Mathematical Modelling of Dengue Fever Incorporating Vaccination as Control

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Abstract

This paper proposes a mathematical model of dengue fever incorporating vaccination. The proposed model is governed by system of first-order differential equations. The model is divided into eight (8) compartments namely; Vaccinated humans, Susceptible humans, exposed humans, Infected humans, Recovered humans. Susceptible vectors, Exposed vectors and Infected vectors. The positivity of the solution is established. We derived the disease-free equilibrium and endemic equilibrium states of the model and carried out stability on the disease-free equilibrium. The results show that the model is both locally and globally asymptotically stable when $R_0 < 1$ *and unstable otherwise. Finally, we numerically solved the model equations to examine the impact of some variables of the model. The graph shows that vaccination and treatment reduce the disease in the population.*

Keywords: Dengue fever, Vaccination, Loss of immunity, Local stability, Global stability, Reproduction number.

1.0 Introduction

Dengue fever is a mosquito-borne disease transmitted through the bites of a female Aedesaegypti mosquito (mosquito). This mosquito genus was first discovered in tropical and subtropical areas, but it has since spread to all continents[1]. Along with the flu, the dengue virus belongs to the flaviviridae family, which also includes other viruses spread by mosquitos and responsible for human diseases. Yellow fever, malaria, chikungunya, and zika virus are all flaviviruses. Although the cause of dengue fever is unknown, it is thought to have arisen in Africa. In the year 992, a Chinese medical encyclopedia published the first record of a clinically compatible disease[2]. Ren Kimura and Susumu Hotta discovered dengue virus in Nagasaki, Japan, and the first recorded dengue outbreak in Nigeria occurred in Abeokuta in which six strains of dengue (DEN–1) were isolated. There are no reports of dengue hemorrhagic fever in any of the patients[3]. Dengue virus is spread primarily by Aedes mosquitos, especially Aedesaegypti. These mosquitoes are most commonly found at elevations of 1,000 meters between the latitudes of 35° North and 35° South (3,300 feet). They bite most often in the early morning and late evening, but they can bite at any time and spread infection. Other Aedes species that transmit the disease include Aedesalbopictus, Aedespolynesiensis, and Aedesscutellaris. Humans are the most common

carriers of the virus, but it can also be present in nonhuman primates. An infection from a single bite is possible. A female mosquito that takes a blood meal from a person infected with dengue fever becomes infected with the virus in the cells lining her gut during the initial 2- to 10-day febrile period. After 8–10 days, the virus spreads to other tissues, including the mosquito's salivary glands, before being released into its saliva. The mosquito appears to be unaffected by the infection, as it remains infected for the remainder of its life. Since it prefers to lay its eggs in artificial water tanks, live next to humans, and feed on humans rather than other vertebrates, Aedesaegypti is particularly interested[4].

Over the last two decades, the number of dengue cases registered by the World Health Organization (WHO) has increased by over 8 times, from505,430 cases in 2000 to over 2.4 million individuals in 2019. Between 2000 and 2015, the number of deaths reported increased from960 to 4032[5]. Dengue fever was identified by the World Health Organization (WHO) as one of ten (10) diseases that could pose a threat in 2019, and the current epidemic in several countries backs up this claim. Dengue virus disease is caused by four closely related dengue viruses known as serotypes: DEN-1, DEN-2, DEN-3, and DEN-4. The genomes of these closely related viruses have around 65% in common. They target antibodies in slightly different ways, but the end result is the same. Dengue fever (DF), dengue hemorrhagic fever (DHF), and dengue shock syndrome (DSS) are the diseases caused by the dengue virus. Outbreaks of febrile illness compatible with dengue fever have been recorded all over the world, with the first epidemic occurring in the West Indies. When an individual is infected with the dengue virus, the virus binds to a human skin cell and starts to replicate. The skin cell membrane folds around the virus and forms a pouch that seals around the virus particle after this attachment. Endosome is the name for this pouch. Endosomes are usually used by a cell to take in large molecules and particles from the outside for nourishment [5].

The dengue virus can invade a host cell by hijacking normal cell processes. The majority of people infected with the dengue virus are asymptomatic (80%) or more have only minor symptoms including a fever[6], [7]. The signs of dengue fever vary depending on age, but the most common are high fever, extreme headache, muscle joint pains, pains behind the eyes, nausea, vomiting, and rashes. The incubation period for humans is eight to twelve (8-12) days, and the incubation period for vectors is four to seven days. Dengue infection confers immunity to dengue of the same serotype, but only for a limited time against the remaining serotypes. Dengue hemorrhagic fever and dengue shock syndrome are the most severe and life-threatening types of dengue fever. According to the available information, there is no particular prescription for treating dengue infection. Instead, doctors recommend paracetamol-based pain relievers and avoid aspirin-based medications, which may cause bleeding. Despite the fact that the Food and Drug Administration (FDA) of the United States of America has approved the use of a vaccine called dangvaxia to help prevent the disease from spreading to those who have already been infected. The best way to avoid contracting the disease is to use bed nets and close doors and windows, as well as to reduce the

2 Dengue Fever Model Formulations

In this paper, the model developed by $[8]$ is modified and made to consist of eight compartments ,Human population has five (5) compartments and the vector population has three(3) compartments .The total human population at time (t) denoted by (N_h), is divided into susceptible human (S_h), vaccinated human (V_h), exposed human (E_h), infected human (I_h), and recovered human (R_h), The total population is given as $N = S_h + E_h + I_h + R_h$, while the vector population consist of susceptible vector (S_v), exposed vector (E_v), and infected vector (I_v), and is given as $N = S_v + E_v + I_v$, The susceptible human population (S_h), grows through the recruitment rate $\Lambda_h N_h$ the class also gain number from those who lost immunity from vaccine due to vaccine duration at a rate ωV_h it also gain number from those leaving the recovered class at a rate σR_h , similarly it is reduced by natural death at a rate $\mu_h S_h$ again the class is reduced due to vaccination at a rate υS_h as well as those progressing to exposed class at a rate $\beta_h b I_v S_h / N_h$, The vaccinated human class (V_h), grows through the vaccination given to the susceptible human population at a rate υS_h is equally reduced by natural death at a rate $\mu_h V_h$ and those who lost their immunity due to vaccine duration at a rate ωV_h ,

The exposed human class grows as a result of susceptible humans being bitten by infected vector at a rate $\beta_h b I_v S_h / N_h$, and is reduced due to progression into the infected class at a rate $\phi_h E_h$ and is further reduced by natural death at a rate $\mu_h E_v$ it also gain number from those who lost their immunity due to vaccine duration at a rate $\psi \beta_h b I_v S_h / N_h$, the infected human class (I_h) recruitment is coming from the exposed class at a rate $\phi_h E_h$ it is also reduced by natural death at a rate $\mu_h I_h$ and dengue virus induced death at a rate δI_h it is further reduced by those who got treatment at a rate $\gamma_h I_h$. The recruitment into the recovered class (R_h) is done by treatment at a rate $\gamma_h I_h$ and is reduced by natural death at a rate μ_h as well as those returning to the susceptible class at a rate σR_h.

The susceptible vector (S_v) population grows through the recruitment rate $\Lambda_v N_v$ and is reduced by natural death at a rate μ_v it is also reduced by those migrating to the exposed class due to infection at a rate $\beta_v b I_h S_v / N_v$, the exposed vector class (E_v) recruitment is done through those susceptible vectors that got infected by infected human at a rate $\beta_v b I_h S_v / N_v$, and is reduced by natural death at a rate μ_v and those moving to the infected class at a rate ϕ_v . The infected

vector class (I_v) gain number from those coming from the exposed compartment at a rate $\phi_v E_v$ and reduced by natural death at a rate $\mu_v I_v$. The schematic diagram for dengue fever model with vaccination is presented in figure 1.

