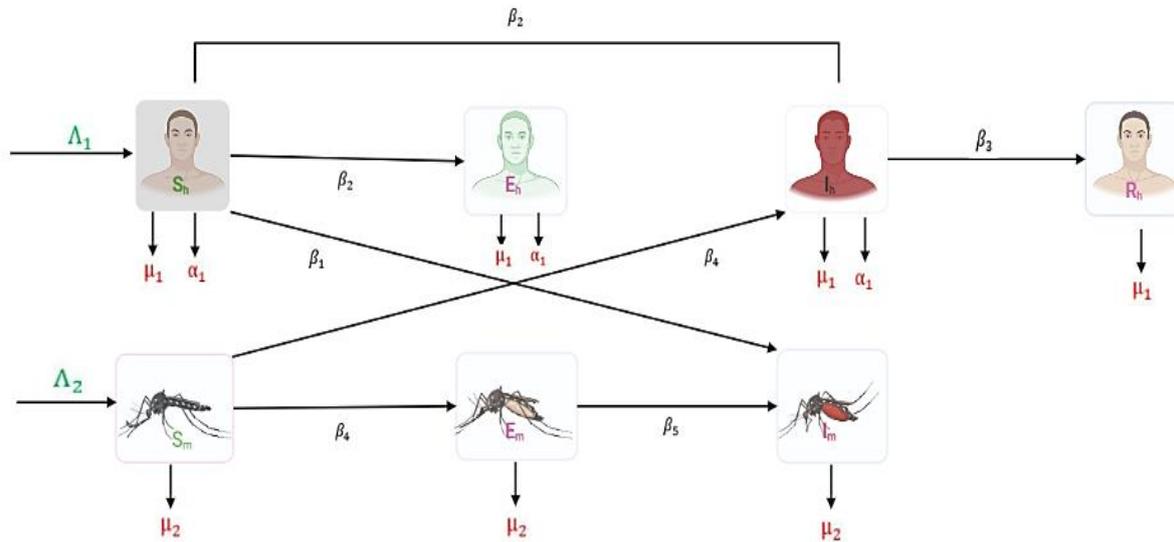


Fig 1. Dengue Virus Transmission Dynamics in Different Population Stages

In our suggested model, we attempt to provide a fresh direction by taking panic, tension, or anxiety into account in the susceptible, exposed, and infected classes to host population. The influence of panic as well as stress, or anxiety on these clusters is discussed in this work. Panic, stress, and anxiety are all harmful to human's health. Anxiety may raise insulin levels, which can have an impact on heart health, diabetes, and blood pressure. At the same time, stress can have a negative impact on human immune system. Extreme stress can impair immunity as well as chronic stress might jeopardize a major health condition. People suffering from panic attacks are more likely to get infected, and the death rate among infected people rises. Therefore, we anticipate that the amount of susceptible, exposed, and infected, is decreasing, i.e., moving to death due to panic, stress, or anxiety. Figure 1 depicts the suggested model's flow diagram as well as the nonlinear system of differential equations that represents the dynamics of host-vector dengue disease, which is represented by:

Human population (h)

$$\begin{aligned}\frac{dS_h}{dt} &= \Lambda_1 - \beta_1 S_h I_m - \beta_2 S_h I_h - \mu_1 S_h - \alpha_1 S_h \\ \frac{dE_h}{dt} &= \beta_2 S_h I_h - \mu_1 E_h - \alpha_1 E_h \\ \frac{dI_h}{dt} &= \beta_1 S_h I_m - \beta_3 I_h - \mu_1 I_h - \alpha_1 I_h \\ \frac{dR_h}{dt} &= \beta_3 I_h - \mu_1 R_h\end{aligned}$$

Vector population (m)

$$\begin{aligned}\frac{dS_m}{dt} &= \Lambda_2 - \beta_4 S_m I_h - \mu_2 S_m \\ \frac{dE_m}{dt} &= \beta_4 S_m I_h - \beta_5 E_m - \mu_2 E_m \\ \frac{dI_m}{dt} &= \beta_5 E_m - \mu_2 I_m\end{aligned}\tag{1}$$

with the initial conditions

$$S_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0, S_m(0) \geq 0, E_m(0) \geq 0, \text{ and } I_m(0) \geq 0,$$

where the biological descriptions of parameters is presented in Table 1.

Table 1. Values for baseline parameters with definitions and biological descriptions of Dengue model

Parameter	Biological descriptions
Λ_1	Recruitment rates of human population
β_1	Infectious rate from vector to host
β_2	Infectious rate within host
μ_1	Humna's natural death rate
α_1	Panic/tension/anxiety rate of human
β_3	Recovery rate of infected human
Λ_2	Recruitment rates of vector population
β_4	Infection rate from human to vector
β_5	Extrinsic incubation of vector
μ_2	Natural death rate of vector population

3. Positivity and boundedness of solutions

The positivity and boundedness of the solutions are crucial features of an epidemiological model. As a result, it is critical to demonstrate that all variables are non-negative for all time $t \geq 0$, implying that any solution with positive beginning values will remain positive for all time $t \geq 0$. So, positivity indicates that the population will survive for a long period.

