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Research Article

Mathematical model on the transmission dynamics of Dengue virus and its global stability

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Abstract: We formulate a mathematical model of Dengue virus by considering two population sizes of human and mosquito. We define a basic reproductive number R_0 and equilibrium points for our model. We prove that the disease-free equilibrium is globally stable when $R_0 < 1$, which means the disease will die out and when $R_0 > 1$, the endemic equilibrium is globally stable. Various numerical simulations using MATLAB are carried out to establish the analytical results. The efficacy of personal protection from mosquito to human and from pregnant women to newborn child are also carried out by numerical simulations.

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INTRODUCTION

Dengue virus (DENV) is a mosquito-borne flavivirus. Dengue is the fastest growing mosquito-borne disease across the world today, causing nearly 400 million infections every year. It is estimated that about 3.9 billion people in 128 countries, approximately 50% of the world's population are at risk of infection with dengue viruses. Dengue is endemic in all states and union territories (UTs) of India. In 2015, a total of 99,913 dengue cases and 220 deaths were reported from 35 states and UTs. The largest dengue outbreaks in worldwide was characterized by the year 2016. The region of Americas reported more than 2.38 million cases in 2016, where Brazil alone contributed approximately 1.5 million cases, around 3 times higher than in 2014. 1032 dengue deaths were also reported in the region. The Western Pacific Region reported more than 375 000 suspected cases of dengue in 2016, of which the Philippines reported 176 411 and Malaysia 100 028 cases, representing a similar burden to the previous year for both countries. The Solomon Islands declared an outbreak with more than 7000 suspected. In the African Region, Burkina Faso reported a localized outbreak of dengue with 1061 probable cases [1]. The *Aedes aegypti* mosquito is the primary vector of dengue. Dengue virus is transmitted to humans through the infected female mosquito bites. After a person is infected, he/she became the main carrier and amplifying host of the virus, serving as a source of the virus for uninfected mosquitoes for 4-5 days; maximum 12 days. Thus, mosquitoes acquire the virus mainly from biting an infected person. Once infective, a mosquito is capable of transmitting the virus to humans for the rest of its life through biting [1].

Dengue infection and illness are caused by four different DENV (DENV-1, DENV-2, DENV-3 and DENV-4) serotypes that cross-react immunologically. Infection with a particular serotype is believed to result in life-long immunity to that serotype and cross-protection to the other serotypes for up to two years [1]. People who have had a single primary infection have been observed to have a higher risk of severe dengue including dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS) upon a second infection, a phenomenon often attributed to antibody enhancement. Infants with waning maternal dengue antibodies have been observed to be at higher risk of DHF and DSS com-pared to infants with no maternal dengue antibodies [2]. There is no specific effective antiviral treatment for dengue illness other than supportive care, especially for severe cases. Good case management of severe dengue cases can greatly reduce the death rate. The only current means for dengue control is various forms of vector control. However, vector control has been only partially successful in reducing dengue disease burden [3].

In past two and half decades, several epidemic models have been formulated for predicting the transmission of epidemic diseases. Derouich and Boutayeb [4] proposed a mathematical model to simulate the succession of two epidemics with variable human populations. Fred Brauer [5]

given a brief outline of some of the important aspects of the development of mathematical epidemiology. Sanches and Massad [6] proposed a comparative analysis of three different methods for the estimation of the basic reproduction number of dengue. Pollett *et al.* [7] formulated a understanding dengue virus evolution to support epidemic surveillance and counter-measure development. Felipo Bacania at el. [8] developed a mathematical modelling for the transmission of dengue: Symmetry and travelling wave analysis. Pliego *et al.* [9] formulated a model in seasonality on the life cycle of Aedes aegypti mosquito and its statistical relation with dengue outbreaks. Hendron and Bonsall [10] developed a mathematical model on the interplay of vaccination and vector control on small dengue networks. Sardar *et al.* [11] formulated a mathematical model of dengue transmission with memory. Yamashita *et al.* [12] proposed travelling wave solutions for the dispersive models describing population dynamics of *Aedes aegypti.* Rodrigues *et al.* [13] developed a mathematical model on dynamics of Dengue epidemics when using optimal control. Zhu *et al.* [14] formulated a coexistence of across diffusive dengue fever model in a heterogeneous environment.

In this paper, we have formulated a mathematical model of Dengue virus transmission for both human and mosquito population. We have considered the Susceptible class, infected class and Recovered class in human population with vertical transmission. In mosquito population, we have considered two classes (Susceptible and Infected) for Dengue virus transmission. We find basic reproduction number $\boldsymbol{R_0}$ gives global dynamical behavior of the model. If $\boldsymbol{R_0 < 1}$, the disease-free equilibrium is globally stable, which means the disease will die out and if $\boldsymbol{R_0 > 1}$, the endemic equilibrium is globally stable. The basic reproduction number $\boldsymbol{R_0}$ is also used in numerical simulations to discuss the effectiveness of control strategies. The paper is organized as follows: Introduction is given in Section 1; the basic assumptions and parameters of the model and the epidemic model is developed in Section 2; Section 3 establishes the stability of the system developed; numerical simulations is given in Section 4; and finally, conclusion in Section 5.

MODEL DESCRIPTION AND FORMULATION

We divide the human population into three classes $S_H I_H R_H$ (Susceptible-Infected-Recovered) and the mosquito population into two classes $S_M I_M$ (Susceptible-Infected). Schematic flow of this model is shown in figure 1 and the state variables and associated parameters of this model are given in Table 1.

