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Analysis for transmission of dengue disease with different class of human population

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Abstract

Objectives: Vector-borne diseases speedily infest the human population. The control techniques must be applied to such ailment and work swiftly. We proposed a compartmental model of dengue disease by incorporating the standard incidence relation between susceptible vectors and infected humans to see the effect of manageable parameters of the model on the basic reproduction number.

Methods: We compute the basic reproduction number by using the next-generation matrix method to study the local and global stability of disease free and endemic equilibrium points along with sensitivity analysis of the model.

Results: Numerical results are explored the global behaviourism of disease-free/endemic state for a choice of arbitrary initial conditions. Also, the biting rate of vector population has more influence on the basic reproduction number as compared the other parameters.

Conclusion: In this paper, shows that controlling the route of transmission of this disease is very important if we plan to restrict the transmission potential.

Keywords: basic reproduction number (R_0); dengue fever; numerical simulation; sensitivity; stability.

Introduction

One of the major concerns of the present era is the birth of a new class of diseases, especially vector-borne diseases like Japanese encephalitis, dengue, malaria, yellow fever etc. Earlier, the sources of these diseases were limited. But, new sources have evolved to a similar kind of ailment, which critically hits the human population. Therefore, one of the significant ways to understand the dynamic phenomenon of disease transmission is mathematical modelling. It helps in determining the intervention techniques for disease control and reduce the mortality rate of humans. Nowadays, a common viral disease in the tropical region of the world is dengue. Previously, it is known as dengue hemorrhagic fever. In the 1950s, the Philippines and Thailand countries were plagued, dengue epidemics and recognized for the first time. Currently, Latin, American and Asian countries are the most affected ones causing hospitalization and death of people from almost all age groups. Here, the mosquitoes act as transmitters, and humans act as receiver, describing the life cycle of the dengue virus (DENV). *Aedes mosquito* is the transmitter of dengue after an infectious bite by anyone of the given dengue virus (DENV-1 to 4). Symptoms of dengue appear in 3–14 days after the infectious bite of mosquitoes. Mostly, adult and young children are affected by viral dengue. Dengue causes grievous fever-like illness due to mosquito-borne viral infection. Sometimes, dengue becomes a potentially lethal

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convolution. The incidence of dengue has increased 30-fold over the last 50 years. Distinct serotypes (DENV-1 to 4) are the disease origin, belonging to the genus *Flavivirus* family. If the human recovered from dengue virus, they are never affected again by the virus and receive immunity for a lifetime against that specific virus.

Only partial and momentary protection produced such immunity against the infection due to distinct serotypes of the virus. In comparison to transmitter, mosquitoes have the only selective method to eradicate the dengue virus. According to WHO (World Health Organisation), the estimation of dengue infections worldwide touches the range of 50–100 million, subjecting nearly half of the population all over the world at grave risk. There is no specific vaccine available for dengue. One recent observation in 2012 predicted that 3.9 billion people of around 128 countries are at potential risk of infection with DENV. Another contemplation in 2013 indicates that out of 390 million, 96 million (67–136 million) people infected through dengue per year and manifest clinically (with any severity of disease) (WHO 2020).

To research the mechanisms of dengue transmission, several mathematical models have been proposed and studied from Ghosh, Tiwari, and Chittopadhyay (2019) to Agosto and Khan (2018). In Ghosh, Tiwari, and Chittopadhyay (2019), the authors analyzed a compartmental model and studied dynamic behaviour of the model. Also, discuss ACF (active case finding) for asymptomatic and symptomatic individuals plays a crucial role in reducing disease prevalence. In Zhu and Xu (2019) studied an SIS-SI dengue model associated with a periodic environment to discuss temporal periodicity and spatial heterogeneity. Driessch and Watmough (2002) utilized a system of ODE to formulate a compartmental model for disease transmission and analyzed the stability and instability at $R_0 < 1$ and $R_0 > 1$ respectively. In Busenberg and Driessch (1990) and Esteva and Vargas (1999), the authors analyzed a dengue transmission model with varying size and applied competitive system theory to discuss the equilibrium's global asymptotic stability. Esteva and Vargas (1998, 2009) analyzed the dynamics of dengue disease with the help of interrupted feeding in the role of vertical and mechanical transmission. Also, discussed a transmission model of dengue with varying vector populations and the constant human population. They also established the global stability of endemic equilibrium with the threshold condition's help to measure the population of vector control parameters. In Linda and Amy (2000), the authors compared the deterministic and stochastic SIS and SIR model in discrete time. Misra and Sharma (2013), evaluate the effect of media awareness by advertising programs on vector-borne diseases. A nonlinear mathematical model is suggested and evaluated in this article. Tewa, Dimi, and Bowong (2009) studied a dengue fever model when only one serotype existed for the dynamics and showed the global asymptotic stable condition of disease-free and endemic equilibrium. In Kar and Jana (2009), the authors proposed a vector-borne disease model with three types of controls for eradicating disease and formulated the optimal control problem. They solved the model by considering control parameters as time-dependent. Syafruddin and Noorani (2013) constructed a model and discussed the system's local and global stability behaviour. Nur, Rachman, and Abdal (2018) analyzed the ODE's SIR model system with climate factors for dengue fever transmission characteristics in closed population and explained the equilibrium's stability using Lyapunov. In Panja, Mondal, and Chattopadhyay (2018), authors identify time-dependent cost-effective control strategies to minimize dengue spread, used the optimal control theory. In this article, the authors proposed a dengue transmission model using a control variable, namely insecticide and vaccination and develop the result based on control strategy (Agosto and Khan 2018). In Bonyah et al. (2017), authors analyze an SEIR Zika epidemic model and investigate the model with basic mathematical results. Treatment is not possible if the number of infectives exceeds a certain threshold, as it is in developing countries such as India, China, Swaziland, and Brazil. In this present paper, susceptible human have categorized by considering two different classes. We have incorporated a modified model, as discussed in Syafruddin and Noorani (2013) and compared it to the single-compartment model of susceptible beings with multiple compartments. Lyapunov function has applied to this model to examine the local and global stability analysis and the basic reproduction number. The application of sensitivity analysis has been analyzing to see the manageable parameters of the model and simulated the variables.

The structure of paper is as follows: the formation and formulation of the model described in Section "Mathematical formulation". The contained covered in Sections "Positivity and boundedness of the system", and "Qualitative analysis of model" are analysis of positivity and boundedness of the system and basic

reproduction number of the model along with estimation of the disease-free, endemic equilibrium point. Stability analysis of the model and performance of sensitivity analysis are in Sections “Stability analysis”, and “Sensitivity analysis”. The simulation results, as seen in Section “Numerical simulation”. Finally, a detailed discussion and conclusion is presented in Section “Conclusions”.