The dynamical model of the transmission shall be investigated into the biologically feasible regions $\Theta \subset \mathbb{R}_7^+$, Such that

$$\Theta = \left\{ S_h(t), E_h(t), I_h(t), R_h(t), S_m(t), E_m(t), I_m(t) \in \mathbb{R}_7^+ : N_h(t) \leq \frac{\Lambda_1}{\mu_1}, N_m(t) \leq \frac{\Lambda_2}{\mu_2} \right\}$$

Theorem 3.1. The feasible region is positively invariant for the model (1) with the initial condition defined by $\Theta \subset R_7^+$.

Proof. Let $N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$, then

$$\frac{dN_h(t)}{dt} = \frac{dS_h(t)}{dt} + \frac{dE_h(t)}{dt} + \frac{dI_h(t)}{dt} + \frac{dR_h(t)}{dt}$$

Hence

$$\frac{dN_h(t)}{dt} = \Lambda_1 - \mu_1 N_h(t) - \alpha_1 S_h - \alpha_1 E_h - \alpha_1 I_h$$

$$\frac{dN_h(t)}{dt} \leq \Lambda_1 - \mu_1 N_h(t)$$

$$\frac{dN_h(t)}{dt} + \mu_1 N_h(t) \leq \Lambda_1$$

$$N_h(t) \leq \frac{\Lambda_1}{\mu_1} N_h(0) e^{-\mu t}$$

Thus, $N_h(t)$ converges for all non-negative time as t approaches infinity, and the results of the system (1) stay in Θ with starting conditions.

Again, $N_m(t) = S_m(t) + E_m(t) + I_m(t)$, then

$$\frac{dN_m(t)}{dt} = \frac{dS_m(t)}{dt} + \frac{dE_m(t)}{dt} + \frac{dI_m(t)}{dt}$$

Hence

$$\frac{dN_m(t)}{dt} = \Lambda_2 - \mu_2 N_m(t)$$

$$\frac{dN_m(t)}{dt} \leq \Lambda_2 - \mu_2 N_m(t)$$

$$\frac{dN_m(t)}{dt} + \mu_2 N_m(t) \leq \Lambda_2$$

$$N_m(t) \leq \frac{\Lambda_2}{\mu_2} N_m(0) e^{-\mu t}$$

Thus, $N_m(t)$ converges for all non-negative time as t approaches infinity, and the results of the system (1) stay in Θ with starting conditions.

Therefore, the feasible region Θ is positively invariant, attracting all solutions in \mathbb{R}_7^+ .

Theorem 3.2. The solution of the system (1) is positive and bounded for all $S_h(t), E_h(t), I_h(t), R_h(t), S_m(t), E_m(t), I_m(t) \in \mathbb{R}_7^+$ for all $t > 0$.

Proof. To demonstrate the solution's positivity, we need to show that on any hyperplane enclosing the positive vector space \mathbb{R}_7^+ from the system (1), we have

$$\begin{aligned} \left. \frac{dS_h}{dt} \right|_{S_h=0} &= \Lambda_1 \geq 0 \\ \left. \frac{dE_h}{dt} \right|_{E_h=0} &= \beta_2 S_h I_h \geq 0 \\ \left. \frac{dI_h}{dt} \right|_{I_h=0} &= \beta_1 S_h I_m \geq 0 \\ \left. \frac{dR_h}{dt} \right|_{R_h=0} &= \beta_3 I_h \geq 0 \\ \left. \frac{dS_m}{dt} \right|_{m=0} &= \Lambda_2 \geq 0 \\ \left. \frac{dE_m}{dt} \right|_{E_m=0} &= \beta_4 S_m I_h \geq 0 \\ \left. \frac{dI_m}{dt} \right|_{m=0} &= \beta_5 E_m \geq 0 \end{aligned}$$

So, the system (1) solution is positive.

4. Qualitative analysis of model

In this section qualitative analysis of the dengue system (1) by calculating disease free equilibrium (DFE) and the endemic equilibrium (EE) with help of basic reproduction number (E_0).