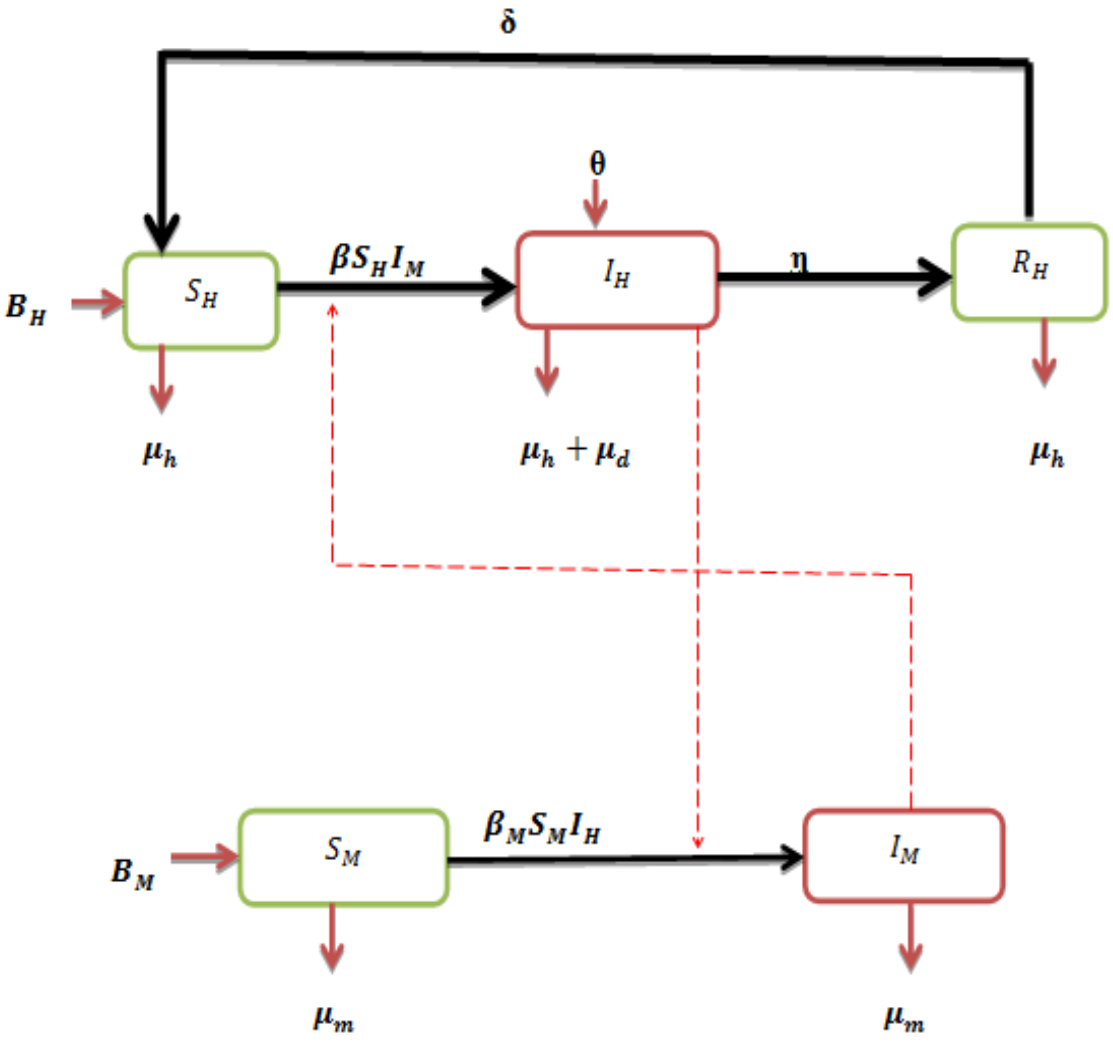

Fig. 1: Schematic flow of dengue transmission dynamics.

Table 1 : The state variables and associated parameters

$S_H(t)$: Susceptible proportions of human in time t
$I_H(t)$: Infectious proportions of human in time t
$R_H(t)$: Recovered proportions of human in time t
$S_M(t)$: Susceptible proportions of mosquito in time t
$I_M(t)$: Infectious proportions of mosquito in time t
$\bar{S}_H(t)$: Susceptible humans in time t
$\bar{I}_H(t)$: Infectious humans in time t
$\bar{R}_H(t)$: Recovered humans in time t
$\bar{S}_M(t)$: Susceptible mosquito in time t
$\bar{I}_M(t)$: Infectious mosquito in time t
$N_H(t)$: Total human population in time t
$N_M(t)$: Total mosquito population in time t
B_H: Birth rate and immigration rate of humans
B_M: Birth rate and immigration rate of mosquito
β: Transmission probability of Dengue virus from human to human and from mosquito to human
β_M: Transmission probability of Dengue virus from human to mosquito
η: Rate of transmission from infected humans to recovered humans
δ: Rate of transmission from recovered humans to susceptible humans
μ_h: Natural death rate of humans
μ_z: Death rate of humans due to Dengue virus
μ_m: Natural death rate of mosquito
θ: Rate of vertical transmission in human population.

Basic assumptions of model

We assume that humans enter the susceptible class through a constant birth rate and immigration rate B_H. When an infected with Dengue virus mosquito bites a Susceptible human, he/she moves to the infected class with the transmission probability β, where β is the sum of the transmission probability of Dengue virus from human to human and from mosquito to human. After some time, the infectious humans recover and move to the Recovered class with a constant rate η. The recovered humans have some immunity to the disease and do not get clinically ill, but they can pass the infection to mosquitoes. After some period of time, they lose their immunity and return to the susceptible class with constant rate δ. Humans leave the population through a natural death rate μ_h, and through a per capita disease-induced death rate μ_z. In human population, the vertical transmission rate θ is assumed, that is the rate of transmission of Dengue virus from infected pregnant women to new born children.