Mathematical formulation

At any time $t > 0$, the total population N_H have been sub-divided into five compartments which are: susceptible human at high risk S_{H_1} (high risk means susceptible are supposed to contact with the disease at a higher rate. i.e. some people are not aware of the precautions, and most areas are much polluted where the virus multiplies rapidly), susceptible human at low risk S_{H_2} (low risk means susceptible are supposed to contact with the disease at a lower rate), human exposed to dengue virus E_H , human with dengue virus I_H and recovered human R_H . Also the total vector population N_v have been sub-divided into three parts: susceptible vector S_v , exposed vector E_v and infected vector I_v also defined in Table 1. In the described part, susceptible vector are supposed to settle at a constant rate of π_v . When these vectors are in contact with an infected human, they acquire dengue and move to an exposed class with rate $\beta\alpha_H S_v I_H / N_H$. We consider standard incidence relation for the interaction between susceptible vector and infected humans. The moving rate γ_v are the vector from exposed class to infected class. At least a vector in every class lose their life from natural reasons with rate μ_v . In the case of vector population, the recovered class is not recognized, if the mosquito is infected from dengue virus, it remains infected throughout their entire life. The recruitment rate of individuals in the susceptible class are joined at a fixed rate of π_H . Newly recruited population rate r , join the high-risk susceptible class S_{H_1} and rest join the low-risk susceptible class S_{H_2} . On effective contact with infected vectors, persons in classes S_{H_1} and S_{H_2} move to exposed class. The interaction between susceptible human and infected vectors are a type of consultative phenomenon. The persons in class S_{H_2} are assumed to contact with disease at a lower rate in comparison to persons in class S_{H_1} . The susceptible class at high risk move to exposed class with rate $(\beta\alpha_v S_{H_1} I_v) / N_H$ and low risk move to the exposed class with rate $(\beta\theta\alpha_v S_{H_2} I_v) / N_H$. The relative possibility of low-risk susceptible with respect to high-risk susceptible is denoted by θ . Human of exposed class move to infected class at rate γ_H . Natural death rate of human is μ_H in each class. The schematic diagram for the transmission dynamics of dengue virus is depicted in Figure 1.

On the basis of transmission dynamics mathematical formulation for above model is:

$$\frac{dS_v}{dt} = \pi_v - \beta\alpha_H S_v \frac{I_H}{N_H} - \mu_v S_v \quad (1)$$

$$\frac{dE_v}{dt} = \beta\alpha_H S_v \frac{I_H}{N_H} - (\gamma_v + \mu_v) E_v \quad (2)$$

Table 1: Variables used in the framework are listed.

Parameters	Significance
N_H	Total human population
S_{H_1}	Susceptible human at high risk
S_{H_2}	Susceptible human at low risk
E_H	Exposed human
I_H	Infected human
R_H	Recovered human
N_v	Total vector population
S_v	Susceptible vector (mosquito)
E_v	Exposed vector
I_v	Infected vector

Figure 1: Compartmental model.

$$\frac{dI_v}{dt} = \gamma_v E_v - \mu_v I_v \quad (3)$$

$$\frac{dS_{H_1}}{dt} = r \pi_H - \beta \alpha_v I_v \frac{S_{H_1}}{N_H} - \mu_H S_{H_1} \quad (4)$$

$$\frac{dS_{H_2}}{dt} = (1-r) \pi_H - \beta \theta \alpha_v S_{H_2} \frac{I_v}{N_H} - \mu_H S_{H_2} \quad (5)$$

$$\frac{dE_H}{dt} = \beta \alpha_v (S_{H_1} + \theta S_{H_2}) \frac{I_v}{N_H} - (\gamma_H + \mu_H) E_H \quad (6)$$

$$\frac{dI_H}{dt} = \gamma_H E_H - (\mu_H + q) I_H \quad (7)$$

$$\frac{dR_H}{dt} = q I_H - \mu_H R_H \quad (8)$$

The meaning of parameters used in (1)–(8) are listed in Tables 1 and 2.

The above system can also be written as:

$$\frac{dS_v}{dt} = \pi_v - d_{11} I_H S_v - \mu_v S_v \quad (9)$$

$$\frac{dE_v}{dt} = d_{11} I_H S_v - d_{12} E_v \quad (10)$$

$$\frac{dI_v}{dt} = \gamma_v E_v - \mu_v I_v \quad (11)$$

$$\frac{dS_{H_1}}{dt} = B - d_{13} I_v S_{H_1} - \mu_H S_{H_1} \quad (12)$$

Table 2: Biological significance of parameters.

Parameters	Significance
π_v	Mobilization rate of susceptible vector population
β	Biting rate of vector per individual
μ_H, μ_v	Natural death rate of humans, vector respectively
α_H	Mobilization rate from infected human to susceptible vector
γ_v	Extrinsic death rate of vector
γ_H	Intrinsic death rate of humans
π_H	Mobilization rate of susceptible human population
q	Recovery rate of disease
r	Section of first time admitted person joining the susceptible class at high risk
α_v	Transmission rate from infected mosquitoes to susceptible human
θ	Relative possibility of low risk susceptible with reference to high risk susceptible

$$\frac{dS_{H_2}}{dt} = A - d_{13}\theta I_v S_{H_2} - \mu_H S_{H_2} \quad (13)$$

$$\frac{dE_H}{dt} = d_{13}I_v(S_{H_1} + \theta S_{H_2}) - d_{14}E_H \quad (14)$$

$$\frac{dI_H}{dt} = \gamma_H E_H - d_{15}I_H \quad (15)$$

$$\frac{dR_H}{dt} = qI_H - \mu_H R_H \quad (16)$$

with initial conditions, $S_v(0) > 0$, $E_v(0) \geq 0$, $I_v(0) \geq 0$, $S_{H_1}(0) > 0$, $S_{H_2}(0) > 0$, $E_H(0) \geq 0$, $I_H(0) \geq 0$, $R_H(0) \geq 0$, where, $d_{11} = \beta \frac{\alpha_H}{N_H}$, $d_{12} = \gamma_v + \mu_v$, $d_{13} = \beta \frac{\alpha_v}{N_H}$, $d_{14} = \gamma_H + \mu_H$, $d_{15} = \mu_H + q$, $A = (1-r)\pi_H$, $B = r\pi_H$.

Positivity and boundedness of the system

System (1)–(8) has positively invariant and bounded solution in the closed set.

Lemma 1. *The region of existence for all solutions initiating in the positive region is recommended by set ω*

$$\omega = \{(S_{H_1}, S_{H_2}, N_v, N_H) : 0 \leq N_v \leq Z_1, 0 \leq S_{H_1} \leq Z_2, 0 \leq S_{H_2} \leq Z_3, 0 \leq N_H \leq Z_4\}$$

where, $Z_1 = \max\left\{\frac{\pi_v}{\mu_v}, N_v(0)\right\}$, $Z_2 = \max\left\{\frac{r\pi_H}{\mu_H}, S_{H_1}(0)\right\}$, $Z_3 = \max\left\{\frac{(1-r)\pi_H}{\mu_H}, S_{H_2}(0)\right\}$, $Z_4 = \max\left\{\frac{\pi_H}{\mu_H}, N_H(0)\right\}$, which is invariant and compact with respect to the system (1)–(8).

