Mathematical Study of Effect of Temperature on Transmission Dynamics of Dengue Disease

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Abstract: *Dengue fever is found in tropical and subtropical regions around the world. It is a vector-borne disease that is transmitted by female aedes mosquitoes infected with one of four dengue viruses (DENV1-DENV4). SEIR compartmental model is used to examine the transmission of the disease in the present work. The model has four compartments for the human population, susceptible, exposed, infected, and recovered and also four compartments for the mosquito population immature, susceptible, exposed, and infected. The impact of temperature on the dynamics of dengue disease transmission is described by this model. The basic reproduction number of the model is computed by implementing the Next Generation Matrix Method. Sensitivity analysis is performed to establish the relative importance of the model parameters and mathematical results are shown graphically.*

Keywords: Dengue disease, SEIR model, Reproduction number, Sensitivity analysis

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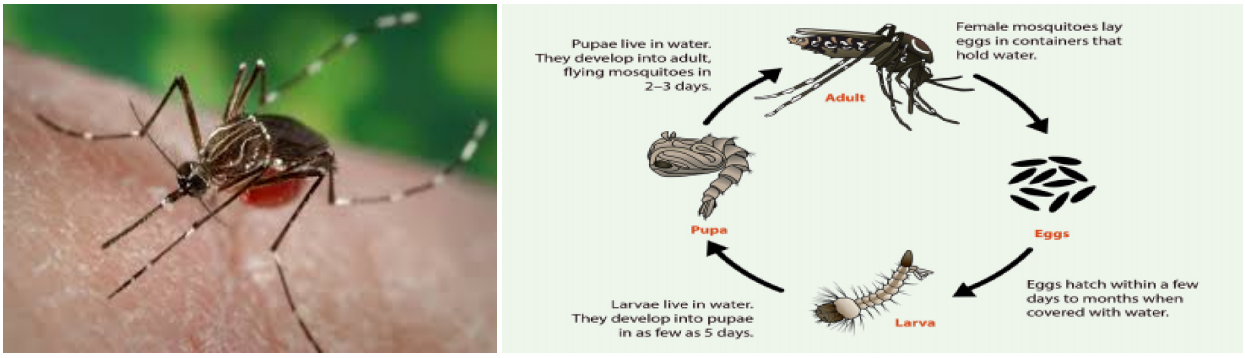
1 Introduction

Aedes mosquito bite can transmit the systemic viral illness dengue to humans. Dengue can be a fatal illness for certain people [28]. In addition to the significant vector control measures, there are presently no licensed vaccinations or special medications that can stop the disease's fast growth and global spread [25]. The Spanish word dengue, which means fastidious or meticulous and would represent the walk of a person experiencing the bone pain caused by the Dengue fever [29], may have been the source of the Swahili word dinga. In the 1780s, Dengue fever epidemics were identified in Asia, Africa, and North America at the same time [29]. The earliest known case report was made in 1789. It is primarily spread by female aedes mosquitoes. Any one of the four dengue virus serotypes can cause dengue [3, 20].

In recent decades, dengue cases have drastically increased all across the world. Dengue cases are reported since a large percentage of them are asymptomatic, moderate, and self-managed. In many instances, other febrile infections are incorrectly diagnosed [26]. According to Poudel [16] a variety of diseases, including severe and even deadly dengue hemorrhagic fever (DHF), dengue shock syndrome, and moderate or asymptotic dengue fever (DF), can be brought on by the dengue virus (DSS).

For the investigation of dengue transmission we employ the SEIR compartmental model. The host population is split into four categories: susceptible, exposed, infected, and recovered, and the mosquito population is divided into four categories: immature, susceptible, infected, and exposed [14, 23]. Here the recovered class is not included for mosquito population. Mosquitoes can not recover from dengue due to their short life span. Approximately, half of the world's population is currently in danger of dengue disease. Although more than half of world population infections occur annually, more than 80 percent of them are often minor and asymptomatic. Approximately 20,000 die from severe
dengue [28].

Dengue fever is one of the most dangerous diseases in Nepal. Dengue fever was first reported in 2004 [18]. It just reported 10,808 dengue cases from 55 districts in 2021. Chitwan, Kaski, Rupendhei, and Kathmandu have recorded the majority of instances. Dengue fever could affect the majority of Nepalese population as major population is classified into tropical and subtropical regions [13].
To conduct a mathematical research on the effect of temperature on the transmission dynamics of dengue



A **B**

Figure 1: **A:** Aedes Aegypti mosquito biting to a human: **B:** Life cycle of mosquito passes from different phases [31, 32].

infections, it is vital to understand what factors are affected by temperature. Mosquitoes go through four stages, all of which are affected by temperature. Female Aedes mosquitoes produce 100 to 200 eggs, each batch on average after consuming a blood meal. It has the capacity to lay up to 5 batches of eggs in its lifetime. Transmission into the larvae phase takes 2-7 days to a month. Transmission period fluctuates according to temperature [30]. The life cycle of Aedes Aegypti mosquitoes goes through four instars, spending some time in each. The fourth instar can take up for three days. As the temperature rises, so does the progression, and vice versa [30].