In mosquito population, we assumed that the mosquito enters the susceptible class through a constant birth rate and immigration rate B_M. When a susceptible mosquito bites an infected human being with Dengue virus, then the mosquito gets infected with Dengue virus and it goes to infected class. The constant transmission probability of Dengue virus from human to mosquito β_M is considered. Mosquitoes leave the population through a constant natural death rate μ_m only is assumed. We do not assume the vertical transmission in mosquito population.

Based on our assumptions and the flow of transmission of Dengue virus in human population as depicted in figure 1, we have the following system of equations:

$$\begin{aligned} \frac{d\bar{S}_H}{dt} &= B_H N_H - \beta \bar{S}_H \bar{I}_M - \mu_h \bar{S}_H + \delta \bar{R}_H \\ \frac{d\bar{I}_H}{dt} &= \beta \bar{S}_H \bar{I}_M - (\mu_h + \mu_d + \eta - \theta)\bar{I}_H \\ \frac{d\bar{R}_H}{dt} &= \eta \bar{I}_H - (\mu_h + \delta)\bar{R}_H. \end{aligned} \tag{1}$$

Similarly, for the flow of transmission of Dengue virus in mosquito population, we have the system of equations as:

$$\begin{aligned} \frac{d\bar{S}_M}{dt} &= B_M N_M - \beta_M \bar{S}_M \bar{I}_M - \mu_m \bar{S}_M \\ \frac{d\bar{I}_M}{dt} &= \beta_M \bar{S}_M \bar{I}_M - \mu_m \bar{I}_M, \end{aligned} \tag{2}$$

with the two conditions $\bar{S}_H + \bar{I}_H + \bar{R}_H = N_H$ and $\bar{S}_M + \bar{I}_M = N_M$.

Without loss of generality, we can write with the proportions
$S_H = \frac{\bar{S}_H}{N_H}, I_H = \frac{\bar{I}_H}{N_H}, R_H = \frac{\bar{R}_H}{N_H}, S_M = \frac{\bar{S}_M}{N_M}, I_M = \frac{\bar{I}_M}{N_H}$.
Since $R_H = 1 - S_H - I_H$ and $S_M = 1 - I_M$, the above two systems (1) and (2) can be reduced to the following equivalent system:

$$\frac{dS_H}{dt} = B_H - \beta S_H I_M - \mu_h S_H + \delta(1 - S_H - I_H)$$
$$\frac{dI_H}{dt} = \beta S_H I_M - (\mu_h + \mu_z + \eta - \theta) I_H \quad (3)$$
$$\frac{dI_M}{dt} = \beta_M (1 - I_M) I_M - \mu_m I_M$$

The feasible region for the system (3) is as follows:
$\Gamma = \{(S_H, I_H, I_M) \in R^3 : S_H > 0, I_H \geq 0, I_M \geq 0, S_H + I_H \leq 1, I_M \leq 1\}$.

Stability of the model

In this section, we find the equilibrium states and basic reproduction number of the model. We also prove that our model is locally and globally stable for both disease-free-equilibrium and endemic equilibrium points.
Finding equilibrium states by setting the right-hand side of all the model equations of system (3) equal to zero, we obtain two equilibrium states:

(i) Disease free equilibrium state: $E_0 = (1,0,0)$
(ii) Endemic equilibrium state: $E_1 = (S_H^*, I_H^*, I_M^*)$

The system being modeled is expected to show different kinds of behavior in the long run. The equilibrium points and the conditions for their existence are that they provide us mathematical conditions based on which the long-term behavior of the system can be predicted and classified into a finite number of possibilities represented by the equilibrium points.
Endemic Equilibrium points of the system (3):

From the third and second equation of the system (3) by equating to zero, we get

$I_H = \frac{\beta S_H I_M}{\mu_m + \mu_d + \eta - \theta}$ and $I_H = \frac{\mu_m I_M}{\beta_M (1 - I_M)}$ respectively.

Solving it, we get

$S_H = \frac{\mu_m(\mu_m + \mu_d + \eta - \theta)}{\beta \beta_M (1 - I_M)}$.

Now, from the first equation of the system (3) by equating to zero, and putting the above value of S_H, we get

$\delta D I_M^2 - (C + \delta + A\beta_H) I_M + CD - AB = 0$, where $A = \mu_m(\mu_m + \mu_d + \eta - \theta)$, $B = \mu_d + \delta$, $C = B_H + \delta$ and $D = \beta\beta_H$.