Proof. System (1)–(8) can be written as:

$$\frac{dY}{dt} = CY + D \quad (17)$$

$$Y = [S_v, E_v, I_v, S_{H_1}, S_{H_2}, E_H, I_H, R_H]^T$$

$$C = \begin{pmatrix} k_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ k_2 & k_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_v & -\mu_v & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & k_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & k_5 & 0 & 0 & 0 \\ 0 & 0 & k_6 & 0 & 0 & k_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_v & -d_{15} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & q & -\mu_H \end{pmatrix}$$

where,

$$k_1 = -\left[\mu_v + \beta\alpha_H\left(\frac{I_H}{N_H}\right)\right], \quad k_2 = \beta\alpha_H\left(\frac{I_H}{N_H}\right), \quad k_3 = -(\gamma_v + \mu_v)$$

$$k_4 = -\left[\mu_H + \beta\alpha_v\left(\frac{I_v}{N_H}\right)\right], \quad k_5 = -\left[\mu_H + \beta\alpha_v\theta\left(\frac{I_H}{N_H}\right)\right], \quad k_6 = \beta\alpha_v\left(\frac{S_{H_1} + \theta S_{H_2}}{N_H}\right)$$

$$k_7 = -(\gamma_H + \mu_H)$$

The vector $D = [\pi_v, 0, 0, r\pi_H, (1-r)\pi_H, 0, 0, 0]^T$ has positive nature and all of $C(Y)$'s off-diagonal entries are non-negative. As a result, for all $Y \in R_+^8$, the matrix $C(Y)$ is Metzler (Abate, Tiwari, and Sastry 2009). Hence, all trajectories of system (1)–(8) which generate from an initial state in $Y \in R_+^8$ are bounded forever. On adding the first three equations of system (1)–(8), we get

$$\frac{dN_v}{dt} = \pi_v - \mu_v N_v$$

Using a standard comparison theorem used in Lakshmikantham, Leela, and Martynyuk (1989) we have, $0 \leq N_v(t) \leq \frac{\pi_v}{\mu_v} + \left(N_v(0) - \frac{\pi_v}{\mu_v}\right)e^{-\frac{\pi_v}{\mu_v}t}$. Thus, as $t \rightarrow \infty$, $0 \leq N_v(t) \leq \frac{\pi_v}{\mu_v}$, for any $t > 0$, $0 \leq N_v(t) \leq Z_1$, where $Z_1 = \max\left\{\frac{\pi_v}{\mu_v}, N_v(0)\right\}$.

Assume that $Z_2 = \max\left\{\frac{r\pi_H}{\mu_H}, S_{H_1}(0)\right\}$, then $0 \leq S_{H_1} \leq Z_2$. Similarly, let $Z_3 = \max\left\{\frac{(1-r)\pi_H}{\mu_H}, S_{H_2}(0)\right\}$, then $0 \leq S_{H_2} \leq Z_3$. By adding last five equations of system (1)–(8), we get

$$\frac{dN_H}{dt} = \pi_H - \mu_H N_H$$

Assume that $Z_4 = \max\left\{\frac{\pi_H}{\mu_H}, N_H(0)\right\}$, then $0 \leq N_H \leq Z_4$.

Therefore, all the feasible solution of system (1)–(8) are entered in the region ω , which means that it is the attracting set for region ω .

Qualitative analysis of model

Basic reproduction number

In this section, the basic reproduction number (R_0) is determined by using the next-generation matrix method as in Driessch and Watmough (2002), and Ullah et al. (2020). It follows that the system will be locally asymptotically stable if $R_0 < 1$, system will be unstable if $R_0 > 1$. The process to find the basic reproduction number through Jacobian matrices F and V are:

$$F = \begin{pmatrix} \frac{\partial F_1}{\partial E_v} & \frac{\partial F_1}{\partial I_v} & \frac{\partial F_1}{\partial E_H} & \frac{\partial F_1}{\partial I_H} \\ \frac{\partial F_2}{\partial E_v} & \frac{\partial F_2}{\partial I_v} & \frac{\partial F_2}{\partial E_H} & \frac{\partial F_2}{\partial I_H} \\ \frac{\partial F_3}{\partial E_v} & \frac{\partial F_3}{\partial I_v} & \frac{\partial F_3}{\partial E_H} & \frac{\partial F_3}{\partial I_H} \\ \frac{\partial F_4}{\partial E_v} & \frac{\partial F_4}{\partial I_v} & \frac{\partial F_4}{\partial E_H} & \frac{\partial F_4}{\partial I_H} \end{pmatrix} \text{ and } V = \begin{pmatrix} \frac{\partial V_1}{\partial E_v} & \frac{\partial V_1}{\partial I_v} & \frac{\partial V_1}{\partial E_H} & \frac{\partial V_1}{\partial I_H} \\ \frac{\partial V_2}{\partial E_v} & \frac{\partial V_2}{\partial I_v} & \frac{\partial V_2}{\partial E_H} & \frac{\partial V_2}{\partial I_H} \\ \frac{\partial V_3}{\partial E_v} & \frac{\partial V_3}{\partial I_v} & \frac{\partial V_3}{\partial E_H} & \frac{\partial V_3}{\partial I_H} \\ \frac{\partial V_4}{\partial E_v} & \frac{\partial V_4}{\partial I_v} & \frac{\partial V_4}{\partial E_H} & \frac{\partial V_4}{\partial I_H} \end{pmatrix}$$

where,

$$F_1 = d_{11}I_H S_v, \quad F_2 = \gamma_v E_v, \quad F_3 = d_{13}I_v(S_{H_1} + \theta S_{H_2}), \quad F_4 = \gamma_H E_H.$$

and

$$V_1 = d_{12}E_v, \quad V_2 = \mu_v I_v, \quad V_3 = d_{14}E_H, \quad V_4 = d_{15}I_H.$$

then,

$$F = \begin{pmatrix} 0 & 0 & 0 & d_{11}S_v \\ \gamma_v & 0 & 0 & 0 \\ 0 & d_{13}(S_{H_1} + \theta S_{H_2}) & 0 & 0 \\ 0 & 0 & \gamma_H & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} d_{12} & 0 & 0 & 0 \\ 0 & \mu_v & 0 & 0 \\ 0 & 0 & d_{14} & 0 \\ 0 & 0 & 0 & d_{15} \end{pmatrix}$$

then $(F.V^{-1})$ is calculated as:

$$F.V^{-1} = \begin{pmatrix} 0 & 0 & 0 & \frac{d_{11}\pi_v}{\mu_v d_{15}} \\ \frac{\gamma_v}{d_{12}} & 0 & 0 & 0 \\ 0 & \frac{d_{13}(B + \theta A)}{\mu_H \mu_v} & 0 & 0 \\ 0 & 0 & \frac{\gamma_H}{d_{14}} & 0 \end{pmatrix}$$