It can take up to five days for the pupae stage to appear. It takes up to 2-3 days for this adult mosquito to emerge by eating air to expand the abdomen and break open the pupae. Adult mosquitoes are the last stage of development. Mosquitoes can fly and travel a short distance. The movement of adult mosquitoes here varies with temperature [17].

Dengue disease has a long history an extensive research are found to be carried out dengue sickness. It has become a threat to the entire world's population. Many scientists and researchers are attempting to build mathematical theories on dengue sickness.

To describe the epidemic sickness, Kermack and McKendrik [9] created a SIR model. This laid the groundwork for the dengue fever epidemic. A dengue disease transmission model with a fluctuating human population was proposed by Estava and Vargas [4]. By defining threshold conditions, they proved the existence of endemic equilibrium. Bartlett [1] designed the stochastic compartmental model. This led to the conclusion that extinction is significant in vector-borne dynamics. It gives an idea to the concept of critical community size in epidemic theory, such as Measles. Fischer [5] used a mathematical model to establish a novel model for dengue related to pathogen of dengue hemorrhagic fever and sequential infection rates. The age distribution of dengue hemorrhagic fever is used to validate subsequent infections. It goes over the double and triple sequential models.

Koopman [10] established a theory of the determinants and predictors of dengue infection. Focks [7] developed a simulation model of urban dengue fever epidemiology. This model takes into account virus development within people as well as virus transmission within. Uncertainties with dengue models was established by Luz et al. [12] in Rio de Janeiro, Brazil. According to this concept, regional variation in the vector population increases the likelihood of epidemics and complicates management methods. This model provides parameters worth investigating in order to develop the dengue transmission model.

Takahasi et al. [24] developed a mathematical model to outline practical strategies for reducing its impact as a dengue vector. In many circumstances, a continuous model demonstrates the presence of a stable traveling wave, and numerical analysis ties wavefront speed to a few critical parameters. Hyun et al. [27] built a model that examines the effect of temperature on dengue transmission. It addressed two simple model parameters controlled experiments and an entomological parameter related to the life cycle of mosquitoes in different temperature levels. It compares the vector reproduction and infection rate.

A mathematical model of dengue transmission with memory was developed by Sardar et al. [21]. The model incorporates memory. It establishes the fractional derivatives α and R_o's relationship to the stability criterion of the disease-free equilibrium E_0. In dengue ODE models, it has a synergistic effect with the idea of backward bifurcation. Additionally, control techniques in respect to the cutoff quantity R_o were studied. Phaijoo and Gurung [15] investigated the impact of temperature and human movement on the persistence of dengue sickness in many patches. They demonstrates that temperature has a substantial impact on the transmission dynamics of dengue illnesses by employing a temperature dependent parameter. Robert et al. [19] looked into how temperature affected dengue fever. They used deterministic ordinary differential equations to examine how temperature affected dengue transmission. They found that the chance of dengue transmission has increased due to climate change.

2 SEIR Model for the Transmission of Dengue Disease

2.1 Mathematical model

The model consists of the four compartments for human population namely Susceptible (S_h), Exposed (E_h), Infected (I_h) and Recovered (R_h). It also consists of four compartments Immature (L), Susceptible (S_v), Exposed (E_v), Infected (I_v) for mosquito population. Figure 2 shows the SEIR model fot the transmission of dengue disease.

As mosquito life starts from the egg it goes through different phases like larvae and pupae these stages are called Immature which is denoted by L in Figure 2. To reproduce young one, cycle takes 12-13 days, but due to increase of temperature, within 8 days the cycle completes.

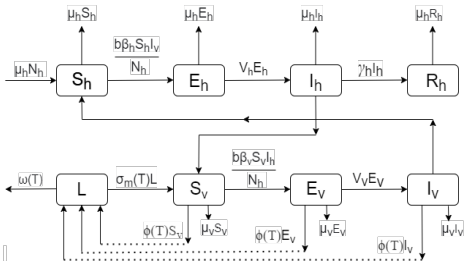


Figure 2: Flow chart of SEIR model for transmission of dengue disease [2].

System of differential equations which describes the dynamic of dengue disease is

$$\frac{dS_h}{dt} = \mu_h N_h - \mu_h S_h - \frac{b\beta_h S_h I_v}{N_h} \tag{1}$$

$$\frac{dE_h}{dt} = \frac{b\beta_h S_h I_v}{N_h} - \mu_h E_h - V_h E_h \tag{2}$$

$$\frac{dI_h}{dt} = V_h E_h - \mu_h I_h - \gamma_h I_h \tag{3}$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h \tag{4}$$

$$\frac{dL}{dt} = \phi(t)[S_v + E_v + I_v] - \omega(T)L - \sigma_m(T)L \tag{5}$$

$$\frac{dS_v}{dt} = \sigma_m(T)L - \mu_v S_v - \frac{b\beta_v S_v I_h}{N_h} \tag{6}$$

$$\frac{dE_v}{dt} = \frac{b\beta_v S_v I_h}{N_h} - \mu_v E_v - V_v E_v \tag{7}$$

$$\frac{dI_v}{dt} = V_v E_v - I_v \mu_v \tag{8}$$

The parameters in the model equations 1-8 are

Variables	Description
μ_h	Mortality rate for people.
μ_v	Mortality rate of mosquito.
β_h	Probability transmission from mosquito to human.
β_v	Probability transmission from human to mosquito.
b	Biting rate of mosquito.
γ_h	Recovery rate of host.
V_h	Development of host exposure.
V_v	Development of exposed mosquitoes.
ϕ_T	Egg deposition rate of mosquito.
σ_m	Maturity rate of mosquito.
$\omega_l(T)$	Mortality rate of Immature mosquito.