4.1. Disease-free equilibrium

To calculate the disease-free equilibrium (DFE) E_0 of the dengue system (1), we set the right-hand side of equals to zero and obtain the following expression's

$$E_0 = (S_h^0, E_h^0, I_h^0, R_h^0, S_m^0, E_m^0, I_m^0) = \left(\frac{\Lambda_1}{\mu_1 + \alpha_1}, 0, 0, 0, \frac{\Lambda_2}{\mu_2}, 0, 0 \right)$$

4.2. Basic reproduction number

For the purpose of assessing an infectious disease, a crucial threshold parameter is the basic reproduction number R_0 . It decides whether the disease will disappear or stay in the community throughout time. R_0 is the secondary infections number which may be caused by a single primary infection whereas the population is susceptible. Assume $R_0 > 1$, and one primary infection can generate in several secondary infections. Therefore, the disease-free equilibrium (DFE) is unstable, also an epidemic occurs. The reproduction number for the Dengue system is calculated utilizing the next generation matrix approach [18]. We look at the F^* and V^* matrices, are designated for the new infections' development and classified migration of infective partitions.

$$F^* = \begin{pmatrix} \beta_2 S_h I_h \\ \beta_1 S_h I_m \\ \beta_4 S_m I_h \\ 0 \end{pmatrix}$$

$$V^* = \begin{pmatrix} (\mu_1 + \alpha_1) E_h \\ (\beta_3 + \mu_1 + \alpha_1) I_h \\ (\beta_5 + \mu_2) E_m \\ -\beta_5 E_m + \mu_2 I_m \end{pmatrix}$$

The Jacobian are calculated by taking the partial derivatives of F and V at DFE point E_0 and are as follows:

$$F = \begin{pmatrix} 0 & \beta_2 S_h & 0 & 0 \\ 0 & 0 & 0 & \beta_1 S_h \\ 0 & \beta_4 S_m & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \mu_1 + \alpha_1 & 0 & 0 & 0 \\ 0 & \beta_3 + \mu_1 + \alpha_1 & 0 & 0 \\ 0 & 0 & \beta_5 + \mu_2 & 0 \\ 0 & 0 & -\beta_5 & \mu_2 \end{pmatrix}$$

The basic reproduction of the dengue system is calculated by using the spectral radius of the matrix $R_0 = \rho(FV^{-1})$, which is provided by the following equation

$$R_0^2 = \frac{\beta_1 \beta_4 \beta_5 \Lambda_1 \Lambda_2}{\mu_2^2 (\mu_1 + \alpha_1) (\beta_5 + \mu_2) (\beta_3 + \mu_1 + \alpha_1)}$$

4.3. Endemic equilibrium

The endemic equilibrium point of the dengue dynamical system (1)

$$E_I = (S_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*)$$

Where,

$$S_h^* = \frac{\Lambda_1}{\beta_1 I_m + \beta_2 I_h + \mu_1 + \alpha_1}, \quad E_h^* = \frac{\beta_2 S_h I_h}{\beta_5 + \mu_2}, \quad I_h^* = \frac{\beta_1 S_h I_m}{\beta_3 + \mu_1 + \alpha_1}$$

$$R_h^* = \frac{\beta_3}{\mu_1}, \quad S_m^* = \frac{\Lambda_2}{\beta_4 I_h + \mu_2}, \quad E_m^* = \frac{\beta_4 S_m I_h}{\beta_5 + \mu_2}, \quad I_m^* = \frac{\beta_5 E_m}{\mu_2}$$

5. Stability analysis

The stability study of disease-free and endemic equilibrium is performed in this part. The basic reproduction number (R_0) is used to observe the equilibrium point's stability of the local as well as global. The Jacobian matrix which gives the eigenvalues can be used to do stability analysis.

5.1. Local stability around equilibrium point

Theorem 5.1. For $R_0 < 1$ the disease-free equilibrium (E_0) of the system (1) is locally asymptotically stable and unstable if $R_0 > 1$.

Proof. The Jacobian matrix of the model (1) at the disease-free equilibrium point (E_0) is

$$J(E_0) = \begin{pmatrix} -(\mu_1 + \alpha_1) & 0 & -\beta_2 S_h^0 & 0 & 0 & 0 & -\beta_1 S_h^0 \\ 0 & -(\mu_1 + \alpha_1) & \beta_2 S_h^0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\beta_3 + \mu_1 + \alpha_1) & 0 & 0 & 0 & \beta_1 S_h^0 \\ 0 & 0 & \beta_3 & -\mu_1 & 0 & 0 & 0 \\ 0 & 0 & -\beta_4 S_m^0 & 0 & -\mu_2 & 0 & 0 \\ 0 & 0 & \beta_4 S_m^0 & 0 & 0 & -(\beta_5 + \mu_2) & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_5 & -\mu_2 \end{pmatrix}$$

The four eigenvalues of $J(E_0)$ at the disease-free equilibrium are $-\mu_1$, $-\mu_2$, $-(\mu_1 + \alpha_1)$ (multiplicity 2) and the remaining eigenvalues are given by the following cubic equation

$$\lambda^3 + (v_1 + v_2 + \mu_2)\lambda^2 + (v_1 v_2 + v_1 \mu_2 + v_2 \mu_2)\lambda + v_1 v_2 \mu_2 - \beta_1 \beta_4 \beta_5 S_h^0 S_m^0 = 0$$