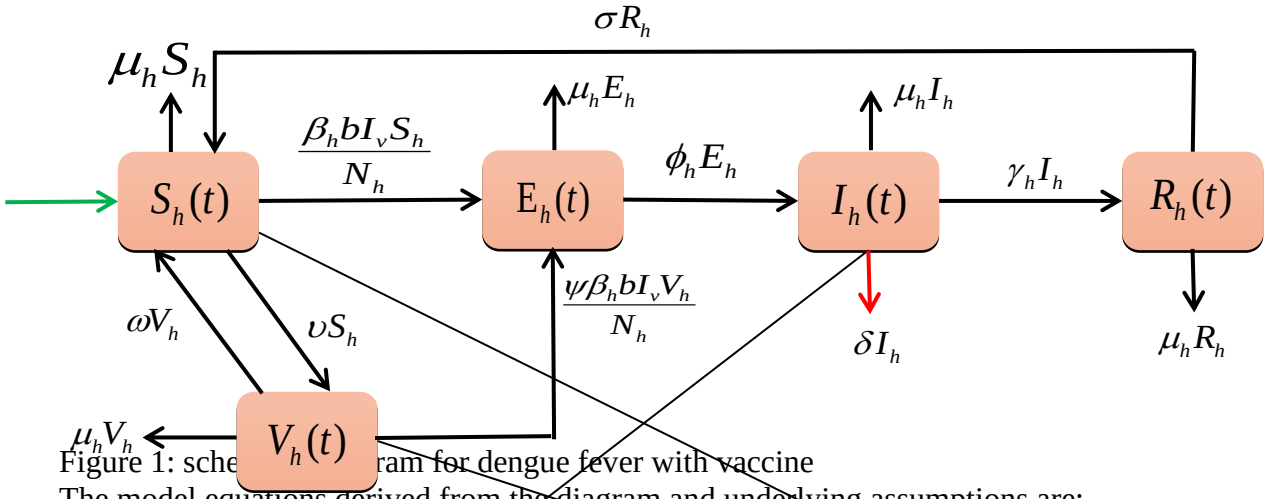


Figure 1: sche ram for dengue fever with vaccine

The model equations derived from the diagram and underlying assumptions are:

$$\frac{dV_h}{dt} = \upsilon S_h - \frac{\psi\beta_h b I_v V_h}{N_h} - \omega V_h - \mu V_h \quad (1)$$

$$\frac{dS_h}{dt} = \Lambda_h N_h + R_h - \upsilon S_h - \frac{\beta_h b I_v S_h}{N_h} \quad (2)$$

$$\frac{dE_h}{dt} = \frac{\beta_h b I_v S_h}{N_h} + \frac{\psi\beta_h b I_v V_h}{N_h} - \phi_h E_h - \mu_h E_h \quad (3)$$

$$\frac{dI_h}{dt} = \phi_h E_h - \mu_h I_h - \delta I_h - \gamma_h I_h \quad (4)$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h - \sigma R_h \quad (5)$$

$$\frac{dS_v}{dt} = \Lambda_v N_v - \frac{\beta_v b I_h S_v}{N_v} - \mu_h S_v \quad (6)$$

$$\frac{dE_v}{dt} = \frac{\beta_v b I_h S_v}{N_v} - \mu_v E_v - \phi_v E_v \quad (7)$$

$$\frac{dI_v}{dt} = \phi_v E_v - \mu_v I_v \quad (8)$$

With the initial conditions for human and vector are;

$V_h(0) = V_{h_0} \geq 0;\ S_h(0) = S_{h_0} \geq 0;\ E_h(0) = E_{h_0} \geq 0;\ R_h(0) = R_{h_0} \geq 0,$

and $S_v(0) = S_{v_0} \geq 0;\ E_v(0) = E_{v_0} \geq 0,;\ I_v(0) = I_v \geq 0.$

The total population of human is $N_h = S_h + E_h + I_h + R_h$ and the total population of vectors is $N_v = S_v + E_v + I_v$

Table 1 Variables for the model with vaccination

Variables	Description
S_h	Susceptible human population at time t.
E_h	Vaccinated human population at time t.
V_h	Exposed human population at time t.
I_h	Infected human population at time t.
R_h	Recovered human population at time t.
S_v	Susceptible vector population at time t.
E_v	Exposed vector population at time t.
I_v	Infected vector population at time t.

Table 2. Parameters for the model with *vaccination*

Parameters	Description
$\Lambda_h N_h$	Recruitment rate into the human population at time t.
$\Lambda_v N_v$	Recruitment rate into the vector population at time t.
ω	Progression from vaccinated human population into the susceptible human.
ϕ_h	Recruitment rate into the infected human population at time t
υ	Progression from susceptible human population into the vaccinated human
$\beta_h b$	Progression from susceptible human population into the exposed human
$\beta_h b$	Progression from susceptible vector population into the exposed vector

γ_h	Recruitment rate into the recovered human population from infected human at time t.
δ	Dengue virus induced death rate.
σ	Recruitment rate into the susceptible human from recovered human population at time t.
ϕ_v	Recruitment rate into the infected vector population at time t.
μ_h	Natural mortality rate for human
ψ	Progression rate from vaccinated human to exposed human

3. Analysis of the model with vaccine

3.1 Existence and positivity of solution

Here, we provide the following results which guarantee that the Dengue fever model with vaccine governed by equation (1)-(8) is epidemiologically and mathematically well-posed in a feasible region Ω given by

$\Omega = \Omega_h \cup \Omega_v \subset \mathbb{R}^5_+ \times \mathbb{R}^3_+$, Where

$$\Omega_h = \left\{ (V_h, S_h, E_h, I_h, R_h) \in R^5_+ : N_h \leq \frac{\Lambda_h N_h}{\mu_h} \right\}$$

$$\Omega_v = \left\{ (S_v, E_v, I_v) \in R^3_+ : \mathrm{N}_v \leq \frac{\Lambda_v N_v}{\mu_v} \right\}$$

3.2 Disease free equilibrium of Dengue fever model

From the equation (1)-(8) At dengue fever free

$E_h = I_h = E_v = I_v = 0$

Therefore the disease free equilibrium (DFE) is

$$\left(S_h^0, V_h^0, \mathrm{E}_h^0, I_h^0, R_h^0, S_v^0, E_v^0, I_v^0\right) =$$

$$\left(\frac{(\omega + \mu_h)\Lambda_h N_h^0}{(\omega + \mu_h)(\upsilon + \mu_h) - \omega\upsilon}, \frac{\upsilon \Lambda_h N_h^0}{(\omega + \mu_h)(\upsilon + \mu_h) - \omega\upsilon}, 0, 0, 0, \frac{\Lambda_v N_v^0}{\mu_v}, 0, 0 \right) \qquad (9)$$

3.3 Endemic equilibrium state

To find the endemic state where $E_h \neq 0, \mathrm{I}_h \neq 0, E_v \neq, I_v \neq 0, R_h \neq 0$ we solved equation (1) to (8) using substitution method

Therefore, the endemic equilibrium point is $E_0^* = \left(V_h^{*,}, S_h^*, \mathrm{E}_h^*, \mathrm{I}_h^*, R_h^{*,}, \mathrm{S}_v^*, \mathrm{E}_v^*, \mathrm{I}_v^*\right) =$

$$\left.\begin{aligned}
V_h^* &= \frac{\upsilon S_h^*}{\left(\omega+\mu_h\right)+b\psi\beta_h\dfrac{I_h^*}{N_h^*}} \\
S_h^* &= \frac{I_h^*\left(\phi_h+\mu_h\right)\left(\mu_h+\delta+\gamma_h\right)}{\phi_h\left(\dfrac{b\beta_h I_v^*\left(\left(\omega+\mu_h\right)N_h^*+\upsilon\psi N_h^*+b\psi\beta_h I_v^*\right)}{N_h^*\left(\left(\omega+\mu_h\right)N_h^*+b\psi\beta I_v^*\right)}\right)} \\
E_h^* &= \frac{I_h^*\left(\mu_h+\delta+\gamma_h\right)}{\phi_h} \\
I_h^* &= \frac{I_v^*\left(\mu_v+\phi_v\right)\mu_v^2 N_v^*}{b\beta_v\phi_v\left(\Lambda_v N^*{}_v-\dfrac{\mu_v}{\phi_v}I_v^*\left(\mu_v+\phi_v\right)\right)} \\
R_h^* &= \gamma_h\frac{I_h^*}{\left(\mu_h+\sigma\right)} \\
S_v^* &= \frac{I_v^*\left(\mu_v+\phi_v\right)N_v^*\mu_v}{b\beta_v\phi_v I_h^*} \\
E_v^* &= \frac{\mu_v}{\phi_v}I_v^* \\
I_v^* &= \frac{b\Lambda_v\beta_v\phi_v I_h^* N_v^*}{\mu_v^2\left(\mu_v+\phi_v\right)N_v^*+b\beta_v\mu_v I_h^*\left(\mu_v+\phi_v\right)}
\end{aligned}\right\} \qquad (10)$$

3.4 Basic Reproduction Number

The basic reproduction number, R_0, is the average number of secondary infection caused by an infectious individual during his/her entire life as an infectious individual. The threshold epidemiological of dengue denoted by $R_0 = \rho(\mathrm{FV}^{-1})$, where ρ denoted the spectral radius. Applying the next generation matrix method, we have four (4) infected classes, the exposed human, the infected human, the exposed vector and the infected vector. Let F_i be the vector rates of appearance of new infections in each compartment for $(\mathrm{i}=1,2,3,4)$, $V_i^+(\mathrm{x})$ be vector rate of transfer of individuals into the particular compartment of i by all other means, $V_i(\mathrm{x})$ be the vector rates of transfer of individuals out of particular compartment i. We can find $R_0 = \rho(\mathrm{FV}^{-1})$.