Since all the parametric values are positive, so we consider only positive root of above equation and endemic equilibriums $E_1 = (S_H^*, I_H^*, I_M^*)$ are as follows:

$$I_M^* = \frac{C + \delta + A\beta_H + \sqrt{(C + \delta + A\beta_H)^2 - 4\delta D(CD - AB)}}{2\delta D},$$
$$I_H^* = \frac{\mu_m I_M^*}{\beta_M (1 - I_M^*)} \text{ and } S_H^* = \frac{\mu_m(\mu_m + \mu_d + \eta - \theta)}{\beta \beta_M (1 - I_M^*)}.$$

Basic reproduction number

For any epidemic model, the basic reproduction number is the average number of secondary infectious cases produced by a single infection in total susceptible population. The basic reproduction number is calculated by $R_0 = \rho(FV^{-1})$, where ρ is spectral radius of the matrix FV^{-1} and F & V are the matrices of new infection terms and the remaining transmission terms respectively.
For the systems (1) & (2), the matrices F and V are as follows:

$$F = \begin{bmatrix} 0 & \beta \\ \beta_M & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \mu_h + \mu_d + \eta - \theta & 0 \\ 0 & \mu_m \end{bmatrix}.$$

Hence, the basic reproduction number of the above model is

$$\boldsymbol{R_0} = \sqrt{\frac{\boldsymbol{\beta\beta_M}}{\boldsymbol{\mu_m(\mu_h + \mu_d + \eta - \theta)}}}.$$

Theorem 1 *: The system (3) is locally asymptotically stable for disease-free equilibrium, when* $R_0 < 1$.
Proof: Jacobian matrix of the system (3) is as follows:

$$J = \begin{bmatrix} -(\mu_h + \delta) & -\delta & -\beta \\ 0 & -(\mu_h + \mu_d + \eta - \theta) & 0 \\ 0 & \beta_M & -\mu_m \end{bmatrix}$$

The eigenvalues of Jacobian matrix J are as follows:

$\lambda_1 = -(\mu_h + \delta), \lambda_2 = -(\mu_h + \mu_d + \eta - \theta)$ and $\lambda_3 = -\mu_m$.

Hence, all eigenvalues of Jacobian matrix J are negative when $R_0 < 1$.
This proves that our the system is locally asymptotically stable when $R_0 < 1$.

Theorem 2*: The unique endemic equilibrium point* E_1 *is globally asymptotically stable if* $R_0 > 1$.
Proof: We will prove the global stability of endemic equilibrium E_1 using the geometric approach to global stability problems, developed by Li and Muldowney [15], which is discussed in briefly as follows:
Consider an autonomous dynamical system $\dot{Y} = f(Y)$, where $f(Y) \in \mathbb{R}^n$is C^1 function of Y in open subset Ω of $\mathbb{R}^n$.
Assumptions of two hypotheses:
H_1 : There exists a compact absorbing set K in Ω.
H_2 : Above dynamical system has unique equilibrium $\bar{Y}$ in Ω.

Define $\bar{q}_2 = lim_{t\to\infty}^{sup} \max_{y_0 \in \Omega} \frac{1}{t} \int_0^t \mu \left(B\left(y(s, y_0)\right)\right) ds$, where the Lozinskii measure of matrix B is defined as $\mu(B) = \lim_{h\to 0} \frac{|I + hB| - 1}{h}$.

If the system satisfy the above hypothesis H_1*-*H_2 *and* $\bar{q}_2 < 0$*, then the unique equilibrium* $\bar{Y}$ *is globally stable in* Ω.
For the general solution $\left(S_H(t), I_H(t), I_M(t)\right)$ of system (3), the Jacobian matrix is

$$J = \begin{bmatrix} -\beta I_M - \mu_h - \delta & -\delta & -\beta S_H \\ \beta I_M & -(\mu_h + \mu_d + \eta - \theta) & \beta S_M \\ 0 & \beta_M(1 - I_M) & -\beta_M I_H - \mu_m \end{bmatrix}.$$

The matrix $J^{[2]}$, the second additive compound matrix of the Jacobian for n=3, is defined as

$$J^{[2]} = \begin{bmatrix} j_{11} + j_{22} & j_{23} & -j_{13} \\ j_{32} & j_{11} + j_{33} & j_{12} \\ -j_{31} & j_{21} & j_{22} + j_{33} \end{bmatrix}.$$

So, its second additive compound matrix $J^{[2]}$ is

$$J^{[2]} = \begin{bmatrix} x & \beta_H S_H & \beta_H S_H \\ \beta_M(1 - I_M) & y & -\delta \\ 0 & \beta I_M & -(\mu_h + \mu_d + \eta - \theta) - \beta_M I_H - \mu_m \end{bmatrix},$$

where $x = -\beta I_M - \mu_h - \delta - (\mu_h + \mu_d + \eta - \theta)$ and
$y = -\beta I_M - \mu_h - \delta - \beta_M I_H - \mu_m$.

Let the function $P = P(S_H, I_H, I_M)$ be defined as

$$P = P(S_H, I_H, I_M) = \begin{bmatrix} 1 & 0 & 0 \\ 0 & \frac{I_H}{I_M} & 0 \\ 0 & 0 & \frac{I_H}{I_M} \end{bmatrix} = diag\left\{1, \frac{I_H}{I_M}, \frac{I_H}{I_M}\right\}$$

$$\text{Then, } P_f P^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{I_H'}{I_H} - \frac{I_M'}{I_M} & 0 \\ 0 & 0 & \frac{I_H'}{I_H} - \frac{I_M'}{I_M} \end{bmatrix} \qquad (4)$$

where P_f is the matrix obtained by replacing each element of P by its derivative in the direction of f.

$$P_f J^{[2]} P^{-1} = \begin{bmatrix} x & \beta S_H \frac{I_M}{I_H} & \beta S_H \frac{I_M}{I_H} \\ \beta_M(1 - I_M)\frac{I_H}{I_M} & y & -\delta \\ 0 & \beta I_M & -(\mu_h + \mu_d + \eta - \theta) - \beta_M I_M - \mu_m \end{bmatrix},$$

where $x = -\beta I_M - \mu_h - \delta - (\mu_h + \mu_d + \eta - \theta)$ and
$y = -\beta I_M - \mu_h - \delta - \beta_M I_H - \mu_m$.