Where,

$$v_1 = \beta_3 + \mu_1 + \alpha_1$$

$$v_2 = \beta_5 + \mu_2$$

Now,

$$\lambda^3 + (v_1 + v_2 + \mu_2)\lambda^2 + (v_1v_2 + v_1\mu_2 + v_2\mu_2)\lambda + v_1v_2\mu_2 \left(1 - \frac{\beta_1\beta_4\beta_5 S_h^0 S_m^0}{v_1v_2\mu_2} \right) = 0$$

$$\lambda^3 + (v_1 + v_2 + \mu_2)\lambda^2 + (v_1v_2 + v_1\mu_2 + v_2\mu_2)\lambda + v_1v_2\mu_2(1 - R_0^2) = 0$$

Here,

$$(v_1 + v_2 + \mu_2) > 0$$

$$(v_1v_2 + v_1\mu_2 + v_2\mu_2) > 0$$

$$v_1v_2\mu_2(1 - R_0^2) > 0, \text{ if } R_0 < 1$$

And

$$(v_1 + v_2 + \mu_2)(v_1v_2 + v_1\mu_2 + v_2\mu_2) > v_1v_2\mu_2(1 - R_0^2)$$

$$\left(\text{Since } \frac{(v_1 + v_2 + \mu_2)(v_1v_2 + v_1\mu_2 + v_2\mu_2)}{v_1v_2\mu_2} > 9 > (1 - R_0^2) \right)$$

Therefore, if $R_0 < 1$, all of the preceding requirements are satisfied. As a result, the disease-free equilibrium point E_0 is locally asymptotically stable according to the Routh-Hurwitz criteria; otherwise, it is unstable.

5.2. Global stability around equilibrium point

In this segment, we will evaluate equilibrium points E_0 and E_1 stability. The next two theorems show the results of the stability analysis of these equilibrium sites.

Theorem 5.2. If $R_0 < 1$, the disease-free equilibrium (E_0) is globally asymptotically stable.

Proof. We consider the Lyapunov function of the form in

$$G(t) = (S_h - S_h^0 \ln S_h) + E_h + I_h + R_h + (S_m - S_m^0 \ln S_m) + E_m + I_m$$

Differentiating w.r.t t, we get:

$$G'(t) = \left(1 - \frac{S_h^0}{S_h} \right) S_h' + E_h' + I_h' + R_h' + \left(1 - \frac{S_m^0}{S_m} \right) S_m' + E_m' + I_m'$$

$$\begin{aligned}
G'(t) = & \left(1 - \frac{S_h^0}{S_h}\right) (\Lambda_1 - \beta_1 S_h I_m - \beta_2 S_h I_h - \mu_1 S_h - \alpha_1 S_h) + \beta_2 S_h I_h - \mu_1 E_h - \alpha_1 E_h \\
& + \beta_1 S_h I_m - \beta_3 I_h - \mu_1 I_h - \alpha_1 I_h + \beta_3 I_h - \mu_1 R_h + \left(1 - \frac{S_m^0}{S_m}\right) (\Lambda_2 - \beta_4 S_m I_h - \mu_2 S_m) \\
& + \beta_4 S_m I_h - \beta_5 E_m - \mu_2 E_m + \beta_5 E_m - \mu_2 I_m
\end{aligned}$$

On solving further get:

$$\begin{aligned}
= & \left(1 - \frac{S_h^0}{S_h}\right) \Lambda_1 + (\mu_1 + \alpha_1) \left(1 - \frac{S_h^0}{S_h}\right) S_h^0 + \beta_1 S_h^0 I_m + \beta_2 S_h^0 I_h - \mu_1 E_h - \alpha_1 E_h \\
& - \mu_1 I_h - \alpha_1 I_h - \mu_1 R_h + \left(1 - \frac{S_m^0}{S_m}\right) \Lambda_2 + \mu_2 \left(1 - \frac{S_m^0}{S_m}\right) S_m^0 + \beta_4 S_m^0 I_h - \mu_2 E_m - \mu_2 I_m
\end{aligned}$$

Using the equilibrium condition $(\mu_1 + \alpha_1) S_h^0 = \Lambda_1$ and $\mu_2 S_m^0 = \Lambda_2$ into the above equation

$$\begin{aligned}
G'(t) = & \left(2 - \frac{S_h^0}{S_h} - \frac{S_h}{S_h^0}\right) \Lambda_1 + \left(2 - \frac{S_m^0}{S_m} - \frac{S_m}{S_m^0}\right) \Lambda_2 - I_m (\mu_2 - \beta_1 S_h^0) - I_h (\mu_1 + \alpha_1 - \beta_2 S_h^0 - \beta_4 S_m^0) \\
& - \mu_1 E_h - \alpha_1 E_h - \mu_1 R_h - \mu_2 E_m \\
= & -\Lambda_1 \frac{(S_h - S_h^0)^2}{S_h S_h^0} - \Lambda_2 \frac{(S_m - S_m^0)^2}{S_m S_m^0} - \mu_1 E_h - \alpha_1 E_h - I_h (\mu_1 + \alpha_1 - \beta_2 S_h^0 - \beta_4 S_m^0) \\
& - \mu_1 R_h - \mu_2 E_m - I_m (\mu_2 - \beta_1 S_h^0)
\end{aligned}$$

The above equation shows that $G'(t) \leq 0$ and $G'(t) = 0$ for $S_h = S_h^0$, $E_h = 0$, $I_h = 0$, $R_h = 0$, $S_m = S_m^0$, $E_m = 0$, $I_m = 0$. So, the largest invariance set is the singleton set $\{E_0\}$. Therefore, by using the principle of LaSalle's invariance the disease-free equilibrium (E_0) is globally asymptotically stable.