So, the basic reproduction number R_0 of system (9)–(16) is defined by maximum eigenvalue of the matrix $(F.V^{-1})$ and it is given by

$$R_0 = \sqrt{\frac{1}{\mu_v^2} \frac{d_{11}d_{13}\pi_v\gamma_v\gamma_H(B+A\theta)}{d_{12}d_{14}d_{15}\mu_H}}$$

Disease-free equilibrium

For system (9)–(16), the disease-free equilibrium point is $W_0 = (\bar{S}_v, \bar{E}_v, \bar{I}_v, \bar{S}_{H_1}, \bar{S}_{H_2}, \bar{E}_H, \bar{I}_H, \bar{R}_H)$. This equilibrium exists without any condition, where,

$$\bar{S}_v = \frac{\pi_v}{\mu_v}, \quad \bar{E}_v = 0, \quad \bar{I}_v = 0, \quad \bar{S}_{H_1} = \frac{B}{\mu_H}, \quad \bar{S}_{H_2} = \frac{A}{\mu_H}, \quad \bar{E}_H = 0, \quad \bar{I}_H = 0, \quad \bar{R}_H = 0.$$

Endemic equilibrium

For Eqs. (1)–(8) the endemic-equilibrium point is $W_1 = (S_v^*, E_v^*, I_v^*, S_{H_1}^*, S_{H_2}^*, E_H^*, I_H^*, R_H^*)$, whose constituents are positive solutions for the equilibrium equation (23) of the system (1)–(8).

A newer variable Z is defined as:

$$Z = \beta\alpha_v \frac{I_v^*}{N_H^*} \quad (18)$$

From the equilibrium equations (23) of the system (1)–(8), we have

$$S_v^* = \frac{\pi_v}{\mu_v + \beta\alpha_H \frac{I_H^*}{N_H^*}}, \quad E_v^* = \frac{\beta\alpha_H \pi_v \frac{I_H^*}{N_H^*}}{(\gamma_v + \mu_v) \left[\mu_v + \beta\alpha_H \frac{I_H^*}{N_H^*} \right]}, \quad (19)$$

$$I_v^* = \frac{\beta\alpha_H \gamma_v \pi_v \frac{I_H^*}{N_H^*}}{\mu_v(\gamma_v + \mu_v) \left[\mu_v + \beta\alpha_H \frac{I_H^*}{N_H^*} \right]}, \quad S_{H_1}^* = \frac{r\pi_H}{Z + \mu_H}, \quad S_{H_2}^* = \frac{(1-r)\pi_H}{\theta Z + \mu_H}, \quad (20)$$

$$E_H^* = \frac{Z}{\mu_H + \gamma_H} \left[\frac{r\pi_H}{Z + \mu_H} + \theta \frac{(1-r)\pi_H}{\theta Z + \mu_H} \right], \quad I_H^* = \frac{\gamma_H E_H^*}{\mu_H + Z}, \quad R_H^* = \frac{qI_H^*}{\mu_H} \quad (21)$$

the total human population is given by;

$$N_H^* = \left[\frac{E_H^* \gamma_H}{\mu_H} + \frac{\pi_H((Z + \mu_H)(\theta Z + \mu_H) + \gamma_H(Z - Zr + Zr\theta + \mu_H))}{(Z + \mu_H)(\theta Z + \mu_H)(\mu_H + \gamma_H)} \right] \quad (22)$$

Now using Eqs. (19)–(22) in Eq. (18), we get the following quadratic equation in Z :

$$B_3 Z^3 + B_2 Z^2 + B_1 Z + B_0 = 0 \quad (23)$$

where,

$$B_3 = \mu_H^2((1 + 4\theta + (\theta)^2)\pi_H(q + \mu_H)^2(\mu_H + \gamma_H)^2\mu_v + \beta^2\theta\pi_v\alpha_H\alpha_v\gamma_H\gamma_v\mu_H(\gamma_v + \mu_v)(2\beta(r(\theta - 1) - \theta)\alpha_H\gamma_H\mu_H + (-1 + r(\theta - 1) - 2\theta)(q + \mu_H)(\gamma_H + \mu_H)\mu_v))$$

$$B_2 = \mu_H^3(2(1 + \theta)\pi_H(q + \mu_H)^2(\gamma_H + \mu_H)^2\mu_v - \beta^2\pi_v\alpha_H\alpha_v\mu_H(\gamma_v + \mu_v)(\beta(r + \theta - r\theta)^2\alpha_H\gamma_H\mu_H - (-\theta(2 + \theta) + r(-1 + \theta^2))(q + \mu_H)(\gamma_H + \mu_H)\mu_v))$$

$$B_1 = \mu_H^4(q + \mu_H)^2(\gamma_H + \mu_H)^2\mu_v^2(\gamma_v + \mu_v)\pi_H(1 - R_0^2)$$

$$B_0 = 0.$$

On applying Descartes' rule of sign, we discussed the various chances for positive roots of Eq. (23) in Table 3. Here $B_0 = 0$, therefore it has no effect on evaluation of roots as in Driessche (2017) and Ghosh, Tiwari, and Chittopadhyay (2019).

Table 3: For $R_0 < 1$ and $R_0 > 1$ number of possible positive roots of Eq. (23).

S.N.	$B_3 B_2 B_1 B_0$	R_0	Change of sign	Possibility of positive root
1	+ + + 0	$R_0 < 1$	0	0
	+ + - 0	$R_0 > 1$	1	1
2	+ - + 0	$R_0 < 1$	2	0, 2
	+ - - 0	$R_0 > 1$	1	1
3	- + + 0	$R_0 < 1$	1	1
	- + - 0	$R_0 > 1$	2	1, 2
4	- - + 0	$R_0 < 1$	1	1
	- - - 0	$R_0 > 1$	0	0

Stability analysis

In this section, the stability analysis of disease-free and endemic equilibrium is carried out. The basic reproduction number (R_0) is used to discuss both the equilibrium points regarding local stability and global stability. Stability analysis can perform, using the sign of eigenvalues of the Jacobian matrix's characteristic equation as in Esteva and Vargas (1998) and Lakshmikantham, Leela, and Martynyuk (1989).

Local stability around equilibrium point

Now in this section, we are going to analyze the stability of two equilibrium points W_0 and W_1 . The outcome of stability analysis of these equilibria are shown in the following two theorems.

Theorem 1. For $R_0 < 1$ the disease-free equilibrium (W_0) is locally asymptotically stable and unstable if $R_0 > 1$.