We have four (4) infectious classes (E_h, I_h, E_v, I_v) and the matrix showing the rate of new infections and transition compartments i is given by

$$F = \begin{bmatrix} E_h \\ I_h \\ E_v \\ I_v \end{bmatrix} = \begin{bmatrix} \frac{\beta_h I_v S_h}{N_h} + \frac{\psi \beta_h b I_v V_h}{N_h} \\ 0 \\ \frac{\beta_h I_v S_v}{N_v} \\ 0 \end{bmatrix} \qquad (11)$$

$$V_i = \begin{bmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{bmatrix} = \begin{bmatrix} \phi_h E_h + \mu_h E_h \\ \mu_h I_h + \delta I_h + \gamma_h I_h - \phi_h E_h \\ \mu_v E_v + \phi_v E_v \\ -\phi_v E_v + \mu_v I_v \end{bmatrix} \qquad (12)$$

The Jacobian matrix of equation (11) is evaluated as

$$F_i = \frac{\partial f_i}{\partial x_j} = \begin{bmatrix} \frac{\partial f_1}{\partial E_h} & \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial E_v} & \frac{\partial f_1}{\partial I_v} \\ \frac{\partial f_2}{\partial E_h} & \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial E_v} & \frac{\partial f_2}{\partial I_v} \\ \frac{\partial f_3}{\partial E_h} & \frac{\partial f_3}{\partial I_h} & \frac{\partial f_3}{\partial E_v} & \frac{\partial f_3}{\partial I_v} \\ \frac{\partial f_4}{\partial E_h} & \frac{\partial f_4}{\partial I_h} & \frac{\partial f_4}{\partial E_v} & \frac{\partial f_4}{\partial I_v} \end{bmatrix} \qquad (13)$$

Substituting equation (11) into (13) and evaluating at disease free equilibrium (E^0) we have

$$F = \begin{bmatrix} 0 & 0 & 0 & \frac{\beta_h b \Lambda_h S_h^0 + \psi \beta_h b V_h^0}{N_h} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_v b S_v^0}{N_v} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \qquad (14)$$

Substituting $q_1 = \frac{\beta_h b \Lambda_h \left((\omega + \mu_h) + \psi \upsilon \right)}{(\omega + \mu_h)(\upsilon + \mu_h) - \omega \upsilon}$ and $q_2 = \frac{\beta_v b \Lambda_v}{\mu_v}$ in (14) we get

$$F = \begin{bmatrix} 0 & 0 & 0 & q_1 \\ 0 & 0 & 0 & 0 \\ 0 & q_2 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{15}$$

Similarly evaluating the Jacobian matrix equation (12) we have

$$V = \frac{\partial v_i}{\partial x_j} = \begin{bmatrix} \frac{\partial v_1}{\partial E_h} & \frac{\partial v_1}{\partial I_h} & \frac{\partial v_1}{\partial E_v} & \frac{\partial v_1}{\partial I_v} \\ \frac{\partial v_2}{\partial E_h} & \frac{\partial v_2}{\partial I_h} & \frac{\partial v_2}{\partial E_v} & \frac{\partial v_2}{\partial I_v} \\ \frac{\partial v_3}{\partial E_h} & \frac{\partial v_3}{\partial I_h} & \frac{\partial v_3}{\partial E_v} & \frac{\partial v_3}{\partial I_v} \\ \frac{\partial v_4}{\partial E_h} & \frac{\partial v_4}{\partial I_h} & \frac{\partial v_4}{\partial E_v} & \frac{\partial v_4}{\partial I_v} \end{bmatrix} \tag{16}$$

Substituting equation (12) into (16) we get

$$V = \begin{bmatrix} \phi_h + \mu_h & 0 & 0 & 0 \\ \phi_h & \mu_h + \delta + \gamma_h & 0 & 0 \\ 0 & 0 & \mu_v + \phi_v & 0 \\ 0 & 0 & \phi_v & \mu_v \end{bmatrix} \tag{17}$$

Now letting $a_1 = \phi_h + \mu_h$, $a_2 = \mu_h + \delta + \gamma_h$, $a_3 = \mu_v + \phi_v$ We get

$$V = \begin{bmatrix} a_1 & 0 & 0 & 0 \\ \phi_h & a_2 & 0 & 0 \\ 0 & 0 & a_3 & 0 \\ 0 & 0 & \phi_v & \mu_v \end{bmatrix} \tag{18}$$

The inverse of equation (18) is given as

$$V^{-1} = |V| * Adj(\mathrm{v}) = \begin{bmatrix} \frac{1}{a_1} & 0 & 0 & 0 \\ \frac{\phi_h}{a_1 a_2} & \frac{1}{a_2} & 0 & 0 \\ 0 & 0 & \frac{1}{a_3} & 0 \\ 0 & 0 & \frac{1}{\mu_v}\frac{\phi_v}{a_3} & \frac{1}{\mu_v} \end{bmatrix} \quad (19)$$

Multiplying equation (15) and (19) we get

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & q_1 \\ 0 & 0 & 0 & 0 \\ 0 & q_2 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{a_1} & 0 & 0 & 0 \\ \frac{\phi_h}{a_1 a_2} & \frac{1}{a_2} & 0 & 0 \\ 0 & 0 & \frac{1}{a_3} & 0 \\ 0 & 0 & \frac{\phi_v}{\mu_v a_3} & \frac{1}{\mu_v} \end{bmatrix} = \begin{bmatrix} 0 & 0 & \frac{\phi_v}{\mu_v a_3} & \frac{q_1}{\mu_v} \\ 0 & 0 & 0 & 0 \\ \frac{\phi_h q_2}{a_1 a_2} & \frac{q_2}{a_2} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (20)$$

Given the characteristic equation

$$|FV^{-1} - \lambda I| = 0 \quad (21)$$

Substituting (20) into (21) we have

$$\begin{vmatrix} 0-\lambda & 0 & \frac{1}{\mu_v}\frac{\phi_v}{a_3} & \frac{1}{\mu_v}q_1 \\ 0 & 0-\lambda & 0 & 0 \\ \frac{\phi_h}{a_1 a_2}q_2 & \frac{1}{a_2}q_2 & 0-\lambda & 0 \\ 0 & 0 & 0 & 0-\lambda \end{vmatrix} \quad (22)$$

Hence, the reproduction number for the dengue infection is the dominant Eigen value, thus; $R_0 = \rho(\mathrm{FV}^{-1})$,where ρ is the spectral radius is given as

$$R_0 = \sqrt{\frac{\phi_h \phi_v q_1 q_2}{\mu_v a_1 a_2 a_3}} \quad (23)$$

Where $a_1 = \phi_h + \mu_h$, $a_2 = \mu_h + \delta + \gamma_h$, $a_3 = \mu_v + \phi_v$, $q_1 = \frac{\beta_h b \Lambda_h \left((\omega + \mu_h) + \psi\upsilon \right)}{(\omega + \mu_h)(\upsilon + \mu_h) - \omega\upsilon}$

and $q_2 = \dfrac{\beta_v b\,\Lambda_v}{\mu_v}$

Thus

$$R_0 = \sqrt{\frac{\phi_h\phi_v b^2 \beta_v \Lambda_v \beta_h \, b\, \Lambda_h \left((\omega+\mu_h)+\psi\upsilon\right)}{\mu_v^2\left(\phi_h+\mu_h\right)\left(\mu_h+\delta+\gamma_h\right)\left(\mu_v+\phi_v\right)\left((\omega+\mu_h)\left(\upsilon+\mu_h\right)-\omega\upsilon\right)}} \qquad (24)$$

3.5 Local Stability of the Disease Free Equilibrium for the Dengue Infection

Theorem 1: The disease free equilibrium point E^0 of the model is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable otherwise