The aforementioned system of differential equation is resolved using the Next Generation Matrix approach to yield to the basic reproduction number. There are a few characteristics in mosquito population that rely on temperature. Numerous studies have shown that such a relationship exists and may be quantitatively explained [8]. The infected mosquito bite humans for meal, and humans become infectious and becomes capable of transmitting the virus to mosquito. Temperature affects the daily biting rate, extrinsic incubation period, mortality rate of mosquitoes and probability of infection from human to mosquito or mosquito to human.

2.2 Basic reproduction number

The given system of ODEs are divided into disease and non-disease classes. The equations (2), (3), (7), and (8) represent disease classes through which disease transmission occurs. Remaining equations in the above system of ODE's form the non-disease classes.

$$\mathcal{F} = \begin{pmatrix} \frac{b\beta_h S_h I_v}{N_h} \\ \frac{b\beta_v S_v I_h}{N_h} \\ 0 \\ 0 \end{pmatrix}, \mathcal{V} = \begin{pmatrix} \mu_h E_h + V_h E_h \\ \mu_v E_v + V_v E_v \\ -V_h E_h + \mu_h I_h + \gamma_h I_h \\ -V_v E_v + I_v \mu_v \end{pmatrix} \tag{9}$$

The secondary infection is a function of E_h, I_h, E_v, I_v, from equation (9)
$f(E_h, E_v, I_h, I_v) = \frac{b\beta_h S_h I_v}{N_h}$, $g(E_h, E_v, I_h, I_v) = \frac{b\beta_v S_v I_h}{N_h}$, $h(E_h, E_v, I_h, I_v) = 0$, $i(E_h, E_v, I_h, I_v) = 0$
The matrix $\mathbf{F}$ of transmission term is obtained as

$$\mathbf{F} = \begin{pmatrix} \frac{\partial f}{\partial E_h} & \frac{\partial f}{\partial E_v} & \frac{\partial f}{\partial I_h} & \frac{\partial f}{\partial I_v} \\ \frac{\partial g}{\partial E_h} & \frac{\partial g}{\partial E_v} & \frac{\partial g}{\partial I_h} & \frac{\partial g}{\partial I_v} \\ \frac{\partial h}{\partial E_h} & \frac{\partial h}{\partial E_v} & \frac{\partial h}{\partial I_h} & \frac{\partial h}{\partial I_v} \\ \frac{\partial i}{\partial E_h} & \frac{\partial i}{\partial E_v} & \frac{\partial i}{\partial I_h} & \frac{\partial i}{\partial I_v} \end{pmatrix} = \begin{pmatrix} 0 & 0 & 0 & \frac{b\beta_h S_h}{N_h} \\ 0 & 0 & \frac{b\beta_v S_v}{N_h} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{10}$$

Again, from equation (10)
$f(E_h, E_v, I_h, I_v) = \mu_h E_h + V_h E_h$,
$g(E_h, E_v, I_h, I_v) = \mu_v E_v + V_v E_v$,
$h(E_h, E_v, I_h, I_v) = -V_h E_h + \mu_h I_h + \gamma_h I_h, \quad i(E_h, E_v, I_h, I_v) = -V_v E_v + I_v \mu_v$

The matrix $\mathbf{V}$ of transmission term is obtained as

$$\mathbf{V} = \begin{pmatrix} \frac{\partial f}{\partial E_h} & \frac{\partial f}{\partial E_v} & \frac{\partial f}{\partial I_h} & \frac{\partial f}{\partial I_v} \\ \frac{\partial g}{\partial E_h} & \frac{\partial g}{\partial E_v} & \frac{\partial g}{\partial I_h} & \frac{\partial g}{\partial I_v} \\ \frac{\partial h}{\partial E_h} & \frac{\partial h}{\partial E_v} & \frac{\partial h}{\partial I_h} & \frac{\partial h}{\partial I_v} \\ \frac{\partial i}{\partial E_h} & \frac{\partial i}{\partial E_v} & \frac{\partial i}{\partial I_h} & \frac{\partial i}{\partial I_v} \end{pmatrix} \tag{11}$$

$$= \begin{pmatrix} \mu_h + V_h & 0 & 0 & 0 \\ 0 & \mu_v + V_v & 0 & 0 \\ -V_h & 0 & \gamma_h + \mu_h & 0 \\ 0 & -V_v & 0 & \mu_v \end{pmatrix} \tag{12}$$

2.3 Disease free equilibrium (DFE)

In the absence of disease,

$$I_h = 0,\ E_h = 0,\ E_v = 0,\ I_v = 0 \text{ and } R_h = 0$$

From 1

$$\begin{gathered} \frac{dS_h}{dt} = 0, \text{ gives} \\ N_h = S_h \end{gathered} \tag{13}$$