Proof. The Jacobian matrix W_{01} for the model system (9)–(16) corresponding to equilibrium W_0 is given by

$$W_{01} = \begin{pmatrix} -\mu_v & 0 & 0 & 0 & 0 & 0 & \frac{-d_{11}\pi_v}{\mu_v} & 0 \\ 0 & -d_{12} & 0 & 0 & 0 & 0 & \frac{d_{11}\pi_v}{\mu_v} & 0 \\ 0 & \gamma_v & -\mu_v & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{-d_{13}B}{\mu_H} & -\mu_H & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\mu_H A \theta}{d_{13}(B + \theta A)} & 0 & -\mu_H & 0 & 0 & 0 \\ 0 & 0 & \frac{\mu_H}{d_{13}(B + \theta A)} & 0 & 0 & -d_{14} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_H & -d_{15} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & q & -\mu_H \end{pmatrix}$$

The four eigen values of W_{01} at disease-free equilibrium are $-\mu_v$, $-\mu_H$ (multiplicity 3) and the remaining four eigenvalues are given by the following biquadratic polynomial

$$\lambda^4 + v_1\lambda^3 + v_2\lambda^2 + v_3\lambda + v_4 = 0 \quad (24)$$

where,

$$\begin{aligned} v_1 &= d_{12} + d_{14} + d_{15} + \mu_v \\ v_2 &= d_{12}d_{14} + d_{12}d_{15} + d_{14}d_{15} + d_{12}\mu_v + d_{14}\mu_v + d_{15}\mu_v \\ v_3 &= d_{12}d_{14}d_{15} + d_{12}d_{14}\mu_v + d_{12}d_{15}\mu_v + d_{14}d_{15}\mu_v \\ v_4 &= d_{12}d_{14}d_{15}\mu_v(1 - R_0^2). \end{aligned}$$

Here, $v_1 > 0$, $v_1 v_2 - v_3 > 0$, $v_1 v_2 v_3 - v_3^2 - v_4 v_1^2 > 0$.

So, if $R_0 < 1$, all of the above conditions are fulfilled. Hence by Routh–Hurwitz criterion the disease-free equilibrium point W_0 is locally asymptotically stable otherwise, it is unstable.

Theorem 2. *The endemic equilibrium W_1 for the system (9)–(16) is locally asymptotically stable under some conditions stated in the proof of this theorem.*

Proof. The Jacobian matrix W_{10} for the model system (9)–(16) corresponding to equilibrium W_1 is given by

$$W_{10} = \begin{pmatrix} -A_1 & 0 & 0 & 0 & 0 & 0 & -d_{11}S_v^* & 0 \\ d_{11}I_H^* & -d_{12} & 0 & 0 & 0 & 0 & d_{11}S_v^* & 0 \\ 0 & \gamma_v & -\mu_v & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -A_2 & -A_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & -A_4 & 0 & -A_5 & 0 & 0 & 0 \\ 0 & 0 & A_6 & A_7 & A_8 & -d_{14} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_H & -d_{15} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & q & -\mu_H \end{pmatrix}$$

where, $A_1 = d_{11}I_H^* + \mu_v$, $A_2 = d_{13}S_{H_1}^*$, $A_3 = d_{13}I_v^* + \mu_H$, $A_4 = d_{13}\theta S_{H_2}^*$, $A_5 = d_{13}\theta I_v^* + \mu_H$, $A_6 = d_{13}(S_{H_1}^* + \theta S_{H_2}^*)$, $A_7 = d_{13}I_v^*$, $A_8 = d_{13}\theta I_v^*$

Four eigenvalues of the above matrix are $-A_1$, $-\mu_H$, $-A_3$, $-A_5$ and the other roots are given with the help of biquadratic polynomial

$$\lambda^4 + w_1\lambda^3 + w_2\lambda^2 + w_3\lambda + w_4 = 0 \quad (25)$$

where,

$$w_1 = d_{12} + d_{14} + d_{15} + \mu_v$$

$$w_2 = d_{15}\mu_v + d_{14}(d_{15} + \mu_v) + d_{12}(d_{14} + d_{15} + \mu_v) - A_6 d_{11}\gamma_H\gamma_v S_v^*$$

$$w_3 = d_{12}d_{14}d_{15} + d_{14}d_{15}\mu_v + d_{12}(d_{14} + d_{15})\mu_v + (-(A_3 + A_5)A_6 + A_2A_7 + A_4A_8)d_{11}\gamma_H\gamma_v S_v^*$$

$$w_4 = d_{12}d_{14}d_{15}\mu_v(1 - A_3A_5R_0^2) + (A_2A_5A_7 + A_3A_4A_8)d_{11}\gamma_H\gamma_v S_v^*.$$

Here, $w_1 > 0$, $w_1 w_2 - w_3 > 0$, $w_1 w_2 w_3 - w_3^2 - w_4 w_1^2 > 0$.

For this, if $w_4 > 0$ and $R_0 > \frac{1}{A_3 A_5}$, the roots of Eq. (25) will have negative real parts. Therefore, Routh–Hurwitz criterion satisfied and the system is locally asymptotically stable for W_1 .

Global stability around equilibrium point

Here, we are going to analyze the stability of two equilibrium points W_0 and W_1 . The outcomes of stability analysis of these equilibrium points are shown in the following two theorems.

Theorem 1. *If $R_0 \leq 1$, the disease-free equilibrium (W_0) is globally asymptotically stable on ω with assumption*

$$d_{13}(\bar{S}_{H_1} + \theta \bar{S}_{H_2}) = \mu_v \quad (26)$$

$$d_{11}\bar{S}_v = q + \mu_H. \quad (27)$$

Proof. Consider the Lyapunov function below:

$$G(t) = (S_v - \bar{S}_v \ln S_v) + E_v + I_v + (S_{H_1} - \bar{S}_{H_1} \ln S_{H_1}) + (S_{H_2} - \bar{S}_{H_2} \ln S_{H_2}) + E_H + I_H + R_H$$

Differentiating with respect to time t , get:

$$\begin{aligned} G'(t) &= S'_v \left(1 - \frac{\bar{S}_v}{S_v}\right) + E'_v + I'_v + S'_{H_1} \left(1 - \frac{\bar{S}_{H_1}}{S_{H_1}}\right) + S'_{H_2} \left(1 - \frac{\bar{S}_{H_2}}{S_{H_2}}\right) + E'_H + I'_H + R'_H \\ G'(t) &= (\pi_v - d_{11}I_H S_v - \mu_v S_v) \left(1 - \frac{\bar{S}_v}{S_v}\right) + d_{11}I_H S_v - d_{12}E_v + \gamma_v E_v - \mu_v I_v \\ &\quad + (B - d_{13}I_v S_{H_1} - \mu_H S_{H_1}) \left(1 - \frac{\bar{S}_{H_1}}{S_{H_1}}\right) + (A - d_{13}\theta I_v S_{H_2} - \mu_H S_{H_2}) \left(1 - \frac{\bar{S}_{H_2}}{S_{H_2}}\right) \\ &\quad + d_{13}I_v (S_{H_1} + \theta S_{H_2}) - d_{14}E_H + \gamma_H E_H - d_{15}I_H + qI_H - \mu_H R_H \end{aligned}$$