Proof: we let

$$\left.\begin{aligned}
f_1 &= \upsilon S_h - \frac{\psi\beta_h b I_v V_h}{N_h} - \omega V_h - \mu_h V_h \\
f_2 &= \Lambda_h N_h + \omega V_h + \sigma R_h - \upsilon S_h - \frac{\beta_h b I_v S_h}{N_h} - \mu_h S_h \\
f_3 &= \frac{\beta_h b I_v S_h}{N_h} + \frac{\psi\beta_h b I_v V_h}{N_h} - \phi_h E_h - \mu_h E \\
f_4 &= \phi_h E_h - \mu_h I_h - \delta I_h - \gamma I_h \\
f_5 &= \gamma I_h - \mu_h R_h - \sigma R_h \\
f_6 &= \Lambda_v N_v - \frac{\beta_v b I_h S_v}{N_v} - \mu_v S_v \\
f_7 &= \frac{\beta_v b I_h S_v}{N_v} - \mu_v E_v - \phi_v E_v \\
f_8 &= \phi_v E_v - \mu_v I_v
\end{aligned}\right\} \qquad (25)$$

The Jacobian matrix J for the system (25) is

$$\begin{bmatrix}
b_{11} & b_{12} & 0 & 0 & 0 & 0 & 0 & b_{18} \\
b_{21} & b_{22} & 0 & 0 & b_{25} & 0 & 0 & b_{28} \\
0 & 0 & b_{33} & 0 & 0 & 0 & 0 & b_{38} \\
0 & 0 & b_{43} & b_{44} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & b_{54} & b_{55} & 0 & 0 & 0 \\
0 & 0 & 0 & b_{64} & 0 & b_{66} & 0 & 0 \\
0 & 0 & 0 & b_{74} & 0 & b_{76} & b_{77} & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & b_{87} & b_{88}
\end{bmatrix} \qquad (26)$$

where

$$\left.\begin{aligned}
&b_{11}=-\frac{\psi\beta_h bI_h V_h}{N_h}-\omega-\mu_h, b_{12}=\upsilon, b_{18}=\frac{\psi\beta_h bV_h}{N_h}\\
&b_{21}=\omega, \mathrm{b}_{22}=-\upsilon-\frac{\beta_h bI_v}{N_h}-\mu_h, \mathrm{b}_{25}=\sigma, b_{28}=-\frac{\beta_h bI_v S_h}{N_h}\\
&b_{31}=\frac{\psi\beta_h bI_v}{N_h}, \mathrm{b}_{32}=\frac{\beta_h bI_v}{N_h}, b_{33}=-\phi_h-\mu_h, b_{38}=\frac{\beta_h bS_h}{N_h}+\frac{\psi\beta_h bV_h}{N_h}\\
&b_{43}=\phi_h, b_{44}=-\mu_h-\delta-\gamma_h\\
&b_{54}=\gamma_h, b_{55}=-\mu_h-\sigma\\
&b_{64}=-\frac{\beta_v bS_v}{N_v}, b_{66}=-\frac{\beta_v bI_h S_v}{N_v}-\mu_v,\\
&b_{74}=\frac{\beta_v bS_v}{N_v}, b_{76}=-\frac{\beta_v bI_h}{N_v}, \mathrm{b}_{77}=-\mu_v-\phi_v\\
&b_{87}=\phi_v, b_{88}=-\mu_v
\end{aligned}\right\} \quad (27)$$

Evaluating (26) at disease free equilibrium gives

$$J_{E^0}=\begin{bmatrix}
r_1 & \upsilon & 0 & 0 & 0 & 0 & 0 & m_1\\
b_1 & r_2 & 0 & 0 & \sigma & 0 & 0 & m_2\\
0 & 0 & r_3 & 0 & 0 & 0 & 0 & m_3\\
0 & 0 & b_2 & r_4 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & b_3 & r_5 & 0 & 0 & 0\\
0 & 0 & 0 & b_4 & 0 & r_6 & 0 & 0\\
0 & 0 & 0 & b_5 & 0 & 0 & r_7 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & b_6 & r_8
\end{bmatrix} \quad (28)$$

where

$$\left.\begin{aligned}
&r_1=(\omega+\mu_h);\ r_2=(\upsilon+\mu_h);\ r_3=(\phi_h+\mu_h);\ r_4=(\mu_h+\delta+\gamma_h);\ r_5=(\mu_h+\sigma)\\
&r_6=(\mu_v+\phi_v);\ r_7=-\mu_v,\ b_1=\omega,\ b_2=\phi_h,\ b_3=\gamma_h,\ b_4=\frac{-\beta_v b}{\mu_v},\ b_5=\frac{\beta_v b}{\mu_v},\ b_6=\phi_v\\
&m_1=\frac{-\psi\beta_h b\upsilon\Lambda_h}{(\omega+\mu_h)(\upsilon+\mu_h)-\omega\upsilon}, m_2=\frac{\beta_h b(\omega+\mu_h)\Lambda_h}{(\omega+\mu_h)(\upsilon+\mu_h)-\omega\upsilon}, m_3=\frac{\beta_h b\Lambda_h(\omega+\mu_h)+\psi\upsilon}{(\omega+\mu_h)(\upsilon+\mu_h)-\omega\upsilon}
\end{aligned}\right\} \quad (29)$$

From(28), $\lambda_1 = -\mu_v$, rewriting (28) we get

$$\begin{bmatrix} r_1 & \upsilon & 0 & 0 & 0 & 0 & m_1 \\ b_1 & r_2 & 0 & 0 & \sigma & 0 & m_2 \\ 0 & 0 & r_3 & 0 & 0 & 0 & m_3 \\ 0 & 0 & b_2 & r_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & b_3 & r_5 & 0 & 0 \\ 0 & 0 & 0 & b_5 & 0 & r_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & b_6 & r_7 \end{bmatrix} \qquad (30)$$

Using scientific workplace, the characteristic polynomial satisfies

$$Q\lambda^7 + Q\lambda^6 + Q\lambda^5 + Q\lambda^4 + Q\lambda^3 + Q\lambda^2 + Q\lambda + Q_0 \qquad (31)$$

We applied the Ruth – Hurwitz criterion which states that all the roots of the polynomial (31) have negative real parts if and only if the coefficients Q_k, are positive and the determinant of the matrix $H_k > 0$ for $k = 1, 2, 3, ...7,$ therefore from the equation (31) we have

For $n = 7$,

$$H_K = \begin{vmatrix} p_6 & p_4 & p_2 & p_0 & p_{-2} & p_{-4} & p_{-6} \\ p_7 & p_5 & p_3 & p_1 & p_{-1} & p_{-3} & p_{-5} \\ 0 & p_6 & p_4 & p_2 & p_0 & p_{-2} & p_{-4} \\ 0 & p_7 & p_5 & p_3 & p_1 & p_{-1} & p_{-3} \\ 0 & 0 & p_6 & p_4 & p_2 & p_0 & p_{-2} \\ 0 & 0 & p_7 & p_5 & p_3 & p_1 & p_{-1} \\ 0 & 0 & 0 & p_6 & p_4 & p_2 & p_0 \end{vmatrix} > 0 \qquad (32)$$

$$H_7 = \begin{vmatrix} Q_1 & Q_3 & Q_5 & Q_7 & 0 & 0 & 0 \\ Q_0 & Q_2 & Q_4 & Q_6 & 0 & 0 & 0 \\ 0 & Q_1 & Q_3 & Q_5 & Q_7 & 0 & 0 \\ 0 & Q_0 & Q_2 & Q_4 & Q_6 & 0 & 0 \\ 0 & 0 & Q_1 & Q_3 & Q_5 & Q_7 & 0 \\ 0 & 0 & Q_0 & Q_2 & Q_4 & Q_6 & 0 \\ 0 & 0 & 0 & Q_1 & Q_3 & Q_5 & Q_7 \end{vmatrix} > 0 \qquad (33)$$

Assuming a positive coefficient for equation (31)

For $k = 1$,

$$H_1 = |Q_2| > 0, \quad Q_2 > 0 \qquad (34)$$

For $k = 2$,

$$H_2 = \begin{vmatrix} Q_2 & Q_4 \\ Q_1 & Q_3 \end{vmatrix} > 0$$

$$H_2 = Q_2 Q_3 - Q_1 Q_4 > 0 \tag{35}$$

$$H_2 = Q_2 Q_3 > Q_4$$

For $k = 3$,

$$H_3 = \begin{vmatrix} Q_2 & Q_4 & Q_6 \\ Q_1 & Q_3 & Q_5 \\ 0 & Q_2 & Q_4 \end{vmatrix} > 0 \tag{36}$$