$B = P_f P^{-1} + P_f J^{[2]} P^{-1} = \begin{bmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{bmatrix}$,

where $B_{11} = [-\beta I_M - \mu_h - \delta - (\mu_h + \mu_d + \eta - \theta)]$, $B_{12} = \begin{bmatrix} \beta S_H \frac{I_M}{I_H} & \beta S_H \frac{I_M}{I_H} \end{bmatrix}$, $B_{21} = \begin{bmatrix} \beta_M (1 - I_M) \frac{I_H}{I_M} \\ 0 \end{bmatrix}$ and

$B_{22} = \begin{bmatrix} y + \frac{I'_H}{I_H} - \frac{I'_M}{I_M} & -\delta \\ \beta I_M & -(\mu_h + \mu_d + \eta - \theta) - \beta_M I_M - \mu_m + \frac{I'_H}{I_H} - \frac{I'_M}{I_M} \end{bmatrix}$,

where $y = -\beta I_M - \mu_h - \delta - \beta_M I_H - \mu_m$.

Now, for a vector (u, v, w) in $\boldsymbol{R}^3$, we select a norm as $|(u, v, w)| = max\{|u|, |v + w|\}$ and denote $\mu(B)$ the Lozinskii measure for this norm.

From (4), it follows that $\mu(B) \leq \sup\{k_1, k_2\}$ (5)

where k_1 and k_2 are defined as follows:

$k_1 = B_{11} + |B_{12}|$ and $k_2 = \mu_1(B_{22}) + |B_{21}|$, where $|B_{12}|$ and $|B_{21}|$ are matrix norms with respect to the vector norm L^1 and μ_1 denotes the Lozinskii measure with respect to the vector norm L^1. So, we have

$$k_1 = B_{11} + |B_{12}|$$
$$= -\beta I_M - \mu_h - \delta - (\mu_h + \mu_d + \eta - \theta) + Sup\left\{\beta S_H \frac{I_M}{I_H}, \beta S_H \frac{I_M}{I_H}\right\}$$

$k_1 = -\beta I_M - \mu_h - \delta - (\mu_h + \mu_d + \eta - \theta) + \beta S_H \frac{I_M}{I_H}$ (6)

Similarly, $k_2 = \mu_1(B_{22}) + |B_{21}|$

$= \beta_M (1 - I_M) \frac{I_H}{I_M} - \beta I_M - \mu_h - \delta - \beta_M I_H - \mu_m + \frac{I'_H}{I_H} - \frac{I'_M}{I_M}$ (7)

From second and third equations of system (3), we can rewrite as

$\frac{I'_H}{I_H} + (\mu_h + \mu_d + \eta - \theta) = \beta S_H \frac{I_M}{I_H}$ (8)

$\frac{I'_M}{I_M} + \mu_m = \beta_M (1 - I_M) \frac{I_H}{I_M}$. (9)

Putting (8) and (9) in (7) and (6) respectively, we get

$k_1 = -\beta I_M + \frac{I'_H}{I_H} - (\mu_h + \delta) \leq \frac{I'_H}{I_H} - (\mu_h + \delta)$

$k_2 = -\beta I_M - \beta_M I_H + \frac{I'_H}{I_H} - (\mu_h + \delta) \leq \frac{I'_H}{I_H} - (\mu_h + \delta)$.

Hence, from (5)

$\mu(B) \leq \frac{I'_H}{I_H} - (\mu_h + \delta)$ and so, $\frac{1}{t}\int_0^t \mu(B) ds \leq \frac{1}{t} \log_e \frac{I'_H}{I_H} - (\mu_h + \delta)$.

So, $\bar{q}_2 < 0$, and hence the Bendixson criteria is also satisfied, which proves the global stability of the endemic equilibrium.

NUMERICAL SIMULATIONS AND EFFECT OF PARAMETRIC VALUES

In this section, using Runge-kutta-Fehlberg method of order 4 and 5 in MATLAB, we numerically simulate our system with the parametric values as given in Table 2 when $R_0 < 1$ & Table 3 when $R_0 > 1$ and establish the stability of models by taking different examples.

Example 1: Consider the system (1) with initial conditions $S_H = 9999$, $I_H = 1000$, $R_H = 100$, $S_M = 4999$, $I_M = 100$ and the parametric values as shown in Table 2. The simulation results are shown in figure 2, which illustrates the behavior of Susceptible, Infected, Recovered classes for human population and Susceptible, Infected classes for mosquito population. These are initially positive in the region of admissible values and asymptotically approach to the disease-free equilibrium for $R_0 = 0.0066 < 1$.

Table 2: Parametric values for Dengue virus model when $\mathbf{R_0 < 1}$.

Parameter	Value	Parameter	Value
β	0.004	η	0.2
β_M	0.01	θ	0.1
δ	0.6	μ_h	0.6
B_H	0.8	μ_m	0.4
B_M	0.7	μ_d	0.3

Table 3: Parametric values for Dengue virus model when $\mathbf{R_0 > 1}$.