From equation 5

$$\begin{gathered} 0 = \phi N_v - L(\omega + \sigma_m) \\ L(\omega + \sigma_m) = \phi N_v \end{gathered}$$

$$L = \frac{\phi N_v}{(\omega + \sigma_m)} \tag{14}$$

From equation 6

$$\begin{gathered} 0 = \sigma_m L - \mu_v S_v - 0 \\ \text{or, } \sigma_m L = \mu_v S_v \\ \text{or, } S_v = \tfrac{\sigma_m}{\mu_v}.L \end{gathered}$$

$$\therefore S_v = \frac{\sigma_m}{\mu_v}.\frac{\phi N_v}{\omega + \sigma_m}$$

Now,

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & b\beta_h \\ 0 & 0 & \frac{b\beta_v S_v}{N_h} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \cdot \begin{pmatrix} \frac{1}{\mu_h + \gamma_h} & 0 & 0 & 0 \\ 0 & \frac{1}{\mu_v + V_v} & 0 & 0 \\ \frac{\mu_v^2 V_h + \mu_v V_h V_v}{\mu_v(\gamma_h + \mu_h)(\mu_h + V_h)(\mu_v + V_v)} & 0 & 0 & 0 \\ 0 & \frac{V_v}{\mu_v(\mu_v + V_v)} & 0 & \frac{1}{\mu_v} \end{pmatrix}$$

$$= \begin{pmatrix} 0 & \frac{b\beta_h V_v}{\mu_v(\mu_v + V_v)} & 0 & \frac{b\beta_h}{\mu_v} \\ \frac{(\mu_v^2 V_h + \mu_v V_h V_v) b\beta_v S_v}{\mu_v(\gamma_h + \mu_h)(\mu_h + V_h)(\mu_v + V_v) N_h} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{15}$$

$$det(FV^{-1} - \lambda \mathrm{I})$$

$$= \begin{vmatrix} -\lambda & \frac{b\beta_h V_v}{\mu_v(\mu_v+V_v)} & 0 & \frac{b\beta_h}{\mu_v} \\ \frac{(\mu_v^2 V_h+\mu_v V_h V_v)b\beta_v S_v}{\mu_v(\gamma_h+\mu_h)(\mu_h+V_h)(\mu_v+V_v)N_h} & -\lambda & 0 & 0 \\ 0 & 0 & -\lambda & 0 \\ 0 & 0 & 0 & -\lambda \end{vmatrix}$$

Suppose $a = \frac{(\mu_v^2 V_h+\mu_v V_h V_v)b\beta_v S_v}{\mu_v(\gamma_h+\mu_h)(\mu_h+V_h)(\mu_v+V_v)N_h}$ and $b = -\frac{b\beta_h V_v}{\mu_v(\mu_v+V_v)}$, then

$$det(FV^{-1} - \lambda \mathrm{I}\ ) = 0 \text{ gives}$$
$$\lambda = 0,$$
$$\text{or, } \lambda^2 = ab$$

$$i.e\ \ \lambda^2 = \left[\frac{(\mu_v^2 V_h + \mu_v V_h V_v)b\beta_v S_v}{\mu_v(\gamma_h + \mu_h)(\mu_h + V_h)(\mu_v + V_v)N_h}\right] \cdot \left[-\frac{b\beta_h V_v}{\mu_v(\mu_v + V_v)}\right]$$

$$= \frac{\mu_v V_h(\mu_v + V_v)b\beta_v S_v b\beta_h V_v}{\mu_v(\gamma_h + \mu_h)(\mu_h + V_h)(\mu_v + V_v)N_h\mu_v(\mu_v + V_v)}$$

At,

$$S_v = \frac{\sigma_m}{\mu_v}.\frac{\phi N_v}{\omega + \sigma_m},$$

$$\lambda^2 = \frac{\mu_v V_h(\mu_v + V_v)b\beta_v b\beta_h V_v \sigma_m \phi N_v}{\mu_v(\gamma_h + \mu_h)(\mu_h + V_h)(\mu_v + V_v)N_h\mu_v(\mu_v + V_v)(\omega + \sigma_m)}$$

$$i.e\ \ \lambda^2 = \frac{V_h b^2 \beta_v \beta_h V_v \sigma_m \phi N_v}{N_h(\gamma_h + \mu_h)(\mu_h + V_h)(\mu_v + V_v)(\omega + \sigma_m)\mu_v}$$

Therefore,

$$R_0 = \lambda = \sqrt{\frac{V_h b^2 \beta_v \beta_h V_v \sigma_m \phi N_v}{N_h(\gamma_h + \mu_h)(\mu_h + V_h)(\mu_v + V_v)(\omega + \sigma_m)\mu_v}} \tag{16}$$

Here R_o is the basic reproduction number.