For $k = 4$,

$$H_4 = \begin{vmatrix} Q_1 & Q_4 & Q_6 & Q_0 \\ 1 & Q_3 & Q_5 & Q_7 \\ 0 & Q_2 & Q_4 & Q_6 \\ 0 & Q_1 & Q_3 & Q_5 \end{vmatrix} > 0 \tag{37}$$

For $k = 5$

$$H_5 = \begin{vmatrix} Q_2 & Q_4 & Q_6 & Q_0 & 0 \\ 1 & Q_3 & Q_5 & Q_7 & 0 \\ 0 & Q_2 & Q_4 & Q_6 & 1 \\ 0 & 1 & Q_3 & Q_5 & Q_7 \\ 0 & 0 & Q_2 & Q_4 & Q_6 \end{vmatrix} > 0 \tag{38}$$

For k=6

$$H_6 = \begin{vmatrix} Q_2 & Q_4 & Q_6 & Q_0 & 0 & 0 \\ 1 & Q_3 & Q_5 & Q_7 & 0 & 0 \\ 0 & Q_2 & Q_4 & Q_6 & 1 & 0 \\ 0 & 1 & Q_3 & Q_5 & Q_7 & 0 \\ 0 & 0 & Q_2 & Q_4 & Q_6 & 0 \\ 0 & 0 & 1 & Q_3 & Q_5 & Q_7 \end{vmatrix} > 0 \tag{39}$$

For $k = 7$

$$H_7 = \begin{vmatrix} Q_2 & Q_4 & Q_6 & Q_0 & 0 & 0 & 0 \\ 1 & Q_5 & Q_5 & Q_7 & 0 & 0 & 0 \\ 0 & Q_2 & Q_4 & Q_6 & 1 & 0 & 0 \\ 0 & 1 & Q_3 & Q_5 & Q_7 & 0 & 0 \\ 0 & 0 & Q_2 & Q_4 & Q_6 & Q_0 & 0 \\ 0 & 0 & 1 & Q_3 & Q_5 & Q_7 & 0 \\ 0 & 0 & 0 & Q_2 & Q_4 & Q_6 & Q_0 \end{vmatrix} > 0 \quad (40)$$

Therefore, all the Eigen values of the polynomial (31) have negative real parts, implying that $\lambda_1 < 0,\ \lambda_2 < 0,\ \lambda_3 < 0,\ \lambda_4 < 0,\ \lambda_5 < 0,\ \lambda_6 < 0,\ \lambda_7 < 0, \lambda_8 < 0$, Since all values of $\lambda_i < 0$ for $i = 1,2,3,...8$ when $R_0 < 0$ we conclude that the disease free equilibrium point is Locally Asymptotically Stable (LAS).

3.6 Global Stability of the Disease-Free Equilibrium

We used the method of $[9]$ to obtain the global stability of the disease equilibrium point. Two conditions which guarantee the global stability of the disease- Free State was considered. Therefore, our system of equations (1)-(8) is re-write in the following form;

$$\left.\begin{aligned} \frac{dX}{dt} &= F(X,Z) \\ \frac{dZ}{dt} &= G(X,Z), G(X,0) = 0 \end{aligned}\right\} \quad (41)$$

Where $X = (S_h, V_h, R_h, S_v)$ denotes the number of uninfected individuals and $X \in R^4$, while $Z = (\mathrm{E}_h, \mathrm{I}_h, \mathrm{E}_v, \mathrm{I}_v)$ denotes the number of infected individuals and $Z \in R^4$. We represent the disease-free state by $E^0 = (X^0, 0)$ .The following two conditions H_1 and H_2 must be met to guarantee a global asymptotic stability:

$$H_1 : \text{for } \frac{dX}{dt} = F(X,0) \quad (42)$$

Where X^0 is globally asymptotically stable

$$H_2 : \hat{G}(X,Z) = CZ - G(X,Z), \quad (43)$$

where $\hat{G}(X,Z) \geq 0,$ for $(X,Z) \in \Omega$, $C = D_Z G(X^0, 0)$ is an $M - matrix$ (the off diagonal of C are non-negative) and Ω is the biological feasible region.

Lemma 1: The point $K^0 = (X^0, 0)$ is called stable global asymptotic equilibrium point, if in addition $R_0 < 1$ and the conditions H_1 and H_2 holds. The following theorem is formed:

Theorem 2: Let $R_0 < 1$, then the disease free equilibrium is globally asymptotically stable and unstable if otherwise.

Proof:

Let $X = (S_h, V_h, R_h, S_v)$, $Z = (E_h, I_h, E_h, I_v)$ and $K^0 = (X^0, 0)$ where

$X \in R^4$,

$$\frac{dV_h}{dt} = \upsilon S_h - \frac{\psi\beta_h b I_v V_h}{N_h} - \omega V_h - \mu_h V_h \tag{44}$$

$$\frac{dS_h}{dt} = \Lambda_h N_h + \omega V + \sigma R_h - \upsilon S_h - \frac{\beta_h b I_v S_h}{N_h} - \mu_h S_h \tag{45}$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h - \sigma R_h \tag{46}$$

$$\frac{dS_v}{dt} = \Lambda_v N_v - \frac{\beta_v b I_h S_v}{N_v} - \mu_v S_v \tag{47}$$

$$F(X,0) = \begin{pmatrix} \upsilon S_h - (\omega + \mu_h) V_h \\ \Lambda_h N_h + \omega V_h - (\upsilon + \mu_h) S_h \\ 0 \\ \Lambda_v N_v - \mu_v S_v \end{pmatrix} \tag{47}$$

$$\left.\begin{aligned} &Z \in R^4 \Rightarrow \\ &\frac{dE_h}{dt} = \frac{\beta_h b I_v S_h}{N_h} + \frac{\psi\beta_h b I_v V_h}{N_h} - \phi_h E_h - \mu_h E_h \\ &\frac{dI_h}{dt} = \phi_h E_h - \mu_h I_h - \delta I_h - \gamma_h I_h \\ &\frac{dE_v}{dt} = \frac{\beta_v b I_h S_v}{N_v} - \mu_v E_v - \phi_v E_v \\ &\frac{dI_v}{dt} = \phi_v E_v - \mu_v I_v \end{aligned}\right\} \tag{48}$$

Evaluating the Jacobian of (48) at disease free equilibrium we have

$$C=\begin{bmatrix} -(\phi_h+\mu_h) & 0 & 0 & \dfrac{\beta_h S_h^0}{N_h}+\dfrac{\psi\beta_h bV_h^0}{N_h} \\ \phi_h & -(\mu_h+\delta+\gamma_h) & 0 & 0 \\ 0 & \dfrac{\beta_v bS_v^0}{N_v} & -(\phi_v+\mu_v) & 0 \\ 0 & 0 & \phi_v & -\mu_v \end{bmatrix} \tag{49}$$

Equation (49) can be rewriting as

$$CZ=\begin{bmatrix} -(\phi_h+\mu_h) & 0 & 0 & \dfrac{\beta_h S_h^0}{N_h}+\dfrac{\psi\beta_h bV_h^0}{N_h} \\ \phi_h & -(\mu_h+\delta+\gamma_h) & 0 & 0 \\ 0 & \dfrac{\beta_v bS_v^0}{N_v} & -(\phi_v+\mu_v) & 0 \\ 0 & 0 & \phi_v & -\mu_v \end{bmatrix}\begin{bmatrix} E_h \\ I_h \\ E_v \\ I_v \end{bmatrix}$$

$$=\begin{bmatrix} -(\phi_h+\mu_h)\mathrm{E}_h+ & 0 & 0 & \dfrac{\beta_h S_h^0 I_v}{N_h}+\dfrac{\psi\beta_h bI_v V_h^0}{N_h} \\ \phi_h E_h & -(\mu_h+\delta+\gamma_h)I_h & 0 & 0 \\ 0 & \dfrac{\beta_v bI_h S_v^0}{N_v} & -(\phi_v+\mu_v)E_v & 0 \\ 0 & 0 & \phi_v E_v- & -\mu_v I_v \end{bmatrix} \tag{50}$$

$$\hat{G}(X,\mathrm{Z})=CZ-G(X,Z) \tag{51}$$

$$\hat{G}(X,Z)=\begin{pmatrix} \hat{G}_1(X,Z) \\ \hat{G}_2(X,Z) \\ \hat{G}_3(X,Z) \\ \hat{G}_4(X,Z) \end{pmatrix}=\begin{pmatrix} \beta_h bI_V\left(\dfrac{S_h^0}{N_h^0}-\dfrac{S_h}{N_h}\right)+\psi\beta_h bI_v\left(\dfrac{V_h^0}{N_h^0}-\dfrac{V_h}{N_h}\right) \\ 0 \\ \beta_v bI_h\left(\dfrac{S_v^0}{N_v^0}-\dfrac{S_v}{N_v}\right) \\ 0 \end{pmatrix} \tag{52}$$

Therefore, since $\frac{S_h}{N_h} \le \frac{S_h^0}{N_h}$, $\frac{V_h}{N_h} \le \frac{V_h^0}{N_h^0}$ and $\frac{S_v}{N_v} \le \frac{S_v^0}{N_v^0}$ we have $\hat{G}(X,Z) \ge 0$, The global stability of $X^0 = \left(S_h^0, V_h^0, 0, 0, S_v^0, 0, 0\right)$ the system $\frac{dX}{dt} = F\left(X^0, 0\right)$ is easy to verify. Therefore X^0 is globally asymptotically stable if $R_0 < 1$. This completes the proof.