Parameter	Value	Parameter	Value
β_H	0.5	η	0.5
β_M	0.7	θ	0.1
δ	0.9	μ_h	0.4
B_H	0.8	μ_m	0.8
B_M	0.7	μ_z	0.9

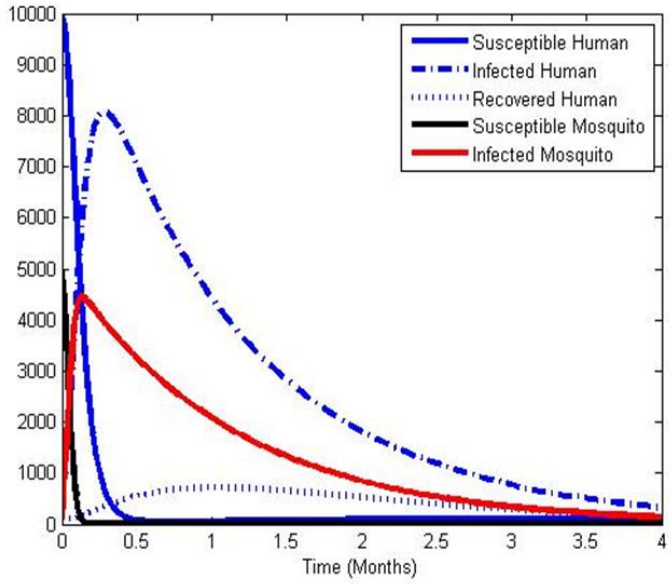

Fig. 2: Comparison of all classes for human and mosquito population.

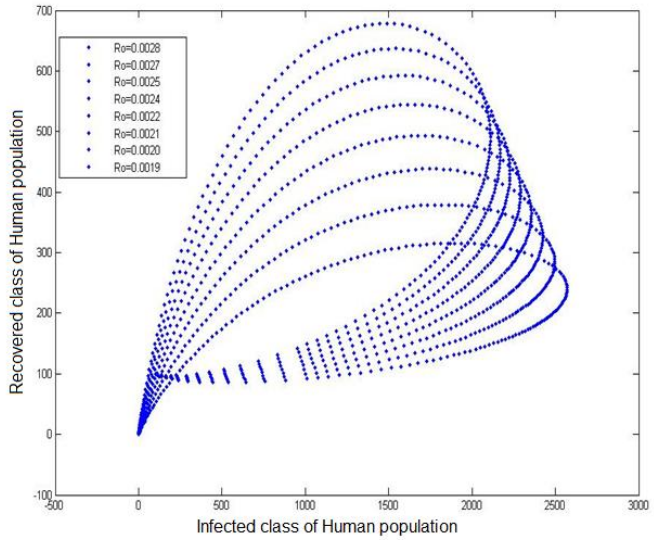

Fig. 3: Infected-recovered phase plane when $R_0 < 1$.

Example 2: Consider the system (1) with initial conditions $S_H = 9999, I_H = 100, R_H = 100, S_M = 999, I_M = 100$ and the parametric values as shown in Table 2. We simulate between infected humans versus recovered humans when $\eta = 0.2$, $\eta = 0.25, \eta = 0.3$, $\eta = 0.35, \eta = 0.4$, $\eta = 0.45, \eta = 0.5$ and $\eta = 0.55$, then the basic reproduction numbers are $R_0 = 0.019$, $R_0 = 0.020, R_0 = 0.021, R_0 = 0.022, R_0 = 0.024, R_0 = 0.025, R_0 = 0.027$ and $R_0 = 0.028$ respectively as shown in figure 3. From figure 3, we observe that the nature of trajectory tends to disease-free equilibrium point in infected-recovered phase plane, which shows the global stability of disease-free equilibrium point, when $R_0 < 1$.

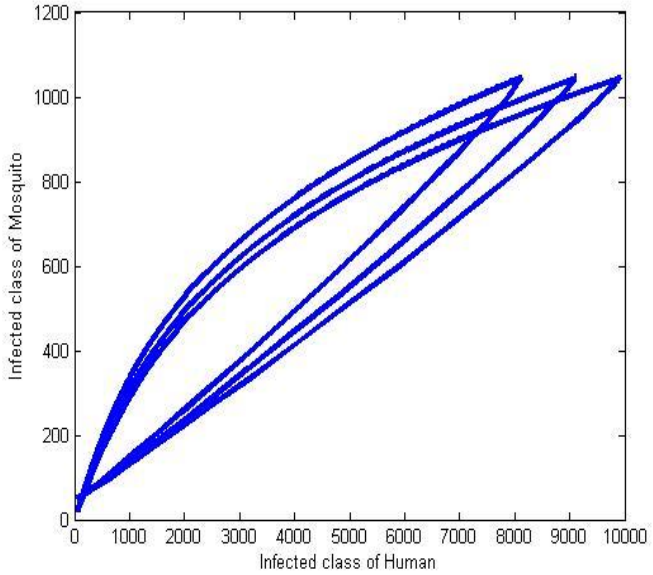

Fig. 4: infected human-infected mosquito plane when $R_0 > 1$.

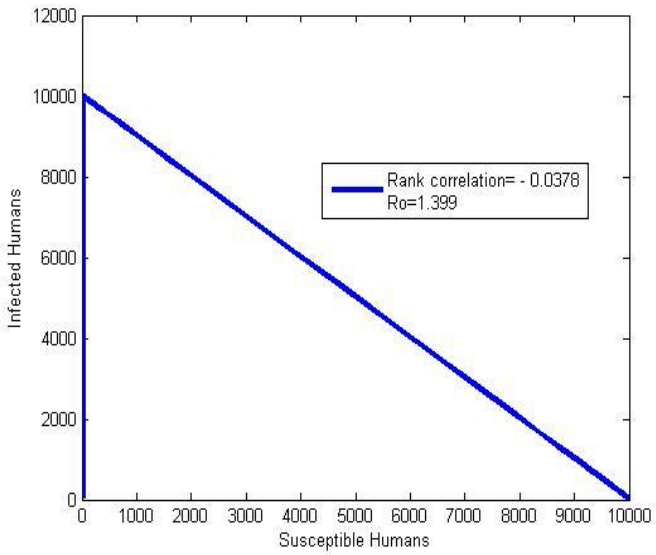

Fig. 5: Comparison between susceptible and infected human when $R_0 > 1$.