Theorem 5.3. If $R_0 > 1$, the endemic equilibrium (E_1) is globally asymptotically stable.

Proof. We consider the Lyapunov function of the form in

$$\begin{aligned}
W(t) = & \frac{1}{2} (S_h - S_h^*)^2 + \frac{1}{2} (E_h - E_h^*)^2 + \frac{1}{2} (I_h - I_h^*)^2 + \frac{1}{2} (R_h - R_h^*)^2 + \frac{1}{2} (S_m - S_m^*)^2 \\
& + \frac{1}{2} (E_m - E_m^*)^2 + \frac{1}{2} (I_m - I_m^*)^2
\end{aligned}$$

Differentiating with respect to time t, we get:

$$W'(t) = (S_h - S_h^*) S_h' + (E_h - E_h^*) E_h' + (I_h - I_h^*) I_h' + (R_h - R_h^*) R_h' + (S_m - S_m^*) S_m' + (E_m - E_m^*) E_m' + (I_m - I_m^*) I_m'$$

$$\begin{aligned}
&= (S_h - S_h^*) (\Lambda_1 - \beta_1 S_h I_m - \beta_2 S_h I_h - \mu_1 S_h - \alpha_1 S_h) + (E_h - E_h^*) (\beta_2 S_h I_h - \mu_1 E_h - \alpha_1 E_h) \\
&+ (I_h - I_h^*) (\beta_1 S_h I_m - \beta_3 I_h - \mu_1 I_h - \alpha_1 I_h) + (R_h - R_h^*) (\beta_3 I_h - \mu_1 R_h) + (S_m - S_m^*) (\Lambda_2 - \beta_4 S_m I_h - \mu_2 S_m) \\
&+ (E_m - E_m^*) (\beta_4 S_m I_h - \beta_5 E_m - \mu_2 E_m) + (I_m - I_m^*) (\beta_5 E_m - \mu_2 I_m)
\end{aligned}$$

Using the equilibrium conditions $\Lambda_1 = \mu_1 S_h^* + \mu_1 E_h^* + \mu_1 I_h^* + \mu_1 R_h^* + \alpha_1 S_h^* + \alpha_1 E_h^* + \alpha_1 I_h^*$ and

$\Lambda_2 = \mu_2 S_m^* + \mu_2 E_m^* + \mu_2 I_m^*$ into the above equation

$$\begin{aligned}
W'(t) &= (S_h - S_h^*) (\mu_1 S_h^* + \mu_1 E_h^* + \mu_1 I_h^* + \mu_1 R_h^* + \alpha_1 S_h^* + \alpha_1 E_h^* + \alpha_1 I_h^* - \beta_1 S_h I_m - \beta_2 S_h I_h - \mu_1 S_h - \alpha_1 S_h) \\
&+ (E_h - E_h^*) (\beta_2 S_h I_h - \mu_1 E_h - \alpha_1 E_h) + (I_h - I_h^*) (\beta_1 S_h I_m - \beta_3 I_h - \mu_1 I_h - \alpha_1 I_h) + (R_h - R_h^*) (\beta_3 I_h - \mu_1 R_h) \\
&+ (S_m - S_m^*) (\mu_2 S_m^* + \mu_2 E_m^* + \mu_2 I_m^* - \beta_4 S_m I_h - \mu_2 S_m) + (E_m - E_m^*) (\beta_4 S_m I_h - \beta_5 E_m - \mu_2 E_m) \\
&+ (I_m - I_m^*) (\beta_5 E_m - \mu_2 I_m) \\
&= -\mu_1 (S_h - S_h^*)^2 - \alpha_1 (S_h - S_h^*)^2 + (\mu_1 + \alpha_1) E_h^* (S_h - S_h^*) + (\mu_1 + \alpha_1) I_h^* (S_h - S_h^*) \\
&\quad - \beta_1 S_h I_m (S_h - S_h^*) - \beta_2 S_h I_h (S_h - S_h^*) + \beta_2 S_h I_h (E_h - E_h^*) - (\mu_1 + \alpha_1) E_h^* (E_h - E_h^*) \\
&\quad + \beta_1 S_h I_m (I_h - I_h^*) - (\beta_3 + \mu_1 + \alpha_1) I_h (I_h - I_h^*) + \beta_3 I_h (R_h - R_h^*) - \mu_1 R_h (R_h - R_h^*) \\
&\quad - \mu_2 (S_m - S_m^*)^2 + \mu_2 E_m^* (S_m - S_m^*) + \mu_2 I_m^* (S_m - S_m^*) - \beta_4 S_m I_h (S_m - S_m^*) \\
&\quad + \beta_4 S_m I_h (E_m - E_m^*) - (E_m - E_m^*) E_m (\beta_5 + \mu_2) - \beta_4 S_m I_h (S_m - S_m^*) \\
&= -\mu_1 (S_h - S_h^*)^2 - \alpha_1 (S_h - S_h^*)^2 - (\mu_1 + \alpha_1) \{E_h^* (E_h - E_h^*) - E_h^* (S_h - S_h^*)\} \\
&\quad - (\mu_1 + \alpha_1) \{I_h (I_h - I_h^*) - I_h^* (S_h - S_h^*) - \beta_1 S_h I_m (S_h - S_h^* - I_h + I_h^*) \\
&\quad - \beta_2 S_h I_h (S_h - S_h^* - E_h + E_h^*) - \beta_3 I_h (I_h - I_h^* - R_h + R_h^*) - \mu_1 R_h (R_h - R_h^*) - \mu_2 (S_m - S_m^*)^2 \\
&\quad - \mu_2 \{(E_m - E_m^*) E_m - E_m^* (S_m - S_m^*)\} - \mu_2 \{(I_m - I_m^*) I_m - I_m^* (S_m - S_m^*)\} \\
&\quad - \beta_4 S_m I_h (S_m - S_m^* - E_m + E_m^*) - \beta_5 E_m (E_m - E_m^* - I_m + I_m^*)
\end{aligned}$$