4.0 Numerical Simulation

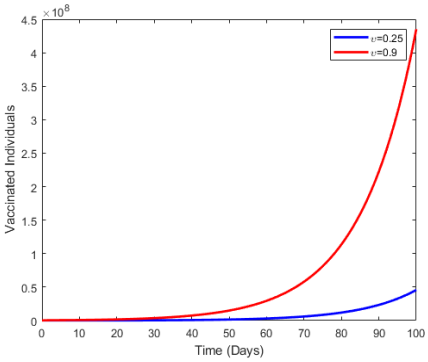
Here, we presented the numerical results for the dengue fever model. The graphs presented in Figure 2 to Figure 6 are generated using variables and parameters in Table 3 and 4.

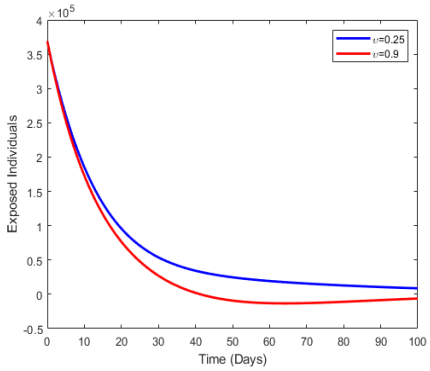
Table 3. Parameter values used for numerical simulations

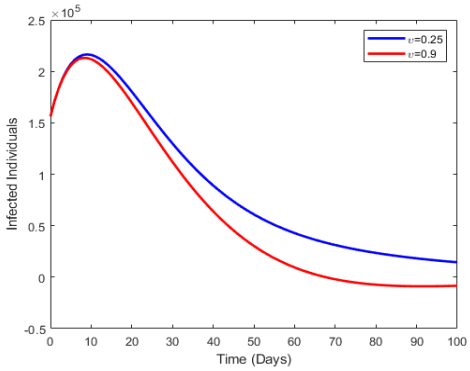
Parameter	Value	Reference
β_h	0.75	[10]
β_v	0.75	[10]
b	0.333	[10]
Λ_v	0.0323000	[8]
Λ_h	0.0000460	[8]
γ_h	0.00019	[11]
ϕ_v	0.75	[11]
ϕ_h	0.723	[11]
μ_h	0.004	[11]
μ_v	0.86	[11]
δ	0.333	[11]
σ	0.0146	[11]
ψ	0.51	Assumed
ω	0.025	Assumed
υ	0.8	[12]

Table 4 Variable initial values used for numerical simulations

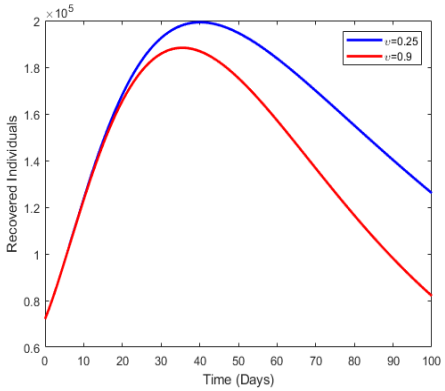
Variable	Value	Reference
E_h	369,150	[11]
E_v	32,000	[11]
I_h	156,170	[11]

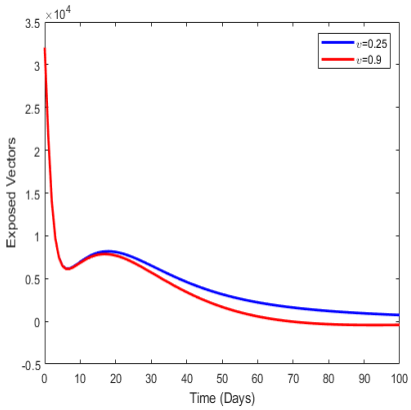


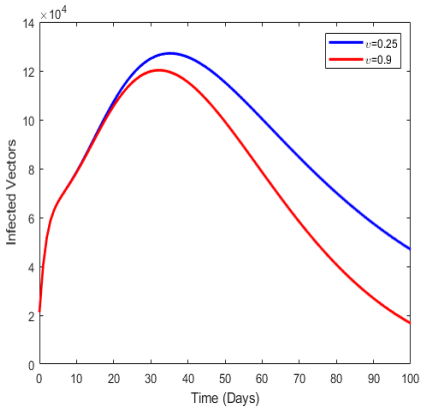




I_v	21,200	[11]
S_h	406,250	[11]
S_v	40,200	[11]
V_h	20000	Assumed

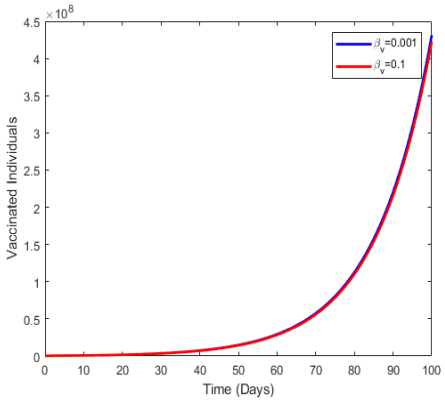






a b

c



d

e

f

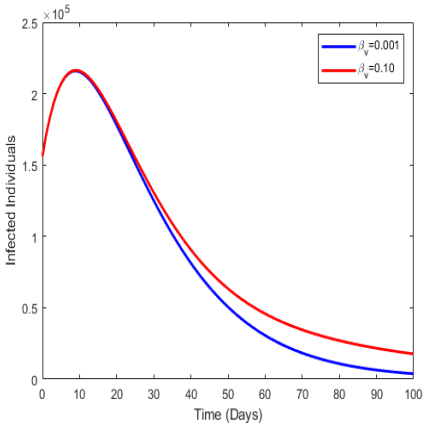
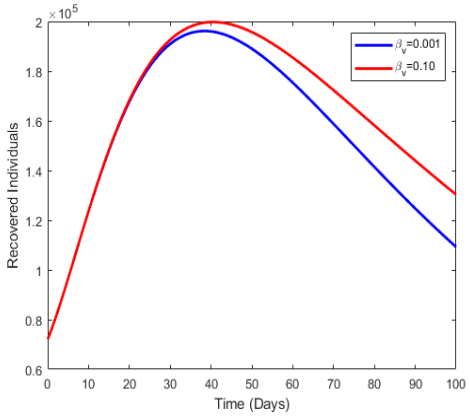
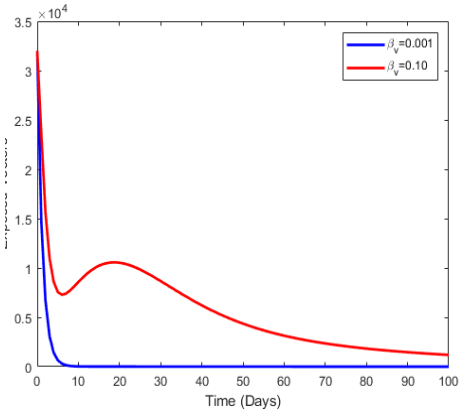