Example 3: To show the global stability of endemic equilibrium point, when $R_0 > 1$, we consider the global dynamics of the infected human-infected mosquito plane and try to understand the nature of the trajectory towards the endemic equilibrium point. Consider the system (1) with three initial conditions of different Susceptible humans $S_H =$

9899, $S_H = 9099, S_H = 8099$ and $I_H = 50, R_H = 10$, $S_M = 999, I_M = 50$ and with parametric values as shown in Table 2. From figure 4, we observe that the nature of trajectory is steady spiral tends to endemic equilibrium point from any three-initial point in infected human-infected mosquito plane, which shows the global stability of endemic equilibrium point, when $R_0 = 1.399 > 1$.

Example 4: To compare the number of susceptible humans with the number of infected humans when $R_0 > 1$, we consider the system (3) with initial conditions $S_H = 9999$, $I_H = 50$, $R_H = 10$, $S_M = 999$, $I_M = 50$ and the parametric values of Table 3. The simulation result is shown in figure 5, which illustrates that total numbers of susceptible humans get infected with Dengue virus and the total number of infected humans increase to $I_H = 9999$ when $R_0 = 1.399 > 1$. From the peak, the infected class decreases because there are no susceptible humans to be infected. The rank correlation co-efficient between susceptible and infected humans are equal to -0.0378. This means that when the number of infected human increases, the number of susceptible human decreases and vice-versa.

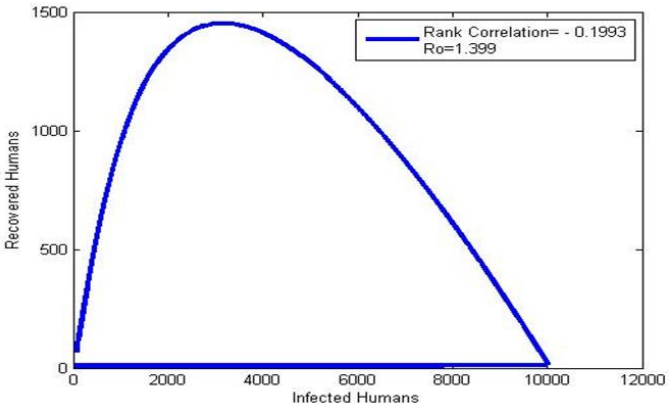

Fig. 6: Comparison between infected and recovered human when $R_0 > 1$

Example 5: To compare the number of recovered humans with the number of infected human when $R_0 > 1$, we consider the system (1) with initial conditions $S_H = 9999$, $I_H = 50$, $R_H = 10$, $S_M = 999$, $I_M = 50$ and the parametric values of Table 3. The simulation result is shown in figure 6. From figure 6, it is clear that number of recovered human increases for short time period and again becomes susceptible human. Hence the total recovered humans become susceptible and finally total numbers of susceptible humans get infected with Dengue virus and the total number of infected human increase to $I_H = 9999$ when $R_0 = 1.399 > 1$. From the peak, the infected class decreases because there are no susceptible humans to be infected. The rank correlation co-efficient between recovered and infected humans are equal to -0.1993.

Example 6: Consider the system (1) with initial condition $S_H = 9999, I_H = 100, R_H = 100$, $S_M = 999$, $I_M = 100$ and the parametric values as shown in Table 2. Figure 7 shows the efficacy of personal protection from the mosquitoes, for different values of β_H.

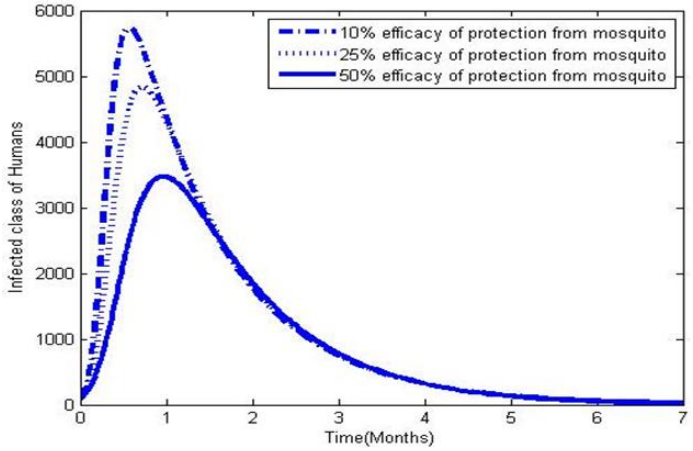

Fig. 7: Efficacy of personal protection of human from mosquito

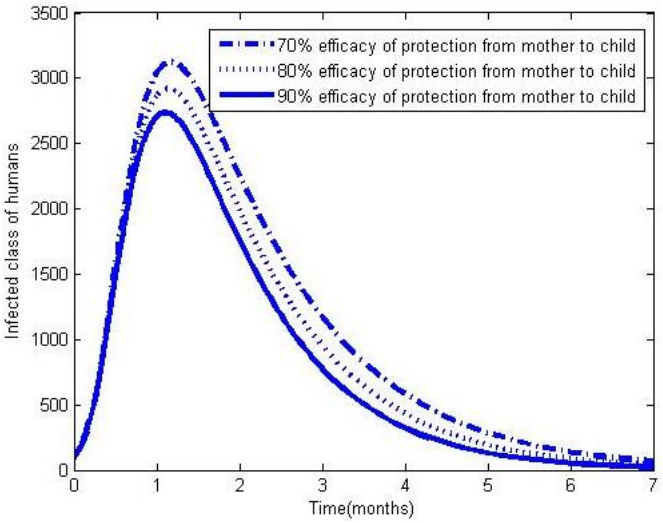

Fig. 8: Efficacy of personal protection from pregnant women to child.