The above equation shows that $W'(t) \leq 0$ and $W'(t)=0$ for $S_h=S_h^*, E_h=E_h^*, I_h=I_h^*, R_h=I_h^*, S_m=S_m^*, E_m=E_m^*, I_m=I_m^*$. So, the largest invariance set is the singleton set $\{E_1\}$. Therefore, by using the principle of LaSalle's invariance the endemic equilibrium E_1 is globally asymptotically stable.

6. Sensitivity analysis of the system

Sensitivity analysis identifies the most effective model parameters that have effects on the Dengue model system's basic reproduction number [19]. Epidemiologists may forecast the important factors that play a significant part in virus-spreading dynamics using such analyses [20]. To avoid or manage the disease's effect, we must first identify the sensitivity induce values, which will give us an idea of which parameters for model would be maintained or monitored.

In the current context, Dengue virus infection is spreading globally at a rapid rate, and this hazardous virus poses a serious threat to the human population. To inhibit the transmission of infection, we must first discover which model parameters are critical to disease transmission. To detect model's such parameters, we must need to evaluate the basic reproduction number variation which depends upon the model parameters; Alternatively, we must compute the normalized forward sensitivity index of the basic reproduction number R_0 with respect to various parameters of the model. Our goal here is to approximate important model parameters that govern the basic reproduction number R_0 . To analyze the sensitivity, we utilize the normalized forward sensitivity index of the basic reproduction number R_0 with regard to the system (1) parameter ρ , which is signified by $\Gamma_{R_0}^\rho = \frac{\partial R_0}{\partial \rho} \cdot \frac{\rho}{R_0}$

Table 2: Sensitivity indices of R_0 evaluated at the baseline parameter values of the model.

Parameter	Sensitivity index
Λ_1	+0.5
Λ_2	+0.5
β_1	+0.5
μ_1	-0.4859780904
α_1	-0.0172362471
β_3	-0.4967856625
β_4	+0.5
β_5	+0.1412037037
μ_2	-1.1412037037

On basic reproduction number R_0 , the parameters higher sensitivity index indicates the more influence sensitive parameter. The system parameter's sensitivity index with positive sign suggests that the basic reproduction number R_0 increases when the parameter increases, and vice versa. In Table 2 and fig. 2, we applied a sensitive index to R_0 in relation to each parameter. According to our research, the most important model parameters are recruitment rates of human population (Λ_1), recruitment rates of vector population (Λ_2), rate of infectious from vector to host (β_1), natural death rate of human (μ_1), panic/tension/anxiety rate of human (α_1), recovery rate of infected human (β_3), infection rate from human to vector (β_4), extrinsic incubation of vector (β_5), and natural death rate of vector population (μ_2). The most significant sensitivity index of the system is the natural death rate of vector population μ_2 .