On solving further get:

$$\begin{aligned} &= \pi_v \left(1 - \frac{\bar{S}_v}{S_v}\right) + \mu_v \bar{S}_v \left(1 - \frac{S_v}{\bar{S}_v}\right) + I_v (d_{13}(\bar{S}_{H_1} + \theta \bar{S}_{H_2}) - \mu_v) - \mu_v E_v + B \left(1 - \frac{\bar{S}_{H_1}}{S_{H_1}}\right) \\ &\quad + A \left(1 - \frac{\bar{S}_{H_2}}{S_{H_2}}\right) + \mu_H \bar{S}_{H_1} \left(1 - \frac{S_{H_1}}{\bar{S}_{H_1}}\right) + \mu_H \bar{S}_{H_2} \left(1 - \frac{S_{H_2}}{\bar{S}_{H_2}}\right) + I_H (d_{11} \bar{S}_v - (q + \mu_H)) - \mu_H E_H \\ &\quad - \mu_H R_H \end{aligned}$$

Using the conditions (26) and (27), also we have $\bar{S}_v = \frac{\pi_v}{\mu_v}$, $\bar{S}_{H_1} = \frac{B}{\mu_H}$ and $S_{H_2} = \frac{A}{\mu_H}$, the above equation can be rewritten as:

$$\begin{aligned} G'(t) &= \pi_v \left(2 - \frac{\bar{S}_v}{S_v} - \frac{S_v}{\bar{S}_v}\right) + B \left(2 - \frac{\bar{S}_{H_1}}{S_{H_1}} - \frac{S_{H_1}}{\bar{S}_{H_1}}\right) + A \left(2 - \frac{\bar{S}_{H_2}}{S_{H_2}} - \frac{S_{H_2}}{\bar{S}_{H_2}}\right) - \mu_H E_H - \mu_v E_v \\ &\quad - \mu_H R_H \\ &= -\pi_v \frac{(S_v - \bar{S}_v)^2}{S_v \bar{S}_v} - B \frac{(S_{H_1} - \bar{S}_{H_1})^2}{S_{H_1} \bar{S}_{H_1}} - A \frac{(S_{H_2} - \bar{S}_{H_2})^2}{S_{H_2} \bar{S}_{H_2}} - \mu_H E_H - \mu_v E_v - \mu_H R_H \end{aligned}$$

Hence the conditions (26) and (27) ensures that $G'(t) \leq 0$. By using Lasalle's extension to Lyapunov's method (Bonyah et al. 2017; Ullah, Khan, and Farooq 2018a), the limit set of each solution is contained in the largest invariant set for which $S_{H_1} = \bar{S}_{H_1}$, $S_{H_2} = \bar{S}_{H_2}$, $R_H = 0$ and $S_v = \bar{S}_v$ is the singleton set $\{W_0\}$. This means, the disease-free equilibrium (W_0) is globally asymptotically stable on ω . This concludes the proof.

Theorem 2. *The endemic equilibrium (W_1) is globally asymptotically stable in ω provided the following inequalities hold*

$$\text{Max} \left\{ \left(\frac{d_{13}\theta\pi_H}{Z + \mu_H} \right)^2, \left(\frac{d_{13}(1-r)\pi_H\theta}{\theta Z + \mu_H} \right)^2 \right\} < \mu_H \mu_v \quad (28)$$

$$\text{Max} \left\{ d_{12}Z_1^2, d_{13}Z_1\theta, \frac{4}{\mu_v} \left(d_{13} \left(\frac{r\pi_H}{Z + \mu_H} + \frac{\theta(1-r)\pi_H}{\theta Z + \mu_H} \right) \right)^2 \right\} < d_{14} \quad (29)$$

$$\text{Max} \left\{ \frac{4(\gamma_H)^2}{d_{14}}, \frac{q^2}{\mu_H}, d_{11}Z_1^2 \right\} < d_{15} \quad (30)$$

$$\text{Max} \left\{ \frac{3}{4} \frac{(d_{11}Z_4)^2}{\mu_v}, \frac{3(d_{11}Z_1)^2}{d_{15}}, \frac{3\gamma_v^2}{\mu_v} \right\} < d_{12} \quad (31)$$

Proof. Consider the following positive definite function, to study the global stability behaviour of the endemic equilibrium (W_1):

$$V(t) = \frac{1}{2}(S_{H_1} - S_{H_1}^*)^2 + \frac{1}{2}(S_{H_2} - S_{H_2}^*)^2 + \frac{1}{2}(E_H - E_H^*)^2 + \frac{1}{2}(I_H - I_H^*)^2 \\ + \frac{1}{2}(R_H - R_H^*)^2 + \frac{1}{2}(S_v - S_v^*)^2 + \frac{1}{2}(E_v - E_v^*)^2 + \frac{1}{2}(I_v - I_v^*)^2$$

Differentiating with respect to time t ,

$$V'(t) = S'_{H_1}(S_{H_1} - S_{H_1}^*) + S'_{H_2}(S_{H_2} - S_{H_2}^*) + E'_H(E_H - E_H^*) + I'_H(I_H - I_H^*) \\ + R'_H(R_H - R_H^*) + S'_v(S_v - S_v^*) + E'_v(E_v - E_v^*) + I'_v(I_v - I_v^*)$$

With the help of Eqs. (9)–(16) on rearranging the terms we get:

$$V'(t) = -\mu_H(S_{H_1}^* - S_{H_1})^2 + \frac{d_{13}r\pi_H}{Z + \mu_H}(I_v - I_v^*)(S_{H_1}^* - S_{H_1}) - \frac{\mu_v}{4}(I_v - I_v^*)^2 \\ - d_{13}Z_1(S_{H_1} - S_{H_1}^*)^2 + d_{13}Z_1(S_{H_1} - S_{H_1}^*)(E_H - E_H^*) - \frac{d_{14}}{4}(E_H - E_H^*)^2 \\ - \mu_H(S_{H_2}^* - S_{H_2}) + \frac{d_{13}(1-r)\pi_H\theta}{\theta Z + \mu_H}(I_v - I_v^*)(S_{H_2}^* - S_{H_2}) - \frac{\mu_v}{4}(I_v - I_v^*)^2 \\ - d_{13}\theta Z_1(S_{H_2} - S_{H_2}^*)^2 + d_{13}\theta Z_1(S_{H_2} - S_{H_2}^*)(E_H - E_H^*) - \frac{d_{14}}{4}(E_H - E_H^*)^2 \\ - \frac{d_{14}}{4}(E_H - E_H^*)^2 + d_{13}\left(\frac{r\pi_H}{Z + \mu_H} + \frac{\theta(1-r)\pi_H}{\theta Z + \mu_H}\right)(I_v - I_v^*)(E_H - E_H^*) \\ - \frac{\mu_v}{4}(I_v - I_v^*)^2 - \frac{d_{15}}{4}(I_H - I_H^*)^2 + \gamma_H(E_H - E_H^*)(I_H - I_H^*) - \frac{d_{14}}{4}(E_H - E_H^*)^2 \\ - \mu_H(R_H - R_H^*)^2 + q(R_H - R_H^*)(I_H - I_H^*) - \frac{d_{15}}{4}(I_H - I_H^*)^2 \\ - d_{11}\left(\frac{\gamma_H}{\mu_H + q}\left(\frac{Z}{\mu_H + \gamma_H}\left(\frac{r\pi_H}{Z + \mu_H} + \frac{\theta(1-r)\pi_H}{\theta Z + \mu_H}\right)\right)\right)(S_v - S_v^*)^2 \\ + d_{11}Z_1(I_H - I_H^*)(S_v - S_v^*) - \frac{d_{15}}{4}(I_H - I_H^*)^2 - \mu_v(S_v - S_v^*)^2 \\ + d_{11}Z_4(S_v - S_v^*)(E_v - E_v^*) - \frac{d_{12}}{3}(E_v - E_v^*)^2 - \frac{d_{12}}{3}(E_v - E_v^*)^2 \\ + d_{11}Z_1(I_H - I_H^*)(E_v - E_v^*) - \frac{d_{15}}{4}(I_H - I_H^*)^2 - \frac{d_{12}}{3}(E_v - E_v^*)^2 \\ + \gamma_v(I_v - I_v^*)(E_v - E_v^*) - \frac{\mu_v}{4}(I_v - I_v^*)^2.$$