Example 7: Consider the system (1) with initial conditions $S_H = 9999$, $I_H = 100$, $R_H = 100$, $S_M = 999$, $I_M = 100$ and the parametric values as shown in Table 2. Figure 8 shows the efficacy of personal protection from the pregnant women to newborn child for different values of vertical transmission θ.

CONCLUSION

The basic reproduction number of the model $R_0 = \frac{\beta_h \mu_h^2 + \sqrt{\mu_m \beta_H^2 + 4(\mu_h + \mu_z + \eta)\beta_M^2}}{2\mu_m^2(\mu_h + \mu_z + \eta)}$ is found and shown that the model is globally asymptotically stable at disease-free equilibrium when $R_0 < 1$. We proved the global stability of endemic equilibrium of the model using geometric approach with the Bendixson criteria when $R_0 > 1$. We also illustrate the analytical results numerically. If $R_0 < 1$, that means that an infective with Dengue virus replaces itself by less than one new infective with Dengue virus and the disease dies out. Furthermore, the susceptible proportion approaches one because everyone is susceptible when the disease has died out and the entire removed human beings who immune have died. If $R_0 > 1$ and the initial proportion of susceptible $S = S_H + S_M$ satisfies $SR_0 > 1$, then proportion of susceptible S

decreases and the infection proportion $I = I_H + I_M$ increases. The infection proportion I increases to peak, then decreases due to lack of sufficient susceptible to be infected. Due to births of new susceptible, when the susceptible proportion is larger and the secondary epidemic is smaller, then the solutions are spiral to endemic equilibrium as shown in figure 3 and figure 4. The comparisons between susceptible human class versus infected human class and infected human class versus recovered human class are shown in figure 5 and figure 6 respectively when $R_0 > 1$. The efficacy of personal protections in infected human is shown in figure 7. Figure 8 shows that the efficacy for pregnant women to newborn child by taking personal protection, which may include N, N-diethyl-m-toluamide (DEET), can be used on children aged greater than 2 months and on a precaution basis pregnant woman should not travel to any area where Dengue virus transmission is present.

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REFERENCES

1. WHO. Dengue and severe dengue. WHO; 2018. _http://www.who.int/mediacentre/factsheets/fs117/en/(accessed March 1, 2018).
2. SC Kliks, S Nimmanitya, A Nisalak, and DS Burke. Evidence that maternal dengue antibodies are important in the development of dengue hemorrhagic fever in infants. The American Journal of Tropical Medicine and Hygiene, 1988; 38: 411–9.
3. O Horstick, S Runge-Ranzinger, MB Nathan, A Kroeger. Dengue vector-controlservices: how do they work? A systematic literature review and countrycase studies. Transactions of The Royal Society of Tropical Medicine and Hygiene, 2010; 104: 379–86.
4. M. Derouich and A. Boutayeb, Dengue fever: Mathematical modelling and computer simulation, Applied Mathematics and Computation, 2006, 177(2):528-544.
5. Fred Brauer, Mathematical epidemiology: Past, present, and future. Infectious Disease Modelling, 2017, 2(2): 113-127.
6. RP Sanches and E Massad, A comparative analysis of three different methods for the estimation of the basic reproduction number of dengue, Infectious Disease Modelling, 2016, 1(1): 88-100.
7. S. Pollett, M.C. Melendrez IB Maljkovic, S Duchêne, H Salje. DAT Cummings and RG Jarman, Understanding dengue virus evolution to support epidemic surveillance and counter-measure development, Infection, Genetics and Evolution, , 2018, 62:279-295.
8. F Bacania, S Dimasb, IL Freireb, NA Maidanab, M Torrisi, Mathematical modelling for the transmission of dengue: Symmetry and travelling wave analysis, Nonlinear Analysis: Real World Applications, 2018,41: 269-287.
9. EP Pliego, J Velázquez-Castro, and AF Collar, Seasonality on the life cycle *of Aedes aegypti* mosquito and its statistical relation with dengue outbreaks, Applied Mathematical Modelling, 2017, 50: 484-496.
10. RWS Hendron and MB Bonsall, The interplay of vaccination and vector control on small dengue networks, Journal of Theoretical Biology, 2016, 407: 349-361.
11. T Sardar, S Rana, and J Chattopadhyay, A mathematical model of dengue transmission with memory, Communications in Nonlinear Science and Numerical Simulation, 2015, 22: 511-525.
12. WMS Yamashitaa, LT Takahashib, and G Chapiro, Traveling wave solutions for the dispersive models describing population dynamics of Aedes aegypti, Mathematics and Computers in Simulation, 2018, 146: 90-99.
13. HS Rodrigues, MTT Monteiro, DFM Torres, Dynamics of Dengue epidemics when using optimal control, Mathematical and Computer Modelling, 2010, 52: 1667–1673.
14. M Zhu, Z Lin,and Q Zhang, Coexistence of across-diffusive dengue fever model in a heterogeneous environment, Computers & Mathematics with Applications, 2018 75(3):1004-1015.
15. MY Li and JS Muldowney, A geometric approach to global-stability problem, SIAM Journal on Mathematical Analysis, 1996, 27: 1070-83.

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