Temperature affects both the daily bite rate and the possibility of transmission of an infection from a human to a mosquito or a mosquito to a human. A female Aedes aegypti's daily bite rate, b rise linearly with temperature for $21^oC < T^o < 32^oC$ [22]

$$\mathrm{b(T)} = 0.0943 + 0.0043\mathrm{T}$$

The possibility that a human will becomes infected by a mosquito after being bitten is given by the expression

$$\beta_v(T) = \begin{cases} \text{-}0.9037 + 0.0729T & \text{if } T < 26.1\,^oC \\ 1, & \text{otherwise} \end{cases}$$

for the temperature range $12.4\,^oC < T^o < 26.16\,^oC$ and it is equal to 1 for $26.1^oC < T < 32.5^oC$ [11].

The possibility that an infected mosquito would infect to a human by a bite β_h increases when $12.4\,^oC < T < 28\,^oC$ and dramatically declines when $T > 28\,^oC$, and equals zero when $T > 32.5\,^oC$. β_h(T) is given as [6]:

$$\beta_h(T) = \begin{cases} 0.001044(T-12.26)\sqrt{32.461-T} & \text{if } T < 32.5\,^oC \\ 0 & \text{otherwise} \end{cases}$$

The reciprocal of the extrinsic incubation period is denoted by V_v. It can be explained by the equation below [6]

$$V_v = 4 + exp(5.15 - 0.123T)$$

The following equations give the definition of μ_v, which stands for the mosquito mortality rate

$$\mu_v = 0.8692 - 0.159T + 0.01116T^2 - 3.408X10^{-4}T^3 + 3.809X10^{-6}T^4$$

2.4 Local stability of disease free equilibrium:

Theorem 1: The disease free equilibrium point for the system of equation is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ [15].

Proof: At the disease free equilibrium point, The Jacobian matrix of the system of differential equations (1-8) is

$$\mathbf{J} = \begin{pmatrix} -(\mu_h + \frac{b\beta_h I_v}{N_h}) & 0 & 0 & 0 & -(\frac{b\beta_h S_h}{N_h} + b\beta_h) \\ \frac{b\beta_h I_v}{N_h} & -(\mu_h + V_h) & 0 & 0 & 0 \\ 0 & V_h & -(-\mu_h + \gamma_h) & 0 & 0 \\ 0 & 0 & \frac{b\beta_v S_h}{N_h} & -(\mu_v + V_v) & 0 \\ 0 & 0 & 0 & V_v & -\mu_v \end{pmatrix} \tag{17}$$

The characteristics polynomial is

$$\lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4 = 0 \tag{18}$$

$A_1 = P_1 + P_2 + P_3 + P_4$
$A_2 = P_1(P_2 + P_3 + P_4) + P_2(P_3 + P_4) + P_3P_4$
$A_3 = P_1P_2P_3 + P_2P_3P_4 + P_3P_1P_4 + P_4P_1P_2$
$A_4 = P_1P_2P_3P_4 - B$
where, $P_1 = \mu_v$, $P_2 = (\mu_v + Vv)$, $P_3 = \mu_h + V_h$, $P_4 = (\mu_h + \frac{b\beta_h I_v}{N_h})$,
$P_5 = \mu_h + \gamma_h$, B=$\frac{b^2\beta_h\beta_v S_v}{N_h}.V_vV_h$

We must show that the roots of the polynomial are located on the left side of the complex plane in order to guarantee the stability of the disease-free equilibrium. The Routh-Hurwitz criteria are employed to determine this, for $i = 1, 2, 3, 4$.
$\det(H_1) = \det A_1 = P_1 + P_2 + P_3 + P_4 > 0$
$\det(H_2) = \begin{vmatrix} A_1 & 1 \\ 0 & A_2 \end{vmatrix} = A_1A_2$
$=(P_1 + P_2 + P_3 + P_4).[P_1(P_2 + P_3 + P_4) + P_2(P_3 + P_4) + P_3P_4] > 0$
$\det(H_3) = \begin{vmatrix} A_1 & 1 & 0 \\ A_3 & A_2 & A_1 \\ 0 & 0 & A_3 \end{vmatrix} = A_3(A_1A_2 - A_3) > 0$
$\det(H_4) = \begin{vmatrix} A_1 & 1 & 0 & 0 \\ A_3 & A_2 & A_1 & 1 \\ 0 & A_4 & A_3 & A_2 \\ 0 & 0 & 0 & A_4 \end{vmatrix}$
The disease-free equilibrium point will be asymptotically stable according to the Routh-Hurwitz criteria if

$R_0 < 1$, which is $det(H_i)$ for $i = 1, 2, 3, 4$.
here if $R_0 < 1$ then $R_0^2 < 1$

$$R_0^2 = \frac{b^2\beta_v\beta_h V_v V_h \sigma_m \phi N_v}{N_h(\gamma_h + \mu_h)(\mu_h + V_h)(\mu_v + V_v)(\omega + \sigma_m)\mu_v} < 1$$

$$R_0^2 = \frac{b^2\beta_v\beta_h V_v V_h S_v}{N_h(\gamma_h + \mu_h)(\mu_h + V_h)(\mu_v + V_v)\mu_v} < 1$$

$$\frac{B}{P_1P_2P_3P_4} < 1$$

Hence,

$$P_1P_2P_3P_4 - B > 0$$

then, $\det(H_4) > A_4(A_1A_2A_3 - A_1^2A_4 - A_3^2)$
All of the terms in the aforementioned polynomial are positive in the expression. $det(H_4)$ is hence positive. Hurwitz matrices have only positive determinants, Hence the polynomial (17) has roots with negative real parts. Therefore, if $R_o > 1$, the disease-free equilibrium is stable. On the other hand, if $R_o > 1$, $P_1P_2P_3P_4 - B < 0$ follows. Because $A_4 < 0$, none of the polynomial (17) roots can have negative real portions. It also mentions that if $R_o > 1$, the disease-free equilibrium point is unstable.