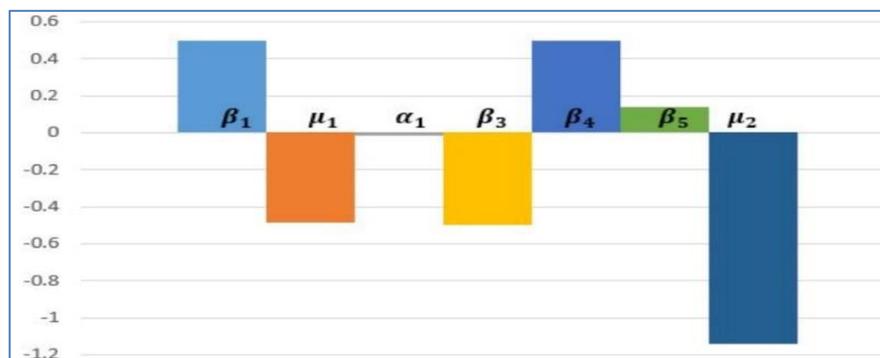


Fig 2. Sensitivity indices of R_0

7. Numerical results

In the numerical part, the suggested model simulation is performed with the assist of MATLAB software. For numerical simulation, the parameter for the system (1) are given in Table 3.

Table 3. System (1) parameters values

Parameter	Values	Units	Reference
Λ_1	.9999	day ⁻¹	[21]
β_1	.8500	day ⁻¹	[22]
β_2	.6794	day ⁻¹	[23]
μ_1	.003468	day ⁻¹	[24]
α_1	.000123	day ⁻¹	assumed
β_3	.5555	day ⁻¹	[22]
Λ_2	.0034	day ⁻¹	[25]
β_4	.7186	day ⁻¹	[26]
β_5	.0062	day ⁻¹	[8]
μ_2	.000244	day ⁻¹	[25]

Figs. 3–6 depict the dynamical system simulation exhibiting the influences of numerous parameters on the transmission dynamics model, demonstrating how parameters are efficient in inducing epidemics on different human populations as well as vector populations. Figs. 3-4 depict the performance of a susceptible and infected host population, as well as the infectious rate from vector to host (β_1) and the rate of panic/tension/anxiety in humans (α_1). Fig. 3(A) describes that there is no effects on different values of β_1 between the early stage 0 to 5 days. Also, it indicates that the susceptible humans decrease with an increase in transmission the infectious rate from vector to host (β_1) and vice-versa. In fig. 3(B) indicates that panic/tension/anxiety rate in humans (α_1) has an impact between 5 to 75 days.

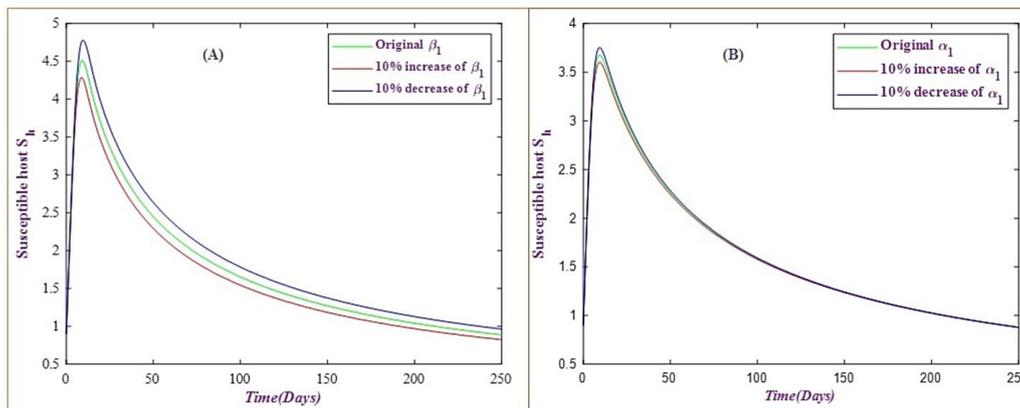


Fig 3. (A) Susceptible host population S_h with different values of β_1 . (B) Susceptible host population S_h with different values of α_1 .

It is interesting to see that in fig. 4 the infected host population decreases 0 to 5 days, after that it grows exponentially. When the interaction between vector to host increases the infected host increases in fig. 4(A), whereas in fig. 4(B) panic/tension/anxiety rate has the opposite effects.

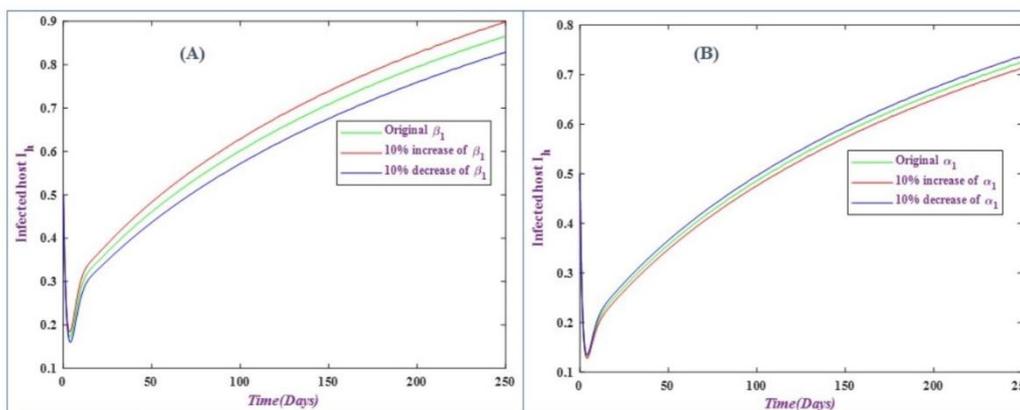


Fig 4. (A) Infected host population I_h with different values of β_1 . (B) Infected host population I_h with different values of α_1 .