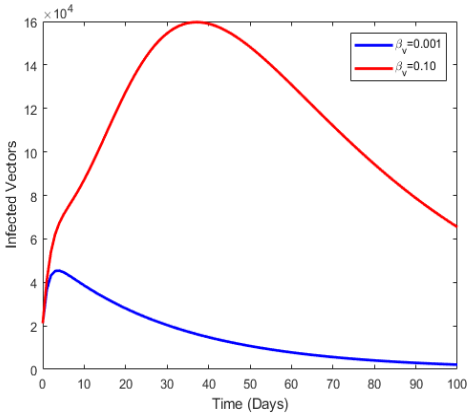
Figure 2:(a-f): Showing the effects of vaccination on the Dengue fever



a



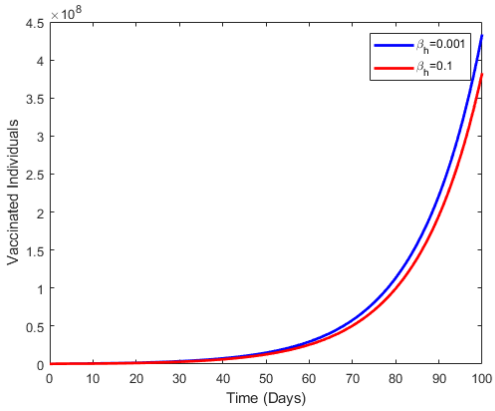
b



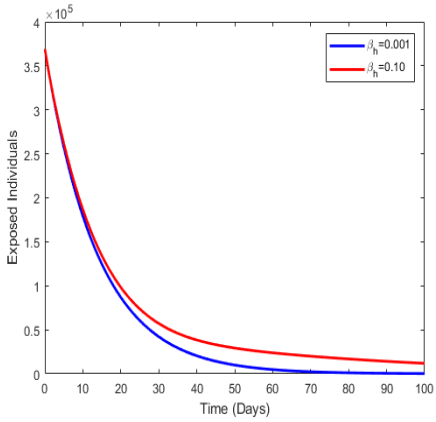
c

d e f

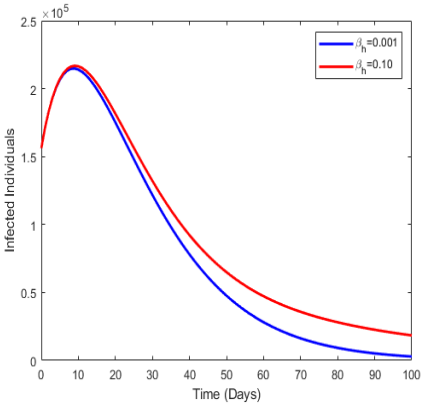
Figure 3(a-f): Showing the effects of probability of vector transmission on the Dengue fever



a

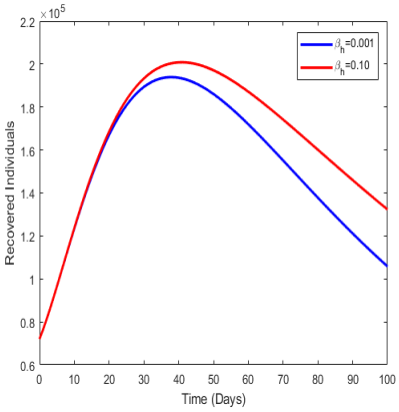


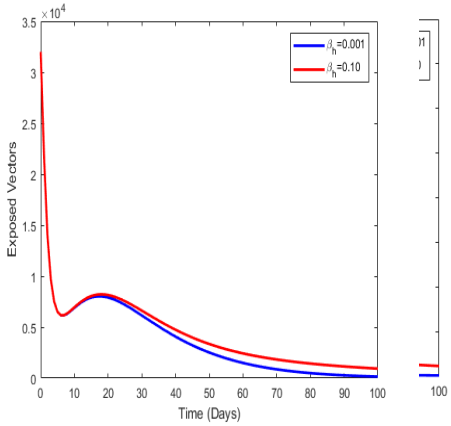
b



c

d e f





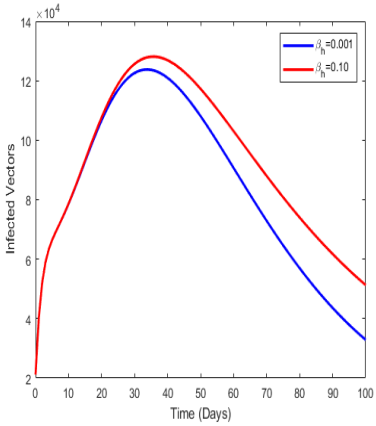
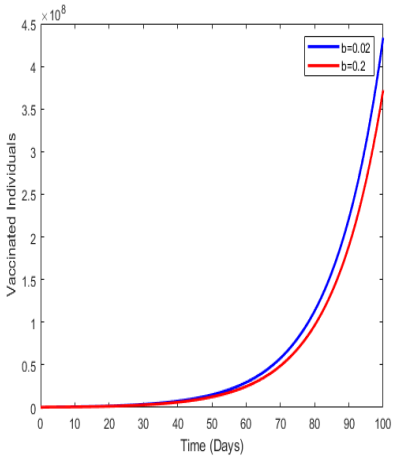
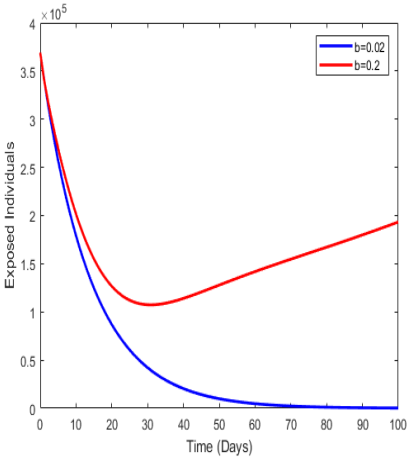
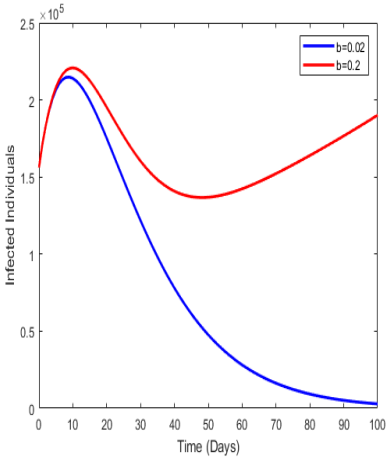


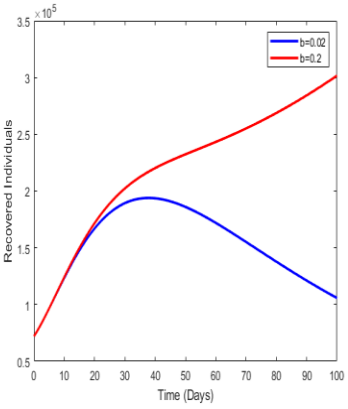
Figure 4:(a-f): Showing the effects of probability of human transmission on the Dengue fever