3 Numerical Results and Discussions

In this section, the effect of temperature on the transmission dynamics of dengue diseases is studied. Humans and mosquito populations vary with the fluctuation of temperature and variation of basic reproduction number at different temperature levels.

3.1 Disease dynamics of susceptible, exposed and infected humans population

The figure 3**A** demonstrates susceptible human population to temperature changes. At $29\,^oC$, when mosquito biting rates are at their peak and susceptible human populations are at their lowest, more susceptible people become ill and migrate into the exposed class. As the temperature rises, more people become susceptible than before.

The figure 3**B** demonstrates the disease transmission in dynamics of exposed humans at various temperature levels. It describes that the temperature affects incubation period of disease. As the temperature rises from $18\,^oC$. The biting rate of mosquitoes reaches its peak at $29\,^oC$, and more sensitive people become ill and therefore exposed. A larger population becomes afflicted as the temperature rises.

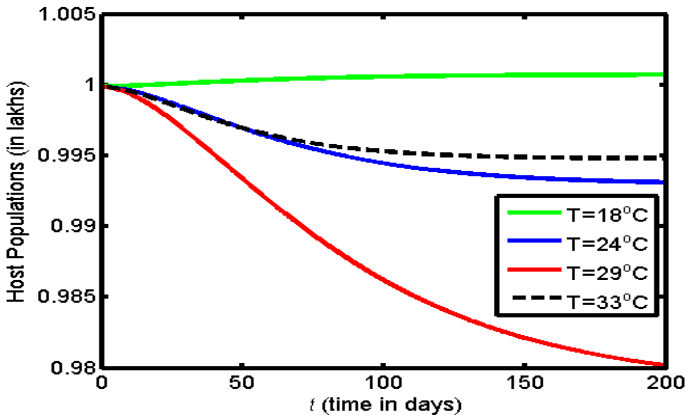
The figure 3**C** demonstrates the dynamics of infectious humans at various temperature levels. The population shrinks over time as a result of natural and dengue-induced death, as well as host population immunity development. At $18\,^oC$, there is a normal change, but at $24\,^oC$, there is significant change in temperature the infectious population reaches its highest value at $29\,^oC$. This is depicted by the red curve in 3**C**. Curve becomes flatter with increasing temperature until temperature reaches $33\,^oC$.

The preceding findings suggest that the human population size in the compartments change with temperature. Human susceptibility, exposure, and infection have all changed at $29\,^oC$. However, other temperature levels indicate the normal variation in the population. This demonstrates that temperature has a significant impact on the transmission of dengue disease.

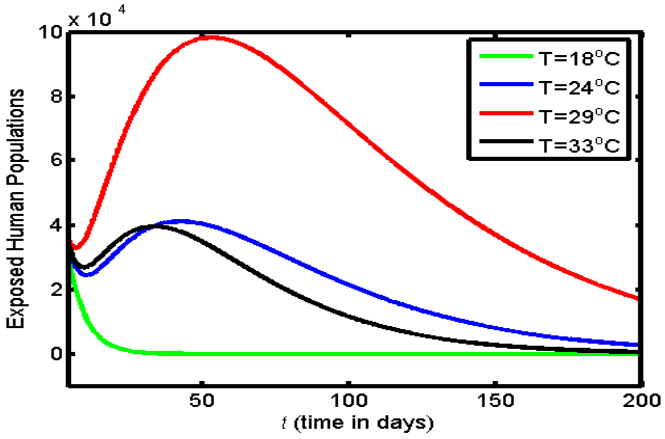
3.2 Transmission dynamic in susceptible, exposed and infected mosquito population

Figure 4**A** shows the dynamic of susceptible mosquito population at different temperature levels. It demonstrates that temperature has positive impact on mosquitoes. As the temperature level increases, the susceptible mosquito population increases. The populations attain its peak at $29\,^o$C temperature.