For system (9)–(16) inside the region of attraction ω , $V'(t)$ can be turned negative definite if conditions (28)–(31) hold as in Baniya and Keval (2020).

Sensitivity analysis

Here, we deal with the sensitivity analysis of the model to see the effect of manageable parameters of the model on the basic reproduction number (R_0) as in Zheng and Nie (2018) and Augusto and Khan (2018). The basic reproduction number behave like an important epidemiological parameter, and sensitivity analysis gives us outputs of the model, divided for quantitative values through variations in different parameter values. Here,

a specified space of parameter examines the response model from parameter variation to output variables. The sensitivity index for a variable γ , which depends differentially on a parameter σ , is defined as:

$$Z_{\gamma}^{\sigma} = \frac{\partial \gamma}{\partial \sigma} \times \frac{\sigma}{\gamma}$$

We measure elasticities (Z_{γ}^{σ}) to see the results of relative changes to the parameter R_0 . Elasticity is the proportional solution to the proportional perambulation.

$$\begin{aligned} Z_{R_0}^{\beta} &= \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} = \frac{(B + A\theta)\beta\pi_v\alpha_H\alpha_v\gamma_H\gamma_v}{\mu_v^2\mu_H N_H^2(\mu_H + q)(\mu_H + \gamma_H)(\mu_v + \gamma_v)\sqrt{G}} \times \frac{\beta}{R_0} > 0. \\ Z_{R_0}^{\alpha_H} &= \frac{\partial R_0}{\partial \alpha_H} \times \frac{\alpha_H}{R_0} = \frac{(B + A\theta)\beta^2\pi_v\alpha_v\gamma_H\gamma_v}{2\mu_v^2\mu_H N_H^2(\mu_H + q)(\mu_H + \gamma_H)(\mu_v + \gamma_v)\sqrt{G}} \times \frac{\alpha_H}{R_0} > 0. \\ Z_{R_0}^{\alpha_v} &= \frac{\partial R_0}{\partial \alpha_v} \times \frac{\alpha_v}{R_0} = \frac{(B + A\theta)\beta^2\pi_v\alpha_H\gamma_H\gamma_v}{2\mu_v^2\mu_H N_H^2(\mu_H + q)(\mu_H + \gamma_H)(\mu_v + \gamma_v)\sqrt{G}} \times \frac{\alpha_v}{R_0} > 0. \\ Z_{R_0}^{\gamma_v} &= \frac{\partial R_0}{\partial \gamma_v} \times \frac{\gamma_v}{R_0} = \frac{-\frac{R_0^2}{(\mu_v + \gamma_v)} + \frac{R_0^2}{\gamma_v}}{2\sqrt{G}} \times \frac{\gamma_v}{R_0} < 0. \\ Z_{R_0}^{\gamma_H} &= \frac{\partial R_0}{\partial \gamma_H} \times \frac{\gamma_H}{R_0} = \frac{-\frac{R_0^2}{(\mu_H + \gamma_H)} + \frac{R_0^2}{\gamma_H}}{2\sqrt{G}} \times \frac{\gamma_H}{R_0} < 0. \\ Z_{R_0}^{\mu_v} &= \frac{\partial R_0}{\partial \mu_v} \times \frac{\mu_v}{R_0} = \frac{-\frac{R_0^2}{(\mu_v + \gamma_v)} - \frac{2R_0^2}{\mu_v}}{2\sqrt{G}} \times \frac{\mu_v}{R_0} < 0. \\ Z_{R_0}^{\mu_H} &= \frac{\partial R_0}{\partial \mu_H} \times \frac{\mu_H}{R_0} = \frac{\left(\frac{R_0^2}{(\mu_H + \gamma_H)} + \frac{R_0^2}{(\mu_H + q)} + \frac{R_0^2}{\mu_H}\right)}{2\sqrt{G}} \times \frac{\mu_H}{R_0} < 0. \\ Z_{R_0}^{\pi_v} &= \frac{\partial R_0}{\partial \pi_v} \times \frac{\pi_v}{R_0} = \frac{(B + A\theta)\beta^2\alpha_H\alpha_v\gamma_H\gamma_v}{2\mu_v^2\mu_H N_H^2(\mu_H + q)(\mu_H + \gamma_H)(\mu_v + \gamma_v)\sqrt{G}} \times \frac{\pi_v}{R_0} > 0. \\ Z_{R_0}^q &= \frac{\partial R_0}{\partial q} \times \frac{q}{R_0} = \frac{(B + A\theta)\beta^2\pi_v\alpha_H\alpha_v\gamma_H\gamma_v}{2\mu_v^2\mu_H N_H^2(\mu_H + q)^2(\mu_H + \gamma_H)(\gamma_v + \mu_v)\sqrt{G}} \times \frac{q}{R_0} < 0. \end{aligned}$$

where, $G = \frac{(B+A\theta)\beta^2\pi_v\alpha_H\alpha_v\gamma_H\gamma_v}{\mu_v^2\mu_H N_H^2(\mu_H+q)(\mu_H+\gamma_H)(\mu_v+\gamma_v)}$

From the above expression, we see that $Z_{R_0}^{\alpha_H} = Z_{R_0}^{\alpha_v} = Z_{R_0}^{\pi_v}$ which implies small changes in any of these parameters α_H , α_v and π provides a significant influence. In Figures 2–10, we have demonstrated the effect of these parameters on basic reproduction number. From Figure 2, we can see that the parameters α_H and α_v have equal impact on R_0 which shows that controlling the route of transmission of this disease is very important if we plan to restrict the transmission potential. From Figures 3–5, it is clear that the parameter β which is biting rate has more influence on the basic reproduction number R_0 as compared to the transmission probabilities α_v , α_H and mosquito death rate μ_v . Although the human to human transmission of this disease is deficient, our proposed model suggests that even a minor increase in the parameter β corresponding to

Figure 2: Influence of α_H and α_v on R_0 .