a b c

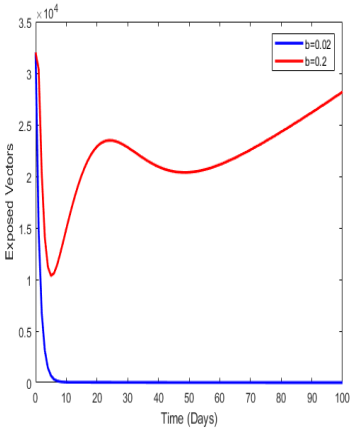




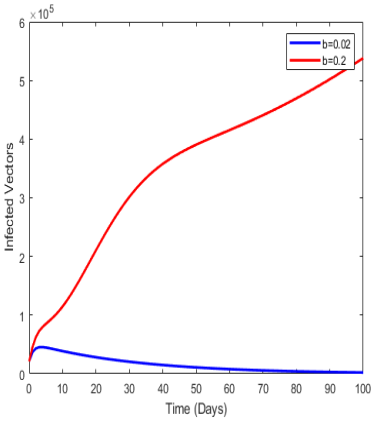




d



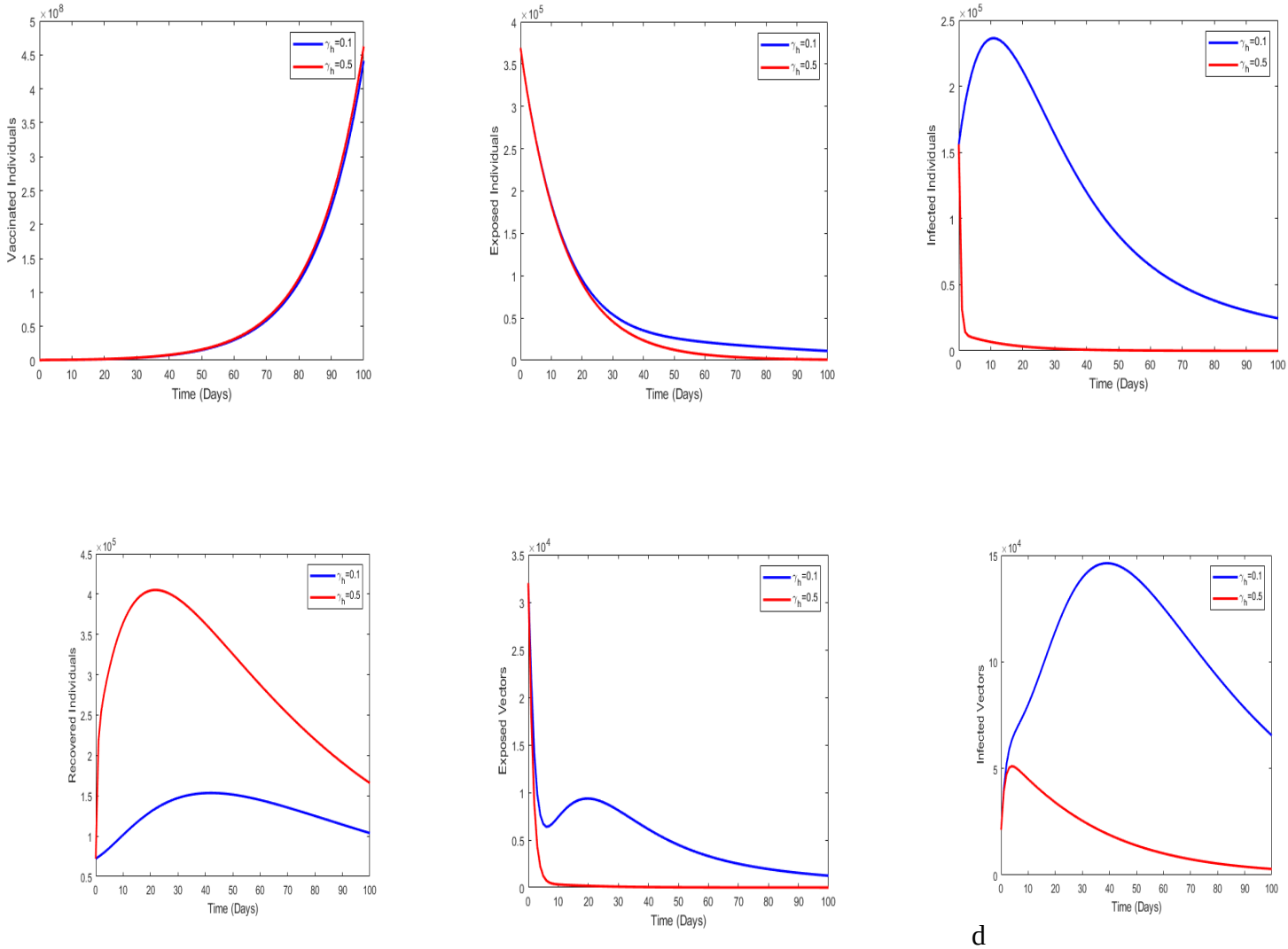
e



f

Figure 5(a-f): Showing the effects of biting rate of mosquitoes on the Dengue fever

a b c



e f d

Figure 6 :(a-f): Showing the effects of treatment rate on the Dengue fever

5.0 Discussion of Results

5.1 Discussion of analytical result

The dengue model consists of eight (8) system of nonlinear ordinary differential equations. The model contained two stages of dengue infections namely, exposed and infected stages. We obtained the positivity of the solution which shows that the system of differential equations have non-negative solutions for all time and is bounded within the given region: The disease-free

and endemic equilibrium points were obtained and the next-generation matrix method was used to determine the basic reproduction number (R_0).

By Ruth – Hurwitz criterion and Castillo – Chavez conditions were applied to prove the local and the global stability. And the disease-free equilibrium is proved to be locally asymptotically stable (LAS) and globally asymptotically stable (GAS) when the associated reproduction number is less than unity $(R_0 < 1)$. This simply means, the disease will be completely wiped out if the system is stable and will be endemic when it is unstable, that is if the reproduction number is greater than unity $(R_0 > 1)$.

5.2 Discussion of numerical simulation results

Figure 2 (a) to Figure 2 (f) Showing the effect of vaccination on the transmission of dengue fever,. From Figure 2 (a) the number of vaccinated individuals started gradually and rises up very fast while the number of those without vaccination dies out . Figure 2 (b) the effect of vaccination on the exposed individuals shows that the exposed individuals decrease to a very low level. Figure 2(c)is similar to the exposed individuals the effect of vaccine reduces the number of infected individuals to a significant level when compared to those without vaccination. From Figure 2(d) we observed that the number of recovered individuals as the vaccination is going on in the population there is less people to treat. Figure 2(e) Exposed vector population decrease and dies out. This is in line with our main objective to find out the effect of vaccination in the prevention of dengue fever epidemic in the community. From Figure 2 (f) we observed that the infected vector population grows at first but later decreased and dies out.

Figure 3(a) to Figure 3(f) showing the effects of probability of vector transmission on dengue fever. From Figure 3(a) we observed that vector transmission has less effect on vaccination we can see that the number of vaccinated individuals keeps on rising despite there is vector transmission in the population. Figure b (b) this figure shows a significant decline inthe number of exposed individuals but towards the end it changes which can be attributed to vaccine failure. Similarly, Figure 3 (c) This figure also shows a similar result to that of (b) above. Figure 3(d) At the beginning the figure shows a significant increase in the number of recovered individuals up to the maximum level before declining and this can be as a result of wining out of vaccine Figure 3(e) Here the figure shows no meaningful result. Figure 3(f) shows no meaningful result.

Figure 4(a) to Figure 4(f) showing the effects of probability of human transmission on dengue fever. Figure 4(a) shows that with vaccination human transmission is les compared to without vaccination. From Figure 4(b) we observed that the exposed individuals decline perfectly before reaching the bottom it started changing which could be associated to wining of vaccine. In Figure 4 (c) this figure shows no significant result. Figure 4(d)This figure shows that the number of recovered individuals increases significantly before dropping which can be due to vaccine failure or wining out of vaccine. Figure 4(e) shows a significant decline in the number of exposed vectors but towards the bottom line there is a little failure. Figure 4(f) The figure shows that since the vectors are not protected vaccination has no effect on human transmission once there is an infected individual can be infected.

Figure 5 (a) to Figure 5(f) showing the effect of biting rate of mosquito on dengue fever. From figure 5(a) we observed that vaccination has a significance on biting rate from the two curves we can see that when there is vaccination less people were infected as a result of biting rate. Figure 5(b) Gives no meaningful result. Figure 5 (c) Here also there is no reasonable result. Figure 4.4 (d) Recovered individuals increased significantly these includes those exposed who also received vaccination would not accommodate more virus so they will recover from dengue. Figure 5(e). Here there is no meaningful result. Similarly, Figure (f).

Figure 6(a) to Figure 6(f) showing the effect of treatment on dengue fever. Figure 6 (a) we observed that there is a positive result for both vaccinated and not vaccinated which could be attributed to prompt intervention for treatment for eradicating dengue. Figure 6(b) gives the interpretation that the higher the treatment the faster the number of exposed individuals is dropping down with vaccination. Figure 6(c) Here the curve for vaccination shows the level at which the infected individuals drop significantly for higher treatment Figure6(d) Here with treatment the recovered population rises very fast and later drops down it can be as a result of remaining only few people to treat due to dengue. Figure 6(e) we observed that with vaccination and treatment the number of exposed vectors drops down and move to extinction. Figure 6(f) here the infected vector population is less in number and move to extinction.

Conclusion

In this research work, we proposed and studied mathematical analysis of dengue fever model with two stages of infections which is a modified version of work of [7] Syafruddin and Salmi (2013 The positivity of the solution is proved. We obtained the basic reproduction number using next generation matrix method. The disease free and the endemic equilibrium points were established. The disease free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. The global stability of the disease free is proved to be globally asymptotically stable when the associated reproduction number is less than unity $(R_0 < 1)$. This implies that the disease will die out completely in a stable equilibrium while it will persist and become endemic in an unstable equilibrium. We recommend that dengue should be treated at early stage of infection because if not treated it progresses to a more serious form of the fever known as dengue hemorrhagic or dengue shock syndrome, where these two are life threatening form of the dengue. Government should provide necessary equipment for identifying dengue from other form of fevers because most of the times it is treated as either malaria or typhoid fever in other to avoid complications.

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