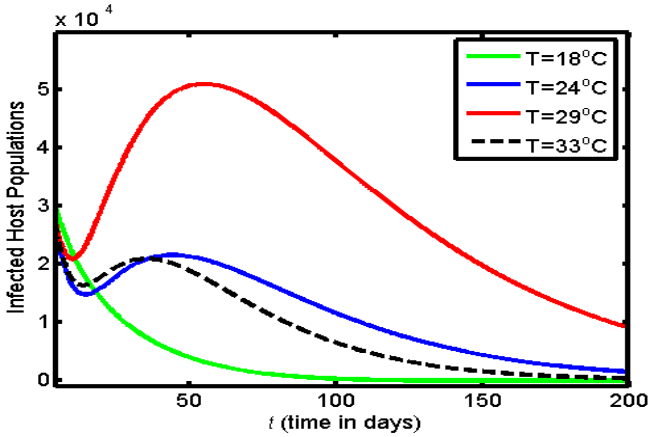
Figure 4**B** shows the dynamics of exposed mosquito population at different temperature levels. At $18\,^o$C,



A

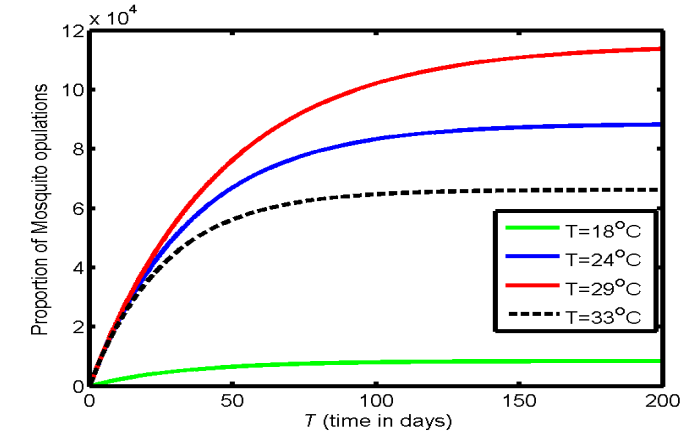


B

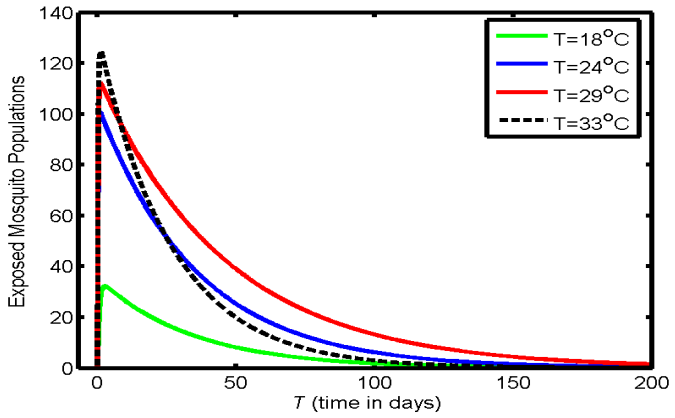


C

Figure 3: **A:** Susceptible human population with varying temperature levels. **B:** Exposed human population with varying temperature levels. **C:** Infected human population with varying temperature levels.



A



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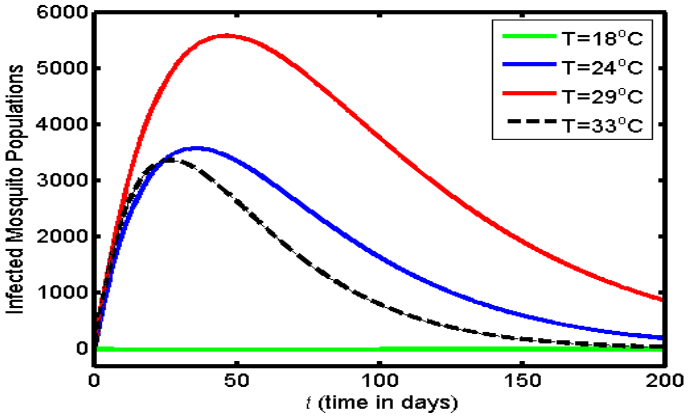


C

Figure 4: **A:** Disease Dynamic in Susceptible mosquito population. **B:** Disease Dynamic in Exposed mosquito population. **C:** Disease Dynamic in Infected mosquito population.

the mosquitoes populations are slightly increasing. As temperature goes to 24 ^{o}C more mosquitoes are getting infected than previous temperature. Infection of mosquito gets higher at temperature 29 ^{o}C and goes downward as increase of temperature level.

Figure 4**C** shows the dynamics of disease in infected mosquito population at different temperature levels. At 18 ^{o}C mosquitoes population increases slightly gets higher with increasing temperature levels. Mosquito

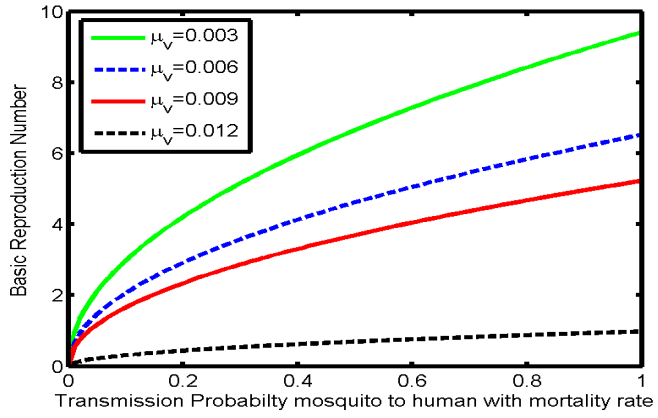
population reaches its peak at temperature 29 ^oC and goes downward as increase in temperature. This shows the positive impact of temperature on infected mosquito population.