Figure 3: Influence of α_H and β on R_0 . Figure 4: Influence of β and α_v on R_0 . Figure 5: Influence of β and μ_v on R_0 .

transmission of dengue virus can lead to a great increase in the R_0 . In Figure 6 demonstrates the fact that the impact of γ_H on R_0 is much higher than the impact of γ_v on R_0 . Another fact clearly shown in Figures 7–9, β is more affected by numerous biological (γ_v), socio-behavioural (π_v), and environmental factors (q) and that govern pathogen transmission. Global sensitivity is performed in Figure 10 to determine the model's valuable parameter. Also, use the combination of Latin hypercube sampling (LHS) in conjunction with partial rank correlation coefficient (PRCC) to see the impact of manageable parameters of the model on basic reproduction number (R_0) (Altaf Khana, Ullah, and Farooq 2018; Khan and Atangana 2020). The parameters used in the present analysis are given in Table 4. The most sensitive parameter is μ_v with a low PRCC value. From the above figures, we can conclude that the control policies should reduce the transmission rates, mobilization rate of vector, biting rate, and human death rate. We can conclude that controlling this disease's transmission route is very important if we plan to restrict its transmission. Therefore, both the cases are closely related to control dengue.

Figure 6: Influence of γ_H and γ_v on R_0 . Figure 7: Influence of β and q on R_0 . Figure 8: Influence of β and π_v on R_0 . Figure 9: Influence of β and γ_v on R_0 .

Numerical simulation

This section aims to find the numerical solution of ODEs Eqs. (1)–(8) with the help of a medium order method in a finite interval of time using Matlab (Derouich and Boutayeb 2006; Khan, Ullah, and Farooq 2018;

Figure 10: PRCC results for significance of parameters in R_0 .

Table 4: Sensitivity indices of R_0 to the parameter values of the model.

Variable	Parameter	Sensitivity index
R_0	β	0.817806
	α_v	0.408903
	γ_v	0.137216
	γ_H	0.189935
	μ_v	-0.955022
	μ_H	-0.412268
	π_v	0.408903
	q	-0.407538

Ullah et al. 2020; Ullah, Khan, and Farooq 2018b), confirming results acquired with analysis and multiple observations give a deeper understanding of the model. For this used parameter values are provided in Table 5. These parameters value satisfy the conditions for the existence of endemic equilibrium, W_1 are obtained as $S_v^* = 1.932 \times 10^7$, $E_v^* = 15,546.5$, $I_v^* = 2.05214 \times 10^6$, $S_{H_1}^* = 361,823.0$, $S_{H_2}^* = 2,007,410.0$, $E_H^* = 6228.1$, $I_H^* = 51,197.9$, $R_H^* = 1.52836 \times 10^7$.

The value of basic reproduction number (R_0) is got as 0.001835 which is less than one as our requirement. Stability conditions are also fully desired for these parameter values. The matrix's eigenvalues are obtained for W_{01} as -0.025 , -0.0154 (multiplicity 3), -4.6130 , -3.3147 , -0.08589 and -0.0140 the polynomial roots satisfied the given stability conditions. It is articulate that all the eigenvalues are negative. Similarly, for the matrix W_{10} , found the condition for basic reproduction number $R_0 > 3.364$ and the matrix eigenvalues are -0.0473 , -0.0154 , -0.1 , -0.8211 , -4.6380 , -3.2928 , -0.0665 and -0.0322 .

To show the nonlinear stability behaviour of disease prevalence in their respected population. For this, we have taken 110 units for susceptible vector and exposed vector, 800 units for infected vector. We plotted the solution trajectories at different initial starts using the above set of parameter values. These trajectories are shown in Figures 11, 12, and 13 respectively. In Figure 11, the variation of S_v w.r.t the time t for the different values of transmission probability $\alpha_v = 0.55, 0.65, 0.75, 0.85$ and 0.95 are shown properly. It suggests that the population of susceptible vectors increase with a decrease in transmission probability. Therefore, to reduce the susceptible vector population's size, we have to increase the transmission between infected mosquito to susceptible human. Also, in Figure 12, it is interesting to note that for different values of α_v , the exposed vector

Table 5: Epidemiological parameters and their values with unit.

Parameters	Values (Ghosh, Tiwari, and Chttopadhyay 2019)	Unit
π_v	350,000	day ⁻¹
β	2	day ⁻¹
α_H	0.75	day ⁻¹
μ_v	0.025(Assumed)	day ⁻¹
γ_v	0.0495(Assumed)	day ⁻¹
π_H	273,600	day ⁻¹
r	0.25	day ⁻¹
α_v	0.75	day ⁻¹
μ_H	0.0154	day ⁻¹
γ_H	3.3	day ⁻¹
θ	0.5	day ⁻¹
q	4.5972	day ⁻¹

Figure 11: The plot of the susceptible vector S_v with different value of α_v . **Figure 12:** The behavior of exposed vectors with α_v .

population become zero in 27 unit of time whenever, if we decrease the transmission rate (α_v), it increases up to 40 unit of time. It indicates that if we decrease the rate of mosquito, the exposed vector become increases. Further, the variation of infected vector w.r.t time t for different values of β is given in Figure 13. It is evident that as the value of β increases, the total infected population (I_v) also increases. So, we infer that by multiple biting of mosquitoes, abundant of infected vector rapidly.

Figure 13: The total number of infected individuals for different values of β .

Conclusions

Vector-borne disease outbreaks have ruined many countries. From which, focused on assessing the dynamics of dengue fever in this article. We proposed a mathematical model, modelling them and incorporating the effect of the variable human with exponential growth. We discussed the positivity and boundedness of the system by Metzler property and found the basic reproduction number (R_0) that control the transmission of disease and the growth rate of the infected human population. The sensitivity of R_0 obtained very sensitive for the model's parameters corresponding to transmission rate, mobilization rate of vector, biting rate, and human death rate. Numerical simulations with different parameter settings illustrate the succession of epidemics, their amplitudes and justify theoretical findings. So, in this paper, main objective is to understand the dynamics of the dengue virus with variable human and investigate the model stability criteria for the dynamics of the dengue virus. Based on stability results and the basic reproduction number, we can say that our model is very beneficial for us. Also, it discussed the sensitivity and numerical simulation of the system. Thus we can conclude that our model gives us the most reliable results on changing the nonlinear model's complexity by incorporating more epidemiological parameters, more saturating functions with respect to different virus types of the disease and mode of transmission.

According to the medical point of view, we believe that our model analysis is beneficial for humans also. It would assist in controlling the spreads of dengue disease.

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