Infection rates from humans to vectors and extrinsic incubation of the vector are shown in figures 5–6 together with the behavior of a susceptible and infected vector population. In between the first 40 days, the susceptible vector grows after that it decreases. Fig. 5(A) shows when the infection rates from humans to vectors increases to 10%, the susceptible vectors slightly down to the original. The effects on extrinsic incubation of the vector in fig. 5(B) shows after the 40 days.

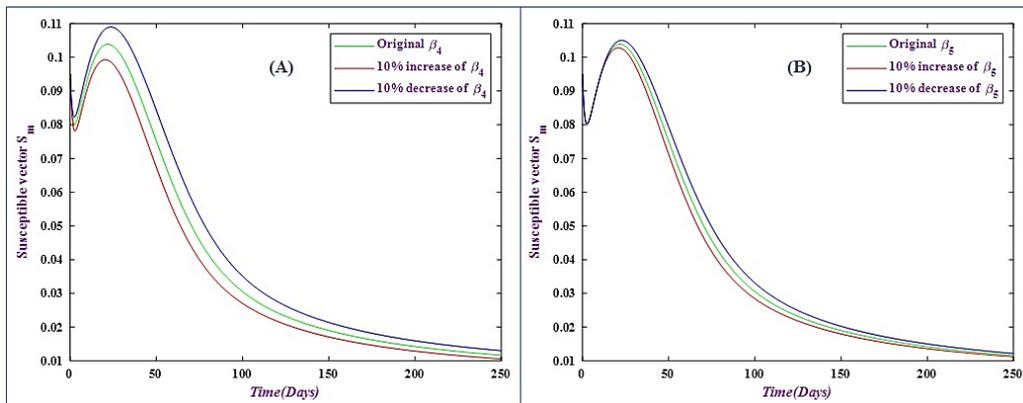


Fig 5. (A) Susceptible vector population S_m with different values of β_4 . (B) Susceptible vector population S_m with different values of β_5 .

Figure 6 shows the fluctuation of the infected vector with respect to time t for various values of β_4 and β_5 . It is obvious that as the value of grows, so does the infected vector. Fig. 6(A) demonstrates the slightly deviation of different β_4 to original, whereas deviation between different β_5 to original is high.

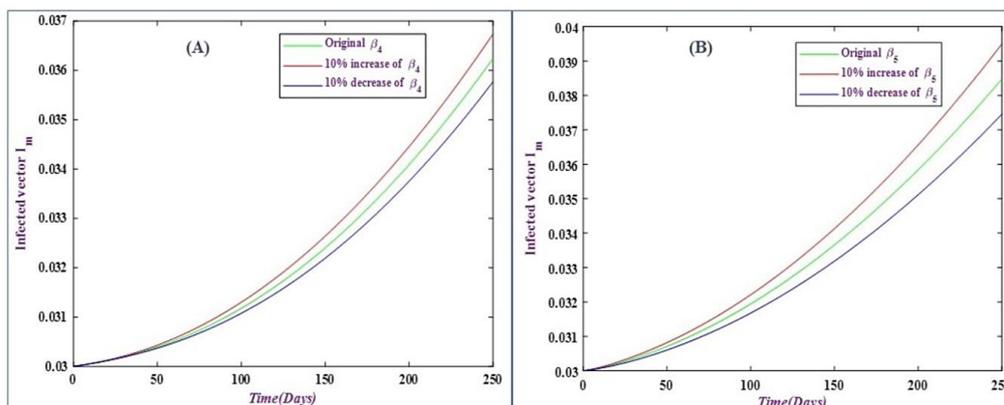


Fig 6. (A) Infected vector population I_m with different values of β_4 . (B) Infected vector population I_m with different values of β_5 .

8. Conclusion

The epidemic vector-borne disease has devastated many nations. Form which, the focus of this article was to analyze dynamic dengue fever. We developed a dynamical mathematical model that would represent them and incorporate the impact of panic, tension, or anxiety on the human population. Model's qualitative analysis was calculated, including illness free equilibrium, endemic equilibrium, and basic reproduction number. Numerical simulation through various parameter settings showed the progression of epidemics, the system's behaviors, and support theoretical results. The maximum sensitivity index was obtained for the vector death rate in the sensitivity study, and this parameter was regarded the most sensitive. It was discovered that increasing the rate of μ_2 results in the greatest decrease in reproduction number, and no other parameter had the same effect on reducing infection. Such model analysis can give vital information to policy makers and health specialists who may be confronted the infectious disease reality.

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