4 Sensitivity Analysis

Sensitivity analysis indicates the importance of each parameter in disease spread. It helps in modeling and improving parameters that affect model structure. Because errors in data collection and expected parameter values are common, sensitivity analysis is routinely used to examine how sensitive a model's predictions are to parameter values. It is used to identify variables that have a significant impact on the basic reproduction number R_o [15]. Sensitivity indices of R_o at the baseline parameter values are given in Table 2:

Parameter	Sensitivity index
b	variable
μ_h	0.0000397
μ_v	0.071429
β_h	0.75
β_v	1
γ_h	0.75
V_h	0.071
V_v	0.071
ϕ_T	+ 0.5
σ_m	0.08
ω_l	0.012

[15]



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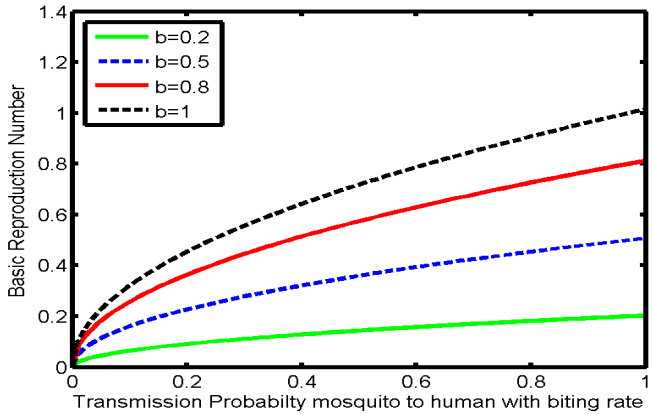

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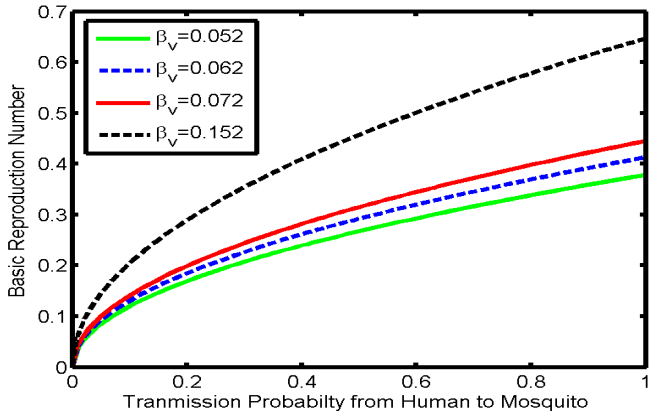

C

Figure 5: **A:** Basic Reproduction number vs probability of mosquito-to-human transmission. **B:** Basic Reproduction number vs probability of mosquito-to-human transmission based on biting rate. **C:** Basic Reproduction number vs Possibility from human to mosquito transmission.

Figure 5**A** shows the effect of transmission probabilities on the basic reproduction number. This demonstrates the positive impact on R_o. The graph depicts the effect of mosquito mortality on basic reproduction number. As the mosquito mortality rate is normal, the impact on basic reproduction number is greater. If we raise the mortality rate, R_o falls, implying that the probability of mosquito-to-human transmission falls, as shown by the red curve in the figure. If we keep the mortality rate as low as possible, the value of R_o decreases.

The figure 5**B** shows the effect of transmission probabilities on the basic reproduction number. The graph depicts the effect of mosquito death rate on basic reproduction number. When mosquito mortality rates are normal the influence on basic reproduction number is higher. If we can raise the mortality rate, the transmission probability from mosquito to human decreases, as demonstrated by the red curve in the picture. If we keep the mortality rate as low as possible, R_o will be reduced accordingly.

The impact of transmission probabilities on the basic reproduction number is seen in Figure 5**C**. The graph shows the effect of mosquito mortality rates on basic reproduction number. When the mosquito mortality rate is normal, it has a greater impact on the basic reproduction number. If we raise the mortality rate, the transmission probability of degree from mosquito to human decreases, as illustrated by the red curve in the figure. If we reduce the mortality rate as low as possible, R_o will be reduced accordingly.

The accompanying graph shows that the basic reproduction number fluctuates with different sensitive parameters. This graph shows the fluctuation in reproduction number as a function of bite rate, mortality rate, and transmission probability from human to mosquito. These parameters affect the basic reproduction number because they are all temperature dependent. As a result, we conclude that the basic reproduction number varies with temperature.

5 Conclusion

Mosquitoes are generally found in the tropical and subtropical regions. The earth's temperature is rising as a result of global warming. Mosquitoes are getting the habitat they need to survive on Earth. As the number of mosquitoes increases, so does the probability of dengue disease transmission. Because dengue fever does not really have a specific prescription. We need to understand how temperature affects mosquito life cycles and dispersal capacity. The basic reproduction number is investigated in this paper using the next generation matrix approach. The effect of temperature on this number is investigated, as well as sensitivity analysis and simulations. Based on the simulation results, we conclude that temperature has a crucial role in the spread of dengue disease. The activism of mosquito varies with temperature.

Conflicts of Interest: The authors declare that there are no conflicts of interest regarding the publication of